UNIVERSITY OF LONDON
FRANCIS GALTON LABORATORY FOR NATIONAL EUGENICS

TREASURY OF HUMAN INHERITANCE

EDITED BY
KARL PEARSON, F.R.S.
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VOLUME I

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The power of man in varying the future human stock vests a great responsibility in each fresh generation, which has not yet been recognised at its just importance, nor deliberately employed.

FRANCIS GALTON, *Inquiries into Human Faculty*, 1883.

LONDON:
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1912

Tenacity, physical and mental

Lucy Barclay (Mrs Samuel Galton) (1757—1817)
Descended from the Apologist Barclay, Cameron of Lochiel and James I of Scotland.
(Photo. Eugenics Laboratory.)

Power of Organisation

Samuel Galton, F.R.S.
(1753—1832)
Leader of Industry and Physicist
(Photo. Eugenics Laboratory.)

Sir Francis Galton, F.R.S.
(1822—1911)
Naturalist and Statist.
(Photo. Maull & Fox.)

Scientific Imagination

Erasmus Darwin, F.R.S.
(1731—1802)
Physician, Naturalist and Poet.
(Photo. Emery Walker.)

Wit and Literary Power

Catherine Sedley, Countess of Dorchester
(1657—1717)
Court Beauty and Wit,
Grandmother of Mrs Erasmus Darwin.
(Photo. Eugenics Laboratory.)

Sir Francis Galton, and some of his Noteworthy Ancestors.

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If the *Treasury* prospers, as is hoped and expected, a vast amount of information will gradually be collected by its means, in a form suitable for analysis, that will enable more exact conclusions to be hereafter drawn and more emphatic advice to be given than is now possible.

In conclusion I may perhaps be permitted to express my own sincere gratification that the Eugenics Laboratory has already become so well equipped and conditioned as to undertake the publication of this large and important serial.

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Against these reasons were others, however, which weighed largely with those directing the Laboratory, when a final decision had to be made. Such a Treasury of Human Inheritance is really a pressing necessity of the time. This is true not only from the standpoint of the science of heredity, but from the standpoint of national eugenics. Questions have arisen and will in future continue to arise demanding answers not only from our legislators, but also from municipal and social workers, and such answers can only be given on the basis of great masses of material patiently collected and published in the present manner. The scientific maintenance of the health, physical and mental, of the nation is becoming the order of the day; and the perpetuation of sound stock in the nation is no less important than a "two-power standard" of the fleet. The Galton Laboratory would therefore have missed an opportunity of carrying out the work for which it was established and endowed had it allowed this pressing need of a *Thesaurus* or *Treasury* to pass unheeded. There were other factors also to be taken into consideration. Among these may be mentioned, the existence of several hundreds of pedigrees of family characters in its archives, the experience already gained by its members in the preparation of

family history, and the elaboration of a combined system of draughtsmanship and lithography by which nearly 600 pedigrees had previously been placed upon plates. Help also was forthcoming from pathologists and clinicians when appeal was made to them, and the present and future volumes will, I believe, demonstrate that we shall not fail for medical assistance. Here also we reach a matter which deserves emphasis. This *Treasury* will do service of a fundamental nature, if it convinces its readers that the human being is to be treated as a whole; there is not one inheritance of disease, another of anthropometric characters and a third of psychical qualities. You cannot divide the human subject up and isolate the pathologist, the anthropologist and the psychologist in their own fields. Each has to recognise the work of his neighbour and to see his own in the light of a broader whole. The old views as to the relationship of temperament and disease aimed at half-seen truths, which we may hope our *Treasury* will help to disclose. The pathological state, the psychical temperament and the physique are correlated characteristics in man. No student of family history can fail to be more and more impressed as his knowledge advances with the many links between mental and physical abnormality. The pathologist must ever be on the watch for mental associations, and the psychologist will find some of the most fruitful directions of investigation arise in connection with pathological defects. If we can in any way contribute to illustrating the unity of the science of heredity both in its ultimate laws, and in the high correlations which often exist between human characters, we shall be repaid for much of the labour spent in the production of this work. To a certain extent the Galton Laboratory may consider itself fitted to bring to a focus the work of anthropologist, psychologist and pathologist.

The publication of family histories—whether they concern physique, abnormality, ability or achievement—whether they be new or old—is the purpose of this *Treasury*. Students of heredity find great difficulty in obtaining easy access to material bearing on human inheritance. The published material is voluminous, scattered over a wide and often very inaccessible journalistic area. The already collected although unpublished material is probably as copious but no central organ for its rapid publication in a standardised form exists at present. The Eugenics Laboratory alone possesses several hundred pedigrees of family characteristics and diseases which it is desirable to make readily accessible. Many medical men possess similar material, and there is a growing desire among genealogists to pay more attention to family characters and supplement the merely nominal pedigrees, current in the past. There is an extraordinary fascination in following out a complex and difficult pedigree, and step by step building up the history of a family character.

A complete pedigree is often a work of great labour, and in its finished form is frequently a real work of art. To the many who have felt the delights of genealogical inquiry, we would say: Widen your outlook, recognise that there is something beyond names, births and deaths worthy of record, and, as it is harder to ascertain, more exciting in the pursuit. The pedigree of temperament, disease, ability, and physique which ought to replace the old nominal pedigree—if not for exhibition—at

least in the family archives is the true measure of the fitness of a stock, and the best guide to the younger members in their choice of career and alliance.

For a publication of this kind to be successful at the present time, it should, as I have indicated above, be entirely free from controversial matter. The *Treasury of Human Inheritance* therefore contains no reference to theoretical opinions. It gives in a standardised form the pedigree of each stock. This is accompanied by a few pages of text describing the individual members of the stock, giving references to authorities, and, if the material has been published, to the *locus* of original publication. When necessary the characteristic is illustrated by photography or radiography. In this way, it is hoped in the course of a few years to place a large mass of material in the hands of the student of human heredity. It will not cut him off from, but directly guide him to original and fuller sources of information. Further the *Treasury* will provide students of eugenics and of sociology, medical men and others with an organ where their investigations can find ready publication, and where as time goes on a higher and more complete standard of family history than has hitherto been usual can be maintained.

Each pedigree and its description appears under the name of the author responsible for its completeness and accuracy; and by aid of a key number to a confidential manuscript register of names and localities, it is hoped that it may be occasionally possible for future investigators to recover traces of individual stocks, or to ascertain whether newly discovered cases can be linked on to previously recorded families[1].

No one who has attempted a collection of this kind drawn from many quarters and prepared by different writers, will be over severe on discrepancies and omissions in the earlier issues. The full work of standardisation can only be carried out as the diverse needs of different types of family characters are better appreciated. It is not always possible to maintain a proper balance between the graphic and verbal descriptions; but I wish most strongly to insist on the point that neither are to be interpreted *alone*; they are component parts of one whole, and the reader who draws conclusions from the engraved pedigrees without consulting the verbal accounts is certain to be led into error. Presence or absence of a character cannot be settled by the simple blacking or omitting to black a circle. The description is practically that of the original observer, whereas the pedigree is the work of the author of the special section of the *Treasury* and he may under- or over-estimate the statement presented to him. As general editor, I feel sure that much care has been taken to reach an unbiassed judgment, and I know that contributors and members of this laboratory have spent many days of labour in following up both original pedigrees and pedigrees in dissertations, books and journals very hard indeed of access.

Suggestions for improvement in the form of our work will be gratefully received and duly considered. We seek aid and contributions of material from all those interested in heredity whatever be their theoretical position, and whether they approach the matter from the standpoint of pathologist, biologist or eugenist. Our

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keenest wish has been that this work should have a long and useful career, and growing more effective with its age, should be a permanent witness to the value of the inquiries fostered and to a great extent initiated by Francis Galton. From this standpoint I rejoice in republishing the few words he wrote three years ago at my request to speed us on our way.

The labour of editing the *Treasury* has been far more onerous, the expense much greater than we anticipated three years ago. Many writers, some with, others without any acknowledgment have reproduced pedigrees or illustrations from this work. We presume therefore that the labour and expense have not been wholly wasted; yet a little more generous recognition might have enabled us to reach that mimimum of subscribers which is essential for the permanency of an encyclopaedic work of this nature. Are we to continue or not is the question which the next year must settle, and the answer must lie largely with the efforts which our present well-wishers make to increase our circulation.

KARL PEARSON.

Galton Laboratory of National Eugenics,
University of London.
January 14, 1912.

CONTENTS OF VOL. I.

ILLUSTRATIVE PLATES.

[1 Owing to a slip on my part, this dwarf is termed Dutch on Plate JJ. K. P.]

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PARTS I AND II

WITH 13 PLATES OF PEDIGREES AND 5 PLATES OF ILLUSTRATIONS

PLATES I—XIII PLATES A—E

PEDIGREES 1—76

LONDON:

Published by the Cambridge University Press, Fetter Lane, E.C. 4

1909

CONTENTS OF PARTS I—II.

(This will be replaced by complete list of contents when the first volume is finished.)

[1] This Plate will appear with Part III.

ILLUSTRATIVE PLATES.

Further portions of the chief Sections of these two Parts will appear in later issues.

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TREASURY OF HUMAN INHERITANCE.

SECTION I *a*. DIABETES INSIPIDUS.

By W. Bulloch, M.D.

Diabetes insipidus (*polyuria, hydruria*) is a condition in which the affected individuals pass large quantities of urine of low specific gravity and without the presence of abnormal constituents. Excessive thirst (polydipsia) usually accompanies the drain of water through the kidneys. Diabetes insipidus may be a congenital or acquired condition, cases of the latter being subdivided according as they are idiopathic or secondary, the usual manifest cause being some injury of the central nervous system. Nothing is known with certainty as to the actual cause of the disease. From time to time, however, during the last seventy years cases have been recorded in which several members of a family have been afflicted. In other respects the individuals may be perfectly healthy and in not a few instances have attained a ripe old age. Nothing characteristic has been found in cases which have come to *post mortem* examination. In recent years it has been customary to regard the polyuria as primary and due to some inherent inability of the kidney to pass a urine of normal concentration. The urinary constituents which must be got rid of require a much larger quantity of water for their solution and thus a constant drain of water is going on which is the essential factor in the creation of the excessive thirst or polydipsia. The most remarkable cases in which heredity has played a part are those recorded by the Weils[1 and 2] (father and son, 1884—1908), Lauritzen[3], Gabriel Pain[4], Orsi[5], Lacombe[6], Clay[7], Deebrey[8], McIlraith[9], Reith[10], Wachsmuth[11], Knöpfelmacher[12], Sasse[13], and Gee[14]. (See Plates I. and II.)

The subject of diabetes insipidus may be studied in the special papers by Lanceraux[15], Meyer[16], Seiler[17], and in such standard works as Nothnagel[18], Clifford Allbutt[19], and Roberts[20].

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HEREDITARY CASES.

Fig. 1. *Weils' Case.* I. 2, Johann Peter Schwarz, born 1772, lived in Frischborn, Oberhessen, and died there in 1855, aged 83. His descendants were 5 children, 29 grandchildren, 66 great-grandchildren, and 119 great-great-grandchildren, altogether 220 persons. Of these 220, 35 had diabetes insipidus, the founder of the family, 3 children, 7 grandchildren, 13 great-grandchildren, 11 great-great-grandchildren. There are besides 16 doubtful cases, viz. 9 grandchildren and 2 great-grandchildren who died young, and 5 great-great-grandchildren whose case is still doubtful. If we omit these 16 cases we have 204 people of whom 169 were normal, and 35 had diabetes insipidus. The doubtful cases are IV. 4, 5, 6, 10, 17, 20, 21, 25, 26; V. 69, 104; VI. 80, 112, 116, 117, 118. II. 2, ♂ (1772—1855), weaver, lived in Frischborn, Kreis Lauterbach, in Oberhessen. According to his three living daughters, he had symptoms of the disease from his earliest youth, till his death. It is said his parents, brothers and sisters, were all perfectly normal. Wife (II. 3) normal, and lived to be 80, nothing stated of her parents. Family of five, one son normal, three daughters with diabetes, one daughter normal. III. 2, ♀ (1801—1875), lived in Maar near Lauterbach. Affected with polydipsia from earliest youth till death, both day and night, otherwise never ill, died of old age. Husband presumably normal, died at 60. Family of five; one son diabetic, one son (IV. 2) normal, married to presumably normal wife and had ten living children, all normal; one stillborn daughter, two daughters died very young—doubtful whether they had diabetes or not, IV. 4—6. IV. 1, ♂ (1834—1855), unmarried, died of small-pox, was affected by diabetes to a great degree, drank 6—8 litres a day—awakened often in the night. III. 4, ♀ (1802—), normal, never ill, married presumably normal husband. Family, two daughters normal, one daughter married and had four normal children, other daughter died of meningitis at age of 14. III. 6, ♀ (1807—1899), affected from her earliest years with diabetes, especially in 17th to 18th year, constant between 20—60. From 60—70 still drank much, in the next 5—6 years, thirst and secretion of urine diminished by one half. She then drank 2½ litres of water per day and awakened regularly twice by night to drink ½ litre, otherwise very healthy; arteries hardly sclerotic; inner organs not visibly altered. From 8 p.m. to 9 a.m. she drank two litres of water and passed 2500 cm. of almost colourless urine. She died in 1899 showing symptoms of diabetes insipidus to the last. She had three illegitimate children, father unknown, of whom one daughter died young (six months), one daughter was diabetic and one son normal. She had six children by Conrad Lachmann before marriage with him; one son diabetic, two daughters normal, and one daughter and a son died young, and one daughter was stillborn. Of the normal children, the son had eight normal children, one daughter married and had four normal children, the other daughter had one normal illegitimate child.

IV. 12, ♀ (1831—1899), lived in Homburg; suffered from very great thirst from youth on; drank two litres from morning till evening, and as a rule awakened once a night in order to drink; urinated frequently and in large quantities, otherwise quite healthy; had two illegitimate children; one son (1855—1884) normal, unmarried, now dead, cause of death unknown, one daughter diabetic. V. 29, ♀ (1859—), exhibited symptoms of great thirst at two years of age, wakened three or four times a night to drink. The thirst has increased with age, but the last 3—4 years has remained constant. She now wakens three and four times a night to drink, and during the day frequently drinks sometimes 1—1½ litres at once; never ill otherwise; has three children, two daughters (VI. 37 and 39) normal, one son (VI. 38) diabetic. IV. 13, ♂ (1838—), his eight children were unaffected by the disease, but two of his daughters (V. 39 and 40) were twins and two of his sons (V. 42 and 43) both idiots, were confined as such; all his grandchildren were normal. IV. 15, ♂ (1841—), affected from childhood and has enormous thirst. Wakens every two hours at night and drinks two litres of water every time. Thirst worse by night than day. By day he drinks 1—1½ litres of water every two hours. Since his 20th year the symptoms have slightly abated. Otherwise he is perfectly healthy. He suffered from great distention and distress of the bladder. Twice passed in Prof. Weil's presence two litres urine. In 12 hours (from 6 a.m. to 6 p.m.) he drank seven litres of water and passed about eight litres urine. Dr Alfred Weil says he drank 15 litres a day. He married and has nine children: four diabetic sons, one diabetic daughter, and four normal daughters; one of the normal daughters unmarried, in lying-in-hospital. V. 44, ♂ (1863—), tailor, brought to hospital with typhoid, was under Weil's care; while fever lasted, showed no trace of diabetes, but during recovery drank on an average nine litres of fluid in 24 hours, and passed ten litres urine, otherwise condition normal; told Weil he had suffered from polydipsia, etc. from childhood. V. 49 normal; stated to be free from diabetes; husband (?) presumably normal, had seven children: four normal daughters, two normal sons, and one diabetic son. This son (VI. 72) is the only case where disease has missed a generation in this family. V. 50, ♂ (1869—), normally developed, intelligent, healthy, had showed symptoms of the family illness for 1½ years; drank hourly by day and five or six times by night ½ to 1 litre of water; passed 5—6 litres of urine by night; suffered from distention of bladder; later went to America. V. 52, ♀ (1871—), healthy, had had no illness, inner organs normal; has an enormous thirst, drinks almost hourly by day and five times by night ½ litre or thereabout; passes 5—6 litres of uriue a night; suffers from distention of bladder; had three children: two normal, one affected; went to America. V. 53, ♂ (1873—1874), said by his father to have had diabetes. V. 54, ♀ (1875—), normal, except for extra finger which the father cut off. Went to America. V. 55, ♂ (1877—), suffers from hernia of right groin. According to his sisters and father, drank ½ litre water 20 times a day and five times a night; passed 3—4 litres urine at night, a typical diabetic. Had an illegitimate diabetic son (VI. 78), born in 1904, one normal son (1905), and a third son, born 1906, whose case is doubtful (VI. 80). III. 8, ♂ (1810—1855), healthy, married. Of wife no statement is made, had five children: one stillborn son and one daughter died young, three sons free from diabetes, one died of "consumption" (tuberculosis). IV. 29, ♂ (1849—), normal, married healthy wife, had five children. Parents say that the three eldest children from two years of age frequently drank, and awakened often in the night. The children denied this and Weil after examination believed the children were in the right, and consequently they are entered as normal. Weil says if they were diabetic, it was an extremely mild form of the disease, not the least resembling that of the other members of the family. III. 11, ♀ (1816—1903), married, said she was never ill in her life. As long as she could remember suffered from great thirst, especially from 18—20 years of age, then she wakened four or five times per night. An abatement of thirst was observed between 56—58, then drank four litres water and wakened twice or three times at night to drink. In 24 hours drank 5400 c.cm. of water and passed about five litres urine. Had eight children, the two eldest before marriage: two sons and two daughters were diabetic, one daughter and three sons normal: the normal daughter was married and had one normal daughter. Of normal sons, two died young, viz. IV. 39 and 40. III. 11 herself died at 87. IV. 31, ♂ (1840—1883), died of stomach complaint, probably ulcus ventriculi simplex. As soon as he was weaned he drank water frequently. The thirst increased to his 15—20th year. According to his widow he wakened five or six times a night and drank about a litre. In the nine years of his illness his thirst remained unchanged till the last three days. He had six children: two normal, four diabetic. V. 88, ♂ (1870—), drank at three-quarters of a year old, when unweaned at end of first year drank ½ litre by night. Later drank ten times daily and four or five times nightly ½ litre water and passed 600 c.cm. urine at a time. As a child was normally developed, the testicles very small, the right situated in the canal of the groin. Had syphilis as child. (Weil thinks the syphilis was not inherited but got from infection, says they lived in such cramped quarters he could easily have taken it from adults.) He has two children: one normal daughter, one diabetic son. V. 91, ♀, free from diabetes, suffered from syphilis in childhood, married and has one normal son. V. 92, ♂ (1875—1901), diabetes was observed from nine months old. Although the mother had plenty of milk the child was always thirsty. The symptoms increased till he drank ½ litre eight or ten times a day and every two hours at night, and passed urine as frequently. As a child he passed 400 c.cm. urine at a time. He also had syphilis, died of lung disease (tuberculosis ?). V. 94, ♀ (1877—), the diabetes was noticed at age of six months, required water as well as

the mother's milk. As a child drank 200—300 c.cm. water, and wakened two or three times a night to drink ½ litre and passed 400 c.cm. urine at a time. She married and has two children: one son undoubtedly affected, one daughter probably affected. V. 95, ♀ (1881—), showed symptoms of diabetes at four months old. As a child, according to the mother, drank 200—300 grammes of water three to four times at night. By day drank water hourly and passed 150 c.cm. urine at a time. IV. 33, ♂ (1842—1902), went to America and died there. Suffered from diabetes from two years of age, otherwise perfectly healthy. (Information from mother and sisters.) IV. 37, ♀ (1847—), according to mother and sisters she suffered from intense diabetes from her earliest years. She was the only one of the family who gave information reluctantly or refused to give it. The information therefore was obtained from the police. She refused examination of urine. She maintained she had only suffered from polydipsia and polyuria for a few years, and had now given up drinking water. She had four illegitimate children: two diabetic sons, one diabetic daughter and one normal daughter who died young. V. 98, ♂ (1870—), suffered from increasing thirst since earliest years; drank ½ litre water about ten times per day and three times per night, otherwise perfectly healthy, internal organs normal. Bladder reached above navel. As a child he drank three litres water from 8 p.m. to 8 a.m. and passed four litres urine. He had three children: one son severely affected, and one normal son, and one daughter whose case is still doubtful. V. 101, ♀ (1872—), very severely affected, otherwise healthy. As a child drank about 800 c.cm. ten times daily and three times nightly, and passed 500 c.cm. urine at a time. From 8 p.m.—8 a.m. she drank four litres water and passed 4500 c.cm. urine. Bladder a little below navel. Married, and has two children: the elder the mother thinks is a water drinker, younger only four weeks old. V. 102, ♂, severely affected; married, has one normal daughter. IV. 41, ♀ (1854—), since youth has drunk much water, but condition remains constant. She now drinks about two litres water during the night and wakens two or three times. By day she drinks ½ litre per hour, passes urine 4—5 times a day and 2—3 times a night. Married, and has three normal children: one boy and two girls. Taking the last generation together we have: VI. 38, ♂, affected from earliest youth. VI. 56, ♀, died of diphtheria, aged seven. Polydipsia symptoms disappeared after diphtheria began. VI. 60, ♂, died of diphtheria, aged nine. Affected with diabetes insipidus. VI. 61, ♀ (1897—), affected. VI. 62, ♀ (1900—), affected. VI. 72, ♂ (1897—1900), had diabetes insipidus, only case where disease has missed a generation. The child was not seen by Weil as it was dead. Died at three years of age. The history however given by the mother was very definite. There are also cases of missing a generation in Gee's pedigree (Fig. 14). VI. 75, ♂, affected. VI. 78, ♂, illegitimate, affected. VI. 80, ♂, doubtful whether affected or not. VI. 109, ♂, affected. VI. 111, ♂, undoubtedly affected. VI. 112, ♀, affection probable. VI. 114, ♂, affected. VI. 116, ♀, doubtful. VI. 117, ♀, mother thinks she is affected. VI. 118, ♂, doubtful, only four weeks old. Weil says the general constitution of the family has been excellent. No trace of insanity (except the two idiots) or neurosis. Two cases of tuberculosis. The only trace of desire for spirituous liquor was in III. 15 (?), and that was only periodic and in a moderate degree. Most of the deaths in the family were either of very old people or of children in the first years of life.

Fig. 2. *Lauritzen's Case.* II. 3, ♂, husband of II. 4, was normal and strong—although up to his 20th year he had nocturnal incontinence of urine which disappeared when he commenced cohabitation. He was not affected with diabetes insipidus and died aged 76. II. 4, ♀; disease originated in this individual, according to statement of her brother, only member of this generation who was alive. He was quite definite that of the three children of this generation she was the only one affected and was affected all her life with polyuria and polydipsia, ultimately dying at 61—her parents were unaffected. She was normal in appearance but affected with intense thirst day and night and suffered from nocturnal enuresis. The brother stated there was no arthritis, lithiasis, neurosis or syphilis in the family. She married and had eight children. III. 2, ♀, afflicted from childhood with thirst and polyuria and also urination through the night, but this disappeared when she became pregnant. She had one diabetic illegitimate son, of whose father nothing was known. She died at 23 from result of an injury to her side. III. 3, 4, 5, 6, ♂, died before they were one year old, it is not known whether they were affected or not. III. 7, ♂, suffered from polyuria, polydipsia, and nocturnal enuresis, the latter ceasing at the age of 19 on cohabitation. He had four children, of whom three were normal. III. 9, ♀, unaffected, died of scarlatina, aged 11. III. 10, ♂, severely affected, had three children, of whom two were normal. IV. 1, ♂, affected from childhood, had also nocturnal enuresis which ceased after cohabitation was begun. IV. 3, ♀, affected, drinking up to 18,850 c.c. per day, and passed as much as 13,160 c.c. IV. 7, ♂, has suffered since birth. V. 1, ♂, evidently affected though only a year old, requires water in addition to milk, polyuric. V. 2, only two months old, so far normal.

Fig. 3. *Pain's Case.* II. 2, affected, died at an advanced age; no statement as to her sibship. III. 1, 55 years of age; a tailor by trade and of poor physique, no offspring, affected with diabetes insipidus from infancy. III. 2, 46 years of age. Polyuric till thirty, but of late years he was much less afflicted; married II. 3, a woman of healthy constitution. III. 4, a carpenter, of good constitution, but

polyuric and polydipsic from birth. He could easily drink several litres of wine, and died suddenly at 35, after an excessive drinking bout. II. 5 and 6, normal, no particulars. IV. 1, normal and of strong constitution. IV. 2, aged eight, polyuric since birth, and suffered from scrofulous glands of neck. IV. 3, aged five, polyuric since birth, of weakly constitution and suffering from chronic kerato-conjunctivitis. IV. 4, age not stated, feeble, puny, very scrofulous, with tubercuIous dactylitis, arthritis of elbow; operations performed for relief of bone disease had no effect on the polyuria, which was more severe than in other members of the family.

Fig. 4. *Orsi's Case.* Six persons in the same family affected by polydipsia. The family lived in Tromello and consisted of nine persons, viz. the parents, four sons and two daughters, and the wife's brother. The father (II. 1) is about 50, and presumably healthy. The mother (II. 2), aged 48, of robust health, has had eight pregnancies, during which polyuria and polydipsia existed to a considerable degree (4 litres a day on an average). Her brother (III. 3), while working in the country, could drink 2—3 litres of water or even six it is said. He perspired freely like a normal person. His death at the age of 63 was from some unknown cause. III. 1 was a soldier 25 years of age, well built, drinks 5—6 litres of water *per diem* and passes as much urine. III. 2, aged 19, is a labourer. He first showed symptoms of diabetes in 1880, about the time that he was suffering from malaria and dysentery. Polydipsia very marked, the patient consuming and passing from 15—16 litres of water a day, which continued unabated at the time of his discharge from the hospital. III. 3 and III. 4, not referred to further.

Fig. 5. *Lacombe's Case.* II. 2, said to be affected by her son (III. 2). II. 3, said to have suffered from intense thirst from infancy by III. 2 and III. 8. III. 2, 59 years old, born at Chatellenaut near Dijon; says his parents died at an advanced age, that his mother, two brothers, a sister, his uncle and some of his uncle's children were all affected. He himself suffered from thirst from earliest infancy. At the age of 30 drank 20—25 litres in 24 hours, a litre an hour. At night he drank less, wakened four or five times a night. Now he drinks four litres of water in 24 hours and wakens 3—4 times a night to drink and urinate. Is married, no children. III. 3 and 4, both affected, killed in battle. III. 5, affected from earliest years. III. 6 and 7, affected; stated by III. 8 to have suffered from intense thirst, now both dead. III. 6, died of cholera; III. 7, of pulmonary phthisis. III. 8 and 9 both normal.

Fig. 6. *Clay's Case.* I. 1, 2, 3, 4, all remained healthy to old age and none of their children were affected, but the number of their children is unknown, or whether any of them, except II. 2 and 3, had descendants. II. 2, ♂, healthy, but was injured by an accident. II. 3, ♀, healthy, but her children were highly tuberculous, although there was no tuberculosis on either her side or her husband's. She had 15 children. III. 1, died of phthisis at 13. III. 2, ♀, began to be thirsty at age of nine, urine colourless, sp. gr. 1001. Passed about 20 pints in 24 hours.

	Average daily amount taken	Average daily amount urine
1st week	18 pts. 6 oz.	16 pts. 5 oz.
2nd ,,	13 ,, 5 ,,	13 ,, 2 ,,
3rd ,,	14 ,, 2 ,,	14 ,, 2 ,,
4th ,,	15 ,, 5 ,,	16 ,, 3 ,,
5th ,,	18 ,, 6 ,,	21 ,, 4 ,,
6th ,,	17 ,, 6 ,,	21 ,, 15 ,,

III 3, healthy. III. 4, died at three weeks old, cause unknown. III. 5, died of phthisis at 15 months. III. 6, still-born. III. 7, healthy. III. 8, ♂, began to be thirsty at age of nine, drinks from 4 pts. 4 oz. to 5 pts. 11 oz. Urinates 4 pts. 4 oz. to 6 pts. 7 oz. III. 9, still-born. III. 10, has good health. III. 11, ♂, aged nine, symptoms just begun, drinks about five pints, passes about four pints. III. 12, 13, 14, triplets, one was still-born, two died of consumption, aged nine months. III. 15 died of consumption, aged nine months.

Fig. 7. *Deebrey's Case.* I. 2, affected. II. 1, soldier, 24 years old, who passed enormous quantities of urine—25 litres in 24 hours. His mother (I. 2), two sisters (II. 2 and 3), and one brother (II. 4) were all similarly affected and were all dead. No record given of their ages or order of birth. The soldier himself was frequently attacked by "fièvre palustre."

Fig. 8. *M'Ilraith's Case.* I. 1, ♂, died of paralysis, over 60, but had no polyuria. I. 4, ♀, died of cancer, otherwise normal. II. 1, ♀, died of paralysis, over 60, no polyuria. II. 2, ♂, died of paralysis, over 60, no polyuria. II. 3, ♂, died of paralysis, over 60, no polyuria. II. 4, ♂, died of apoplexy, aged 74, no polyuria. II. 5, ♀, only slightly affected, married, and had four affected, three unaffected children. II. 6, ♀, only slightly affected, no children. II. 7, ♀, died of cancer, no polyuria, no children. II. 8, ♀, died of phthisis, no polyuria, no children. II. 9, ♂, severely affected, no children. III. 2 ♀, slightly affected, married, fairly healthy husband (III. 1), and had three affected and two normal

children. III. 3, 4, 5, ♂, died of wasting, aged five, seven and eleven months, all had excessive thirst and polyuria. III. 6, died of phthisis, aged 16, unaffected. IV. 1, 2, 3, ♂, aged 17, 14, 9, all affected. IV. 4, ♀, died of diphtheria, unaffected. IV. 5, ♀, alive and healthy, unaffected.

Fig. 9. *Reith's Case.* I. 1, affected. II. 1, affected. II. 2, aged 24, stated to have suffered from polydipsia and polyuria for nine months. It was noted her father and brother had suffered in same way; nothing said of any other relatives.

Fig. 10. *Wachsmuth's Case.* I. 1 and 2, noted to have suffered from diabetes insipidus from infancy; nothing said of other relatives.

Fig. 11. *Knöpfelmacher's Case.* I. 1, ♂, presumably normal. I. 2, ♀, affected, had one son who was affected. It is not known whether she had other children or not. II. 1, ♂, affected, married presumably normal wife, had one affected son, may have other children, but it is not known. III. 1, ♂, affected, had two affected children, nothing known of his wife. IV. 1, affected slightly since birth, now 12 years old. IV. 2, eight years old, affected, had also intense ichthyosis congenita.

Fig. 12. *Sasse's Case.* Family living at Fischenich near Cologne. I. 3, on the authority of II. 2, stated to have been a "water drinker." II. 2 and II. 3, both affected with excessive thirst. III. 1, aged 22, married. Suffered from convulsions in youth, but was healthy up to age of 21. He gave the origin of his trouble as due to drinking a large quantity of water after hard work. Quantity drunk *per diem* as much as 10—14 litres. Under observation he passed quantities of water up to 6900 c.c. and of low specific gravity. He had a child who died of convulsions. III. 2, 3, 4, 5, sex and order of birth not given, but stated to be "water drinkers."

Fig. 13. *Gee's Case.* II. 1, not actually examined by Gee, but he was reputed to have been a great sufferer and to have frequently become faint if unable to quench his thirst. In adult life his customary draught was 2—3 quarts of water at a time. Two gallons were regularly provided for his supply through the night. He became paralysed at 28 and died at 41. II. 3, a brother, was afflicted in like manner. III. 2, afflicted like her father, but excessive symptoms have abated in her old age; had nine children, a son and a daughter inheriting the disease, two other daughters themselves unaffected transmitted it to their offspring. III. 4, unaffected, married and had many children, all of whom escaped with one exception (IV. 8), a son. IV. 1 and 2, affected, likewise IV. 8. V. 1, affected, but less as he grew older. V. 3, 4, 5, 6, all severely affected from birth. V. 3 cried for many hours after birth and could only be comforted by a drink of water, the mother suspecting that he had inherited the family malady. He died in six months, his thirst being marked to the last. V. 4, died at four months. V. 5 and V. 6, now eight and nine years respectively, have suffered from unquenchable thirst since they were born.

HEREDITARY MALFORMATIONS OF THE HANDS AND FEET.

By Thomas Lewis, M.D., D.Sc., M.R.C.P.

SECTION II α. HEREDITARY SPLIT-FOOT.

(Containing an abstract of six families (Plate III, Figs. 14 and 15; Plate IV, Figs. 16, 17 and 18; Plate VI, Fig. 37), and illustrated by Plate A, Figs. 1 and 1^{bis}, 2 and 2^{bis}.)

Hereditary split-foot is a gross deformity of the extremities, which has a marked tendency to hereditary transmission. A definitive description of it is difficult or impossible owing to the remarkable variation presented by the separate deformed individuals of the same family. The fundamental, or most constant, lesion appears in the form of cleft feet, a lesion which is symmetrical as regards the two sides of the body. The clefting falls at the second or third digit and is associated with defect (*ectrodactylism*) of the corresponding bones. The portions of the foot separated by

the cleft are each welded by skin or bony union into a single mass, conical in form and terminating in one or more distorted digital points. The foot is short and broad. The variation in the degree of the defect is wide in its range; it may involve at least four toes and may spread to the tarsus and even to the bones of the leg. Those digits are most affected which lie towards the central cleft, and the defect is in a distal bone before it is in a proximal one. The hands, which have never been recorded as affected in the absence of foot lesion, show one of two main types of deformity. Either it is analogous to the foot lesion, the defect falling mainly upon the centre of the hand, or it affects the pre-axial border of the hand. As a rule the phalanges of at least two or three digits are absent; in rare cases the carpus is involved. The variation in the hands is even wider than in the feet; and though symmetry is the rule, it holds good in smaller measure than is the case in the feet. In the presence of foot malformation, one or both hands may escape entirely. The hands may show polydactylism; and syndactylism is the rule, either by bone or by soft tissues, in remaining digits which are adjacent. The heads of the metacarpals may be joined by regular cross-bones, which articulate with them.

As a whole the deformity shows segregation in high degree, no undoubted case having as yet been recorded of its missing a generation to re-appear in the next. It has been reported in association with cleft palate. As a rule the deformed offspring, of a deformed parent, outnumber the undeformed offspring.

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Hereditary Cases.

The following six families are the chief recorded examples of hereditary split-foot. The complexity of the defects necessarily renders imperfect the account here given. Those interested should refer to the original memoirs. The description of the feet

in this account does not include a statement of the presence of clefting of the foot in each case. It may be assumed, where bony deformity of these extremities is stated, to be present or is understood. In the trees illustrating the following families the mates are inserted as normal, where no statement is made in the original account.

Fig. 14. *Pearson's Case.* I. 1 and 2; II. 3 and 4; V. 26, 27 and 28, condition of extremities unknown. III. 10 and 13; IV. 4, 5, 7, 9 and 14; V. 7 and 15, *hands and feet* deformed. II. 2, Ann J., *hands*, one digit on each; *feet*, digits 1 and 5 on each. III. 3, *hands*, perfect; *feet*, digits 1 and 5 present. III. 6, *right hand*, two fingers; *left hand*, one finger and thumb present; *feet*, digits 1 and 5 present. IV. 21, *hands*, one digit on each; *feet*, digits 1 and 5 present. IV. 22, *right hand*, supernumerary little finger; *left hand*, distorted digits; *feet*, digits 1 and 5 present. IV. 23, *hands*, one digit only; *feet*, digits 1 and 5 only. IV. 24, *right hand*, digits 4 and 5 alone present and syndactylised; *left hand*, two bent fingers and a thumb present; *feet*, digits 1 and 5 present. IV. 26, *right hand*, two digits only; *left hand*, two fingers and thumb; *feet*, digits 1 and 5 present. IV. 27, *hands*, one digit on each; *feet*, digits 1 and 5 present. V. 4, *hands*, only digits 4 and 5 present and these syndactylised; *feet*, digits 1 and 5 only. V. 5, *hands and feet*, only digit 5 present. V. 6, *right hand*, only digit 5 present; *left hand*, only digits 1 and 5 present; *feet*, digits 1 and 5 present. V. 19 and 20, *hands*, deformed; *feet*, only digit 5 present. V. 22, *hands and feet*, only digit 5 present. V. 23, *hands*, deformed; *feet*, digit 5 only. V. 24, *hands*, one digit only; *right foot*, digits 1 and 5 present; *left foot*, digit 5 only. V. 25, *hands and feet*, deformed. The following table gives the detailed deformities of IV. 21, V. 22 and V. 24:

Hands	Mother IV. 21 Age 37		Elder Daughter V. 22 Age 10		Younger Daughter V. 24 Age 2½		Feet	Mother IV. 21		Elder Daughter V. 22		Younger Daughter V. 24	
	Right	Left	Right	Left	Right	Left		Right	Left	Right	Left	Right	Left
Scaphoid ...	+	+	•	+	•	•	Calcaneum ...	+	+	+	+	+	+
Lunar ...	+	+	+	+	•	•	Talus	+	+	+	+	+	+
Pyramidal ...	+	+	+	+	+	+	Navicular ...	+	+	+	+	+	+
Pisiform ...	+	+	•	•	•	•	Intl. Cuneiform	+	+	+	+	+	•
Trapezium ...	+?	+	•	•	•	•	Mid. Cuneiform	+	+?	•	•	•	•
Trapezoid ...	+	+	•	•	•	•	Extl. Cuneiform	+	+	•	•	•	•
Magnum ...	+	+	+?	+	+	+	Cuboid ...	+	+	+	+	+	+
Unciform ...	+	+	+	+	+	+	Metatarsal 1 ...	+	+	•	•	+	•
Metacarpal 1	•	•	•	•	•	•	„ 2 ...	•	•	•	•	•	•
„ 2	+	+	•	•	•	•	„ 3 ...	•	•	•	•	•	•
„ 3	+	+	+	+	+?	+	„ 4 ...	•	•	•	•	•	•
„ 4	+	+	+	+	+	+	„ 5 ...	+	+	+	+	+	+
„ 5	+	+	+	+	+	+	Digit 1 ...	+	+	•	•	+	•
Digit 1	•	•	•	•	•	•		2 ph. fused	2 ph.			1 ph.	
„ 2	•	•	•	•	•	•							
„ 3	•	•	•	•	•	•	„ 2 ...	•	•	•	•	•	•
„ 4	•	•	•	•	•	•	„ 3 ...	•	•	•	•	•	•
„ 5	+	+	+	+	+	+	„ 4 ...	•	•	•	•	•	•
	3 ph.	3 ph.	3 ph.	3 ph.	3 ph.	3 ph.	„ 5 ...	+	+	+	+	+	+
								3 ph.	3 ph.	?2 ph.	2 ph	1 ph.	2 ph.?

Oxfordshire Family. Names and addresses deposited in the Eugenics Laboratory (Bibl. No. 9, p. 69).

Fig. 15. *Lewis and Embleton's Case.* II. 2, J. G., *hands*, grossly deformed, and many fingers; *feet*, of family type. III. 2, J. H. G., *hands*, only one finger on each; *feet*, each two toes. III. 3, no information as to individual deformities. III. 7, Mrs P., *hands*, perfect; *feet*, two toes on each. IV. 1, 18 and 30 (miscarriages), the first two deformed, the last doubtful. IV. 3, H. C. G., *right hand*, syndactylism of digits 4 and 5; *left hand*, extra finger attached to metacarpal 1; *right foot*, deficiencies of digits 2 and 3; middle cuneiform absent; syndactylism; *left foot*, similar, but misses terminal phalanx of digit 4. IV. 7, 10, 11, 12, 13 and 21, deformed in *hands and feet*; no details. IV. 9, W. G., *hands and feet*, said to have been like those of IV. 24. IV. 17, M. A., *right hand*, digits 1 and 2 almost completely absent; cross-bone joins heads of metacarpals 4 and 5 and articulates at outer end with the single set of phalanges present; *left hand*, digit 1 almost completely absent, and phalanges of 2 and 3 quite absent; remaining phalanges syndactylised to form cross-bone; *right*

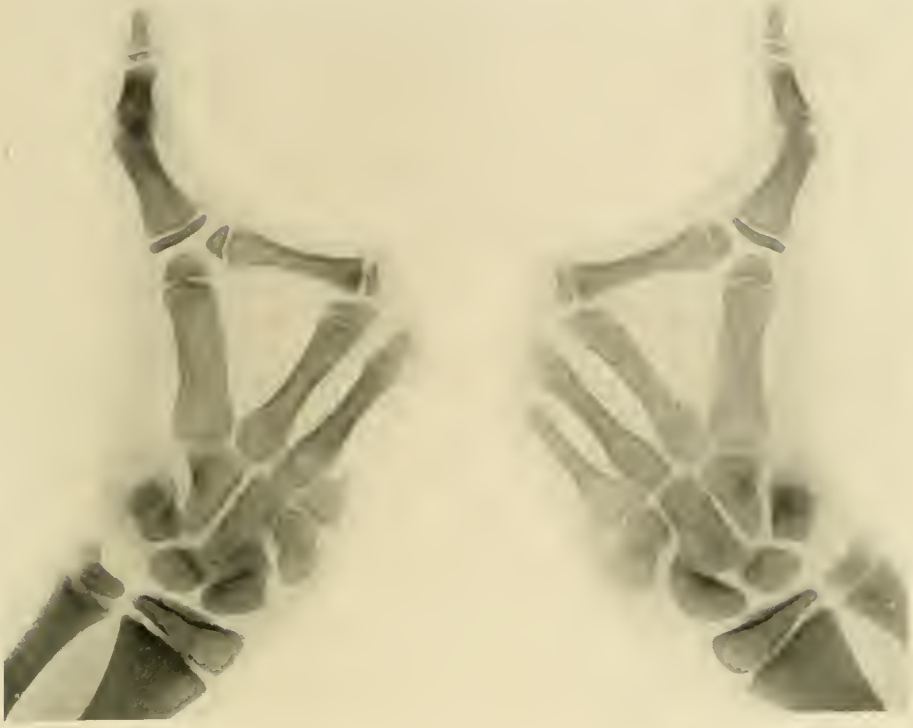


Fig. 1. Skiagram of the hands of R.E.G. (Family No. 15, V. 41). Hereditary Split-foot. The hands are seen from the dorsal aspect.

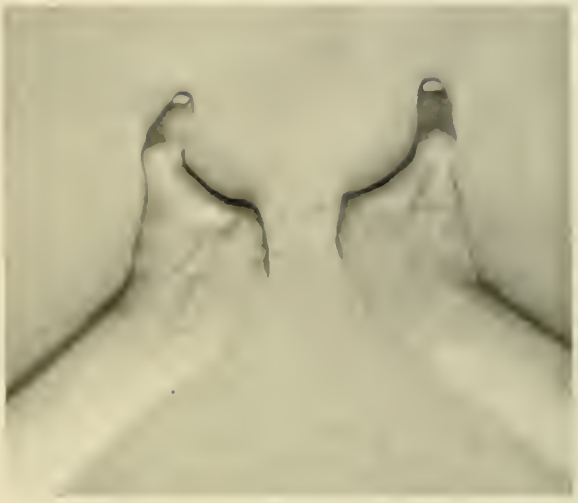


Fig. 1bis. Photograph of hands of R.E.G.

Fig. 2. Feet of R.E.G. (from dorsal aspect).

Fig. 2bis. Photograph of feet of R.E.G.

The figures on this plate are reproduced by kind permission from *Biometrika*, Vol. VI., Plates II. and III.

foot, the three central digits show gross defect; *left foot*, similar. IV. 23, E. G., *hands*, defect falls mainly on digits 2 and 3; 4 and 5 syndactylised; *feet*, the main defect appears to fall on digit 2; the phalanges of 3 and 4 are absent also. IV. 25, I. G., *right hand*, digit 1 practically absent; phalanges of remaining digits defective, the defect decreasing towards the ulnar side, where there is syndactylism; *left hand*, very similar; *feet*, only digits 1 and 5 are perfect; 2 is almost completely absent, and 3 and 4 partially absent on the right side; the arrangement is similar on the left. IV. 31, E. P., *hands*, main deformity falls on digits 1 and 2; 3 lacks phalanges; 4 and 5 are complete and syndactylised; *feet*, digits 1 and 5 alone perfect. V. 6, A. S., *right hand*, slight defect of digit 2; digit 1 had two sets of phalanges; *left hand*, metacarpal of digit 1 bears a set of three phalanges, and shows scar where another set was removed; *feet*, digits 1 and 5 and remains of 4 are alone present. V. 7, H. C., *right hand*, digit 1 has an extra phalanx; defect of terminal phalanx of 3; *left hand*, digit 1 lacks phalanges; 4 has two sets, which are syndactylised to adjacent fingers; *feet*, similar to those of V. 6. V. 18, *hands*, similar to those of IV. 25; *feet*, each two toes. V. 24, W. H. A., *hands*, extremely complex deformity, combining ectrodactylism, syndactylism and probably, in one hand at least, polydactylism; cross-bone; *feet*, digit 5 only. V. 25 (miscarriage), one digit on each extremity. V. 27, L. A., *right hand*, minor defects and syndactylism; *left hand*, defect of phalanges of digits 1 and 2 and attempt at polydactylism; *feet*, toes 1 and 5 complete, 2 absent, 3 and 4 very imperfect. V. 28, H. A., two digits on each extremity. V. 31, J. A., *right hand*, digit 1 absent; digits 2, 3 and 4 show defective phalanges; *left hand*, similar, but phalanges of 4 also present; *right foot*, digits 1 and 5 alone present; *left foot*, digit 5 only. V. 36, M. A., *hands*, one finger on each alone perfect; *feet*, each two digits. V. 38, J. A., *right hand*, digit 1 lacks phalanges; digits 2, 3 and 4 have four sets of phalanges between them, all syndactylised by skin; *feet*, digits 1 and 5 alone perfect. V. 39, E. W. G., *right hand*, like the left of V. 41; *left hand*, similar; *feet*, digits 1 and 5 alone perfect; remains of 2 and 4. V. 40, E. M. G., *right hand*, absence of digit 1, and phalanges of 2; digit 3 has a short phalanx; remaining phalanges are distorted in an extraordinary manner, and a cross-bone is present; *left hand*, digit 1, and phalanges of 2 and 3 almost completely absent, 4 and 5 normal; cross-bone present; *feet*, digits 1 and 5 perfect, also metatarsal of 4 is present. V. 41, R. E. G., *hands*, very similar; the hands are shown in Plate A, Figs. 1 and 1bis; *feet*, alike; shown in Plate A, Figs. 2 and 2bis. V. 42, J. T. G., *right hand*, digit 1 and phalanges of 2 and 3 absent; cross-bone between the heads of metacarpals 3 and 4; *left hand*, phalanges of 2 absent; digit 3 has a half phalanx only; *feet*, digits 1 and 5 perfect; remains of 3 and 4. V. 45, L. V. G., *right hand*, similar to right hand of V. 42; *left hand*, similar; metacarpal 1 is also represented; *feet*, digits 1 and 5 perfect; remains of 3 and 4. V. 46 (miscarriage), deformity doubtful. V. 47, K. G., *right hand*, digit 1 and phalanges of 2 and 3 markedly defective; 4 and 5 are syndactylised; *left hand*, digit 1 has extra phalanx; digit 2 has two phalanges; *feet*, digits 1 and 5 perfect; some remains of metatarsals 2, 3 and 4. V. 48, W. G., *right hand*, digit 1 and phalanges of 2 and 3 markedly defective, 4 and 5 syndactylised; *left hand*, similar; *feet*, digits 1 and 5 complete; remains of digits 2 and 4. VI. 1, M. A. S., *right hand*, had an extra thumb; *left hand*, minor defect; *feet*, each had two toes. London Family. The names and addresses of the members of this family are deposited in the Royal College of Surgeons' Library (Bibl. No. 5, p. 26).

Fig. 16. *Parker and Robinson's Case.* II. 9, S. F., *right hand*, digit 1 completely and 2 and 3 partially absent; syndactylism of 4 and 5 by skin; *left hand*, digit 1, and phalanges of 2, absent; 3 and 4 joined by bony syndactylism; *feet*, said to be like those of IV. 7. III. 6, M. A. W., *hands*, perfect; *right foot*, digits 2 and 3 markedly deficient; syndactylism of soft tissues of digits 4 and 5; *left foot*, very similar. III. 8, E. H., *hands*, perfect; *feet*, said to be like those of IV. 7. III. 10, S. F. (twin) and III. 11, A. F., *hands and feet*, said to be like those of IV. 7. III. 12, T. F., *hands and feet*, deformed. III. 15, J. F., *feet*, deformed. III. 16, G. F., *right hand*, deficiencies of digits 1 and 2; syndactylism of 3 and 4; *left hand*, deficiencies of digits 2 and 3; syndactylism of 3 and 4; *feet*, almost complete absence of digits 2, 3 and 4. III. 19, S. F., like IV. 7. III. 20, H. F., *hands and feet*, deformed. IV. 2, A. W., *right hand*, defective nail, and displaced phalanx; *left hand*, presumably normal; *right foot*, digits 2 and 3 markedly deficient, bony terminal syndactylism of digits 4 and 5; *left foot*, the same, but digit 3 is less defective. IV. 4, H. W., *hands*, perfect; *right foot*, digits 1 and 2, and digits 3 and 4, syndactylised; *left foot*, digits 3 and 4 united; presumably no bony defect. IV. 6, *feet*, like those of IV. 7. IV. 7, E. W., *hands*, phalanges of digit 2 almost absent; digits 3 and 4 show terminal bony syndactylism; *right foot*, digit 2 absent, digit 3 partially absent, 4 and 5 show bony syndactylism; *left foot*, similar. IV. 9, *hands*, perfect; *feet*, like those of IV. 7. IV. 13, G. H., *hands*, perfect; *feet*, syndactylism by skin of digits 1 and 2. IV. 16, A. H., *one hand*, perfect; *other hand and feet*, like IV. 7. London Family (Bibl. No. 7, p. 181).

Fig. 17. *Fotherby's Case.* Only IV. 2 and 5, and V. 8 were seen by the author. I. 1, II. 1 and III. 2, "claw footed and handed." III. 4, "claw footed"; *hands*, perfect. III. 5, "rest of family perfect." IV. 2, J. A., *hands*, probably, absence of metacarpal 3, and absence of all phalanges except those of digits 4 and 5, which are syndactylised; *feet*, split; digits 2, 3 and 4 absent; two outer and

two inner toes welded into separate masses, containing, in the main, bones of digits I and 5. IV. 3, *hands*, thumbs broad; two supernumerary digits (rudimentary) on outer border of each hand; *feet*, conform to family type. IV. 5, M. A., *hands*, have digit 5 only; *feet*, "as above." IV. 7, *hands*, 2 fingers on one, 6 on the other; *feet*, "as above." IV. 8, *hands*, 7 fingers on one, 6 on the other; *feet*, "as above." IV. 9, *right hand*, like those of IV. 5; *left hand*, like those of IV. 2; *feet*, "as above." IV. 11, *hands and feet*, as in IV. 5. IV. 12, *hands*, shortened in three digits of each; *feet*, three digits in one and two in the other. IV. 13, *hands and feet*, as in IV. 12. V. 3, *hands*, 7 fingers on one and 6 on the other; *feet*, "after family type." V. 4 and 5, *hands*, "variously deformed"; *feet*, "as above." V. 8, *hands and feet*, digits "represented by little fingers and toes only." London Family (Bibl. No. 3, p. 975).

Fig. 18. *Mayer's Case.* I. 2, Jesaïas, *hands and feet*, affected. II. 2, Hermann B., *right hand*, phalanges of digit 3 absent; syndactylism of 4 and 5 by skin; metacarpal 2 has an extra attached piece of bone; *left hand*, phalanges of digit 3 absent; syndactylism of 4 and 5; metacarpal 3 distorted and 4 articulates with a free knob of bone in addition to its phalanges; *right foot*, bones of digits 1 and 5 alone complete; tarsus disturbed; *left foot*, very similar. II. 3, Moritz, *right hand*, affected; *left hand*, normal; *feet*, deformed. II. 5, 7, 8 and 9, Nathan, Julius, Joseph and Bertha, *hands*, normal; *feet*, deformed. III. 2, Hugo B., *right hand*, six digits; 1 and 2, and 5 and 6 syndactylised; 3 and 4 lack phalanges, and metacarpals 3 and 4 are reduced distally; *left hand*, phalanges of digit 3 absent; remaining two groups of phalanges syndactylised; extra piece of bone between metacarpals 3 and 4; *right foot*, toes 1 and 5 alone complete and separated by usual cleft; *left foot*, similar, but metatarsal 4 present and enlarged. III. 3, Nathan, *right hand*, normal; *left hand*, digit 3 absent, with its metacarpal; 1 and 2 syndactylised; *feet*, as usual. III. 8, Berthold B., *right hand*, phalanges of digit 3 fail; gross irregularity of phalanges of 1 and 2, associated with syndactyly; *left hand*, digit 3 has two displaced phalanges; a cross-bone lies between heads of metacarpals 3 and 4; *right foot*, toes 1 and 5 alone complete; remains of other metatarsals welded; *left foot*, toes 1 and 5 present, and metatarsal 4. III. 11, Max. B., *right hand*, digit 3 absent, 4 and 5 syndactylised; *left hand*, 4 and 5 syndactylised; *feet*, similar to others. III. 12, Herbert, *feet*, alone affected. IV. 1, Frida, syndactylism alone (in which extremities is not stated). IV. 2, Julius B., *right hand*, metacarpal 2 and phalanges of 2 and 3 absent; syndactylism of 4 and 5; *left hand*, phalanges of digit 3 absent; syndactylism of 4 and 5; *feet*, toes 1 and 5 alone developed at all completely. (Bibl. No. 6, p. 20.)

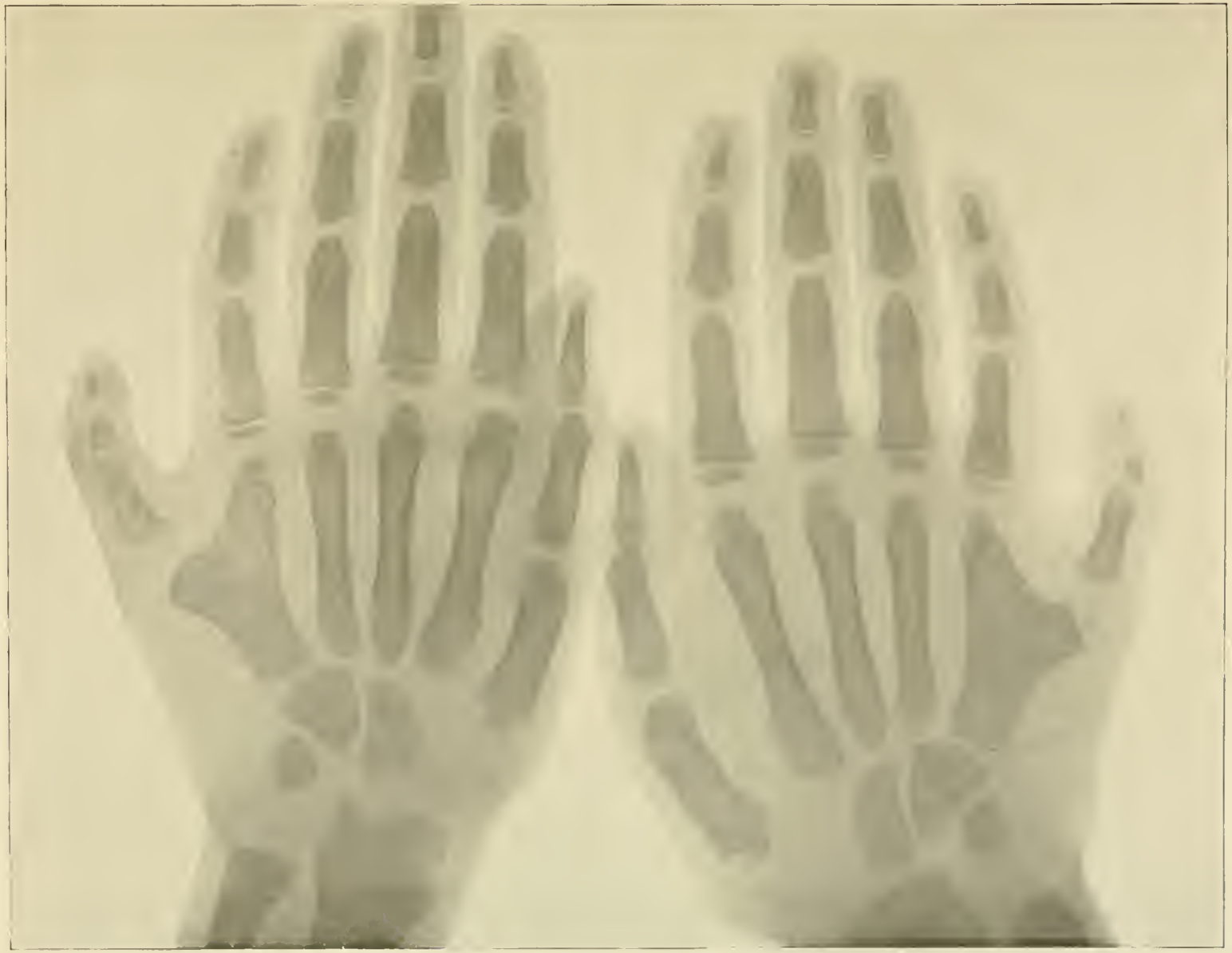
Fig. 37. *Bédart's Case.* II. 1, Auguste Faurie, *hands*, deformed; *feet*, split. III. 2, Louis Faurie, deformed *hands and feet*; III. 5, Elizabeth F., *hands and feet*, deformed. III. 7, Marie Fabre (*née* Faurie), *right hand*, digit 1 almost completely and phalanges of 2 absent, 3, 4 and 5 are syndactylised and have in common two complete sets of phalanges; *left hand*, defective phalanges of digits 1, 2 and 5; digits 3 and 4 syndactylised; *feet*, split and grossly deformed, the defect falling mainly on digits 2, 3 and 4. IV. 1, 2 and 3, one boy and two girls, all deformed. IV. 6, Marie F., *right hand*, phalanges of digits 1, 2 and 3 defective; 4 and 5 syndactylised; *left hand*, defect of phalanges of digits 2 and 3; 4 and 5 syndactylised; *feet*, of usual type. IV. 7, Nathalie F., *right hand*, defect of phalanges of digits 2 and 3; cross-bone between heads of metacarpals 3 and 4; syndactylism of 4 and 5; *left hand*, split and grossly deformed; *feet*, as above. IV. 8, Delphine F., *hands*, very similar, both right and left, to IV. 7; *feet*, of usual type. IV. 9 and 10, Clémentia and Louis F., deformed. Diagrams of deformities in original. (Bibl. No. 2, p. 367.)

SECTION III α. POLYDACTYLISM.

By THOMAS LEWIS, M.D., D.Sc., M.R.C.P.

(Containing an abstract of eighteen families (Plate IV, Figs. 19 and 20; Plate V, Figs. 21, 22, 23, 24, 25 and 26; Plate VI, Figs. 27, 28, 29, 30, 31, 32, 33, 34, 35 and 36, and illustrated by Plate B and Plate C, Figs. 1 and 2).)

Polydactylism, or the presence of extra digits, or portions of them, is found in man, horses, the monkey, dog, cat, fowl and other animals. In man it may affect one, two, three or four extremities. It is found in conjunction with syndactylism frequently, and with other malformations, such as hare-lip, ectrodactylism, hereditary split-foot, and other abnormalities of the limb bones occasionally. It may be sym-



Skiagram of an unpublished case of family polydactylism. We are indebted to Mr H. S. Clogg of Charing Cross Hospital for the figure, and for the information that the mother and several individuals in the family of the patient were similarly affected.

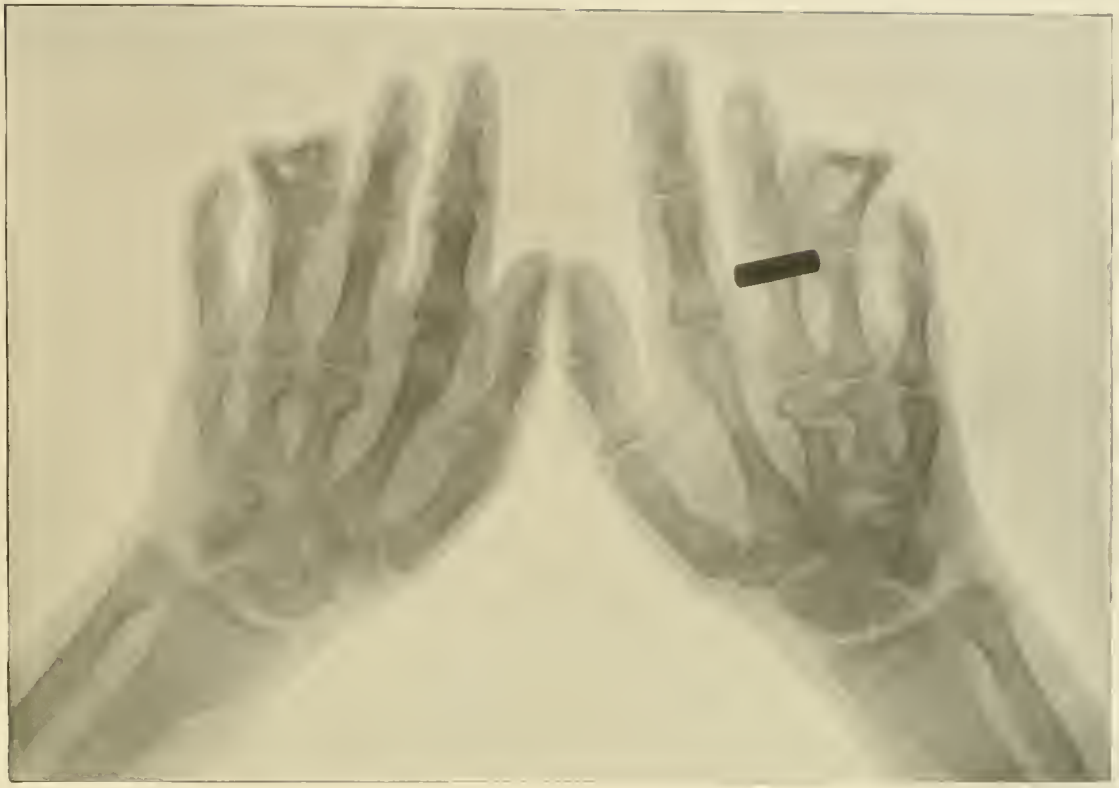


Fig. 1. Skiagram of the hands of VI. 7, Family 19. The figure, for which we are indebted to Mr Mathew of the Middlesex Hospital, shows an exceptional form of polydactylism, in that it is associated with brachydactylism. The right hand is to the left in the figure.

Fig. 2. Photograph of the hands of the same patient.

metrical or asymmetrical. It shows a marked tendency to hereditary transmission, and the type of deformity and situation of the deformity may vary widely from individual to individual in the same family. Transmission has also been noted in the lower mammals (for which consult Bateson: *Materials for the Study of Variation*, London, 1894).

In man it is most frequently post-axial (towards little finger or toe), but many varieties are known. Thus it may be pre-axial (towards thumb or great toe), in which case the hereditary tendency is probably less marked; or the reduplication may be central. Of the grades of deformity the following are the most important:

(1) Small appendages in the form of fibrous skin-clad nodules, which may or may not contain bone, and attached as a rule post-axially.

(2) Bifurcations of normal digits with complete or partial reduplication of the part. The bifurcation may be of any extent, and may originate at the distal end, middle or proximal end of either of the digital bones, or at either of the joints. In the full form there is complete reduplication of the digit, as far as carpus or tarsus. The dichotomising digit is as a rule symmetrically placed about a central line, and is usually, though not invariably, laterally disposed.

(3) Irregular and intermediate forms; for example, attachment of digits by anchylosis or joint to the side of a metacarpal or phalanx.

Cases are described in which there are 6, 7, 8, 10 or even 12 or 13 digits upon one or more extremities.

The condition has been regarded:

(*a*) As an atavistic phenomenon (Albrecht, *Centralbl. f. Chir.*, 1886, Suppl. to No. 24, S. 105).

(*b*) As the result of intra-uterine injuries (Ahlfeld, *Missbildungen des Menschen*, Leipzig, 1880, S. 106; Zander, *Virchow's Archiv*, Bd. cxxv. 1891, S. 453).

(*c*) As a result of variation in the germ plasm (Weismann, *The Germ-Plasm*, Eng. Trans. pp. 428—31, London, 1893; Förster, *Die Missbildungen des Menschen*, Jena, 1861, S. 43—4; Sutton regards it as an example of "dichotomy," *Evolution and Disease*, pp. 107, 158, London, 1890).

For further information reference may be made to the works quoted, and to the special reports from which the following genealogies have been compiled. An instance in which inbreeding occurred in an isolated village, and in which nearly the whole population became polydactylous, is given (without the desirable details) by Devay (*Arch. gén. de méd.*, 1863, Vol. I. p. 763).

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11. Wilson, Gregg. Hereditary Polydactylism. *Journal Anat. and Physiolog.* O. S. Vol. xxx. pp. 437—48, London, 1896.

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Hereditary Cases.

In the trees illustrating the following families, the mates are inserted as normal, where there is no statement in the original account.

Fig. 19. *Mathew's Case.* VI. 7 (the hands are illustrated in Plate C, Figs. 1 and 2), digits 3, 4 and 5 of both hands have shortened metacarpals; digit 4 has a bifurcated second phalanx, and this bears two terminal phalanges, the inner of which is bent so as to articulate with the outer; no other deformity present. Patient states that the malformation is exclusively confined to the ring fingers in all the individuals affected, and has been symmetrical in every case, except in that of her mother (V. 2), "in whom one hand only was deformed. There have been practically no deviations from the type as exemplified in the photographs, and the deviations (which have occurred) have always been slight." "There is no record of any member of any family possessing a split finger or toe or an extra digit." London Family (Bibl. No. 5, p. 969).

Fig. 20. *Struthers' Case.* II. 1, W., similar to IV. 3 (which hand unknown). IV. 3, B., additional thumb on left hand. Linlithgowshire Family (Bibl. No. 10, p. 92).

Fig. 21. *Lucas' Case.* In this figure the deformed are all black, whatever the degree of deformity. The full names will be found in the original.

I. 1, Mrs S. M., supernumerary digits. I. 2 described as a giant. II. 1, "six toes on each foot." III. 3, "over six feet in height"; "six toes on each foot[1]." III. 7, "hare-lip and six toes on each foot." III. 8, "six toes on each foot." III. 10 and 13, six fingers on each hand. IV. 2, 4, 6, 12, 14 and 17, nature of deformity not stated. IV. 19, 22, 25, 28, "extra toes." IV. 34, "one extra finger." IV. 35, six toes on one foot; seven toes on the other; five fingers and a thumb on each hand. IV. 40, six toes on one foot; seven toes on the other; remaining toes webbed; five fingers and thumb on one hand. V. 1, "five fingers and a thumb on each hand." V. 2, "supernumerary digits on both hands and feet, number not known." V. 5, extra toes on both feet. V. 6, hare-lip and cleft palate; web between toes 1 and 2 of each foot. V. 10, five fingers and thumb on each hand; six toes each foot; webbed toes. Suffolk Family (Bibl. No. 4, p, 417).

Fig. 22. *Smith and Norwell's Case.* IV. 11, supernumerary toes removed; hands "exactly" like the right of IV. 12. IV. 12, E. J., *right hand*, same as V. 1, but an extra nodule; *left hand*, same as left of V. 1 but third digit has no joint between phalanges 2 and 3; *feet*, same as in V. 1, but digit 1 small. V. 1, F. J., *right hand*, digits 3 and 4 syndactylised; each finger complete in bones, though there are alterations in size, shape and position; extra nodule of bone between phalanges 1 of digits 3 and 4; *left hand*, very similar, but there is a further bony nodule attached to the head of metacarpal 2; *feet*, each has six toes,

[1] For a case of polydactylism, accompanied by hereditary giantism, among the Philistines see 2 Samuel xxi. 15—22.

digits 2 and 3 are webbed, as are also 5 and 6; digit 1 large, digit 6 appears to be the supernumerary and has but two phalanges. V. 2, 3 and 4 died young, had same malformation of hands as IV. 12. V. 8, 9 have both hands exactly like the right of IV. 12. The remaining deformed individuals are indicated as such in a diagram. It is stated that all the deformed were affected in hands and feet. Scottish Family (Bibl. No. 9, p. 8).

Fig. 23. *Struthers and Wilson's Case.* I. 1, Esther L. (*née* E. P.), extra little finger on one hand. II. 2, Charles L., and III. 2 and 3, James and Thomas L., extra little fingers. III. 4, John L., one extra little toe and two extra little fingers. IV. 5, Jane L., one extra little toe and finger on both sides. IV. 13, James J., and V. 4, Maggie N., extra little finger on each hand. V. 9, "thumb of right hand reduplicated." VI. 2, "one extra little finger and the rudiments of another." Falkirkshire Family (Bibl. No. 11, p. 437 and No. 10, p. 91).

Fig. 24. *M'Kellar's Case.* VI. 4, Janet —, *hands*, symmetrically malformed; each first digit has a metacarpal and three phalanges. "Each of these thumbs has a rudimentary offshoot on the external lateral aspect, that on the right hand arising by an apparently cartilaginous connection opposite the distal joint, and consisting of two portions admitting of passive motion; that on the left is somewhat similar, but is longer, arises by a distinct and partially movable joint from the metacarpo-phalangeal articulation, and consists of three portions, distinguished by nodules at the joints, which seem to be anchylosed." *Feet*, digit 1 bifid, and webbed from metatarsal onwards. There is no further information which has not been included in the tree, except that the side of defect in VII. 4 is uncertain. (Bibl. No. 6, p. 390.)

Fig. 25. *Wilson's Case.* I. 2, S. T., "extra little toe on each foot." II. 2, 3, 4, 5 and 6, "by his first marriage he (I. 2) had a number of children, who are all said to have shown digital abnormality; and one of whom, J. T. (II. 6), is known to have had an extra minimus on each hand and an extra toe on the back of the middle digit of one of his feet"; the foot affected was the left. II. 7, "great toes reduplicated." III. 2, 3 and 4, "each of them had one small extra post-axial finger on one hand." The hand was in each case the right. IV. 1, J. I. "extra little finger," side not stated. Peebles Family (Bibl. No. 11, p. 430).

Fig. 26. *Carlisle's Case.* II. 2, G. (*née* Kendall), *hands and feet*, each had six digits. III. 2, Abigail Colburn (*née* Green), both feet and one hand had six digits. III. 3, as reported by IV. 2, there were eleven in this family, all having six digits on hands and feet. IV. 2, Abiah C., *hands and feet*, each had six digits. IV. 3, one hand and one foot had six digits. IV. 4 and 5, *hands and feet*, each six digits. V. 1, Green C., one foot and both hands bore six digits. V. 3, Zebina C., *hands and feet*, six digits on all. V. 4 and 5 (twins), David and Jonathan C., Jonathan had six digits one each extremity. V. 6, Zerah C., *hands*, each had a completely formed extra digit growing from "outside" of metacarpus (presumably ulnar side, for it is stated that there were five fingers and a thumb); *feet*, extra digit on both, growing from "outer" side of metacarpus. Zerah was exhibited for extraordinary powers of arithmetical computation. American Family (Bibl. No. 2, p. 94).

Fig. 27. *Greene's Case.* II. 1, III. 2, IV. 1, V. 4 and 8, VI. 1, extra little fingers on each hand. V. 14, extra little finger on each hand, and supernumerary pedunculated little toe on the right foot. VI. 3, supernumerary digit on the right hand; consisted of end phalanx with perfect nail, attached as a whole by fleshy pedicle, $\frac{3}{4}$-inch long, to ulnar side on a level with centre of metacarpus. (Bibl. No. 3, p. 859.)

Fig. 28. *Riville's Case.* II. 2, Gratiò Kalleia, *hands and feet*, six digits on each; the surplus fingers were bound to the index and middle digits. III. 2, Salvator K., *hands and feet*, six digits on each. III. 3, George K., *hands*, the two thumbs are longer and broader than usual, grooved in the middle and each presents the appearance of two digits bound into one; *feet*, syndactylism of digits 1 and 2 on the left side. III. 7, Marie K., like III. 3. IV. 1, 2, 5 and 6, *hands and feet*, six digits on each. IV. 3, in one part of the account, reported as having six digits on hands and feet; elsewhere it is stated that the hands and feet, "ne sont aucunement difformes[1]." IV. 7, six digits on both hands and the right foot. IV. 12, six digits on the left foot. Maltese Family (Bibl. No. 8, p. 377).

[1] The following are two extracts from the original:

(1) "Salvator...et a eu jusqu'à présent deux garçons et une fille avec six doigts aux mains et aux pieds."

(2) "Les fils de Salvator ont les mains et les pieds mieux formés et ils peuvent travailler. Je m'intéresse au mariage de sa fille qui a déjà quatorze ans, et dont les pieds et les mains ne sont aucunement difformes; je suis curieux de savoir si elle fera des enfants à six doigts, quoiqu'elle épouse un mari qui n'en ait que cinq. Si elle arrive, voilà des exemples contraires, et alors il sera vrai de dire que le principe de la génération réside dans l'un et l'autre sexe. Nous avons déjà pour première preuve Marie fille de Gratio, qui a fait un garçon avec six doigts au pied gauche, mais la fille de Salvator pourra nous fournir quelque chose de plus instructif." The writer appears to use the term "difformes," in the beginning of the second passage, in the sense that in addition to polydactylism there was no malformation of the fingers. The child is consequently marked in the tree as deformed, for there is a positive statement to this effect.

Fig. 29. *Morrish's Case.* II. 1, "six digits on each hand and foot, with bilateral webbing of the second and third toes." III. 3, six digits on each hand, with bony union to the rest of the hand; the additional finger was on the ulnar side; syndactylism of 3 and 4 also present; six toes on the left foot, webbing of digits 1, 2 and 3 on each. III. 4, *hands*, the same as III. 3; *feet*, slight webbing of digits 2 and 3. III. 5, *hands*, same as III. 3; *feet*, six toes on each; complete webbing of digits 2 and 3. III. 6, *hands*, extra digit on ulnar side in each, attached by pedicle; *left foot*, "spatulate condition of each great toe, giving appearance of a fusion of two separate toes"; *right foot*, the same, and in addition two little toes. III. 7, *hands*, extra little fingers attached by pedicles; *feet*, slight degree of webbing of digits 2 and 3. (Bibl. No. 7, p. 369.)

Fig. 30. *Withrow's Case.* II. 1 and 2, extra little toe on each foot; *hands*, normal. II. 5, *right hand*, a "jelly-like mass hanging by a mere thread of tissue" from the ulnar side of the metacarpo-phalangeal joint; *left hand*, perfectly formed extra little finger in corresponding situation; *feet*, a perfectly formed extra little toe, growing from the metatarso-phalangeal joint of each. (Bibl. No. 12, p. 558.)

Fig. 31. *Struthers' Case.* IV. 1 and 2, J. B., supernumerary little finger on outer side of left hand. Northumberlandshire Family (Bibl. No. 10, p. 88).

Fig. 32. *Struthers' Case.* IV. 11, M. O., a sixth toe on the outer side of each foot. IV. 12, J. O., six fully developed digits on hands and feet; the fifth and sixth digits rest in each case upon a single bifurcated metacarpal or metatarsal bone. IV. 17, C. O., almost identical with IV. 12. Edinburgh Family (Bibl. No. 10, p. 84).

Fig. 33. *Struthers' Case.* III. 1, sixth digit on ulnar side of one hand. III. 3, H. K., additional thumb on outer side of right hand; the extra digit is smaller and shorter than the thumb and webbed to it. Kincardineshire Family (Bibl. No. 10, p. 88).

Fig. 34. *Struthers' Case.* III. 1, possessed a double thumb. III. 7, S. M., additional thumb on right hand; it had two phalanges and a metacarpal. Argyleshire Family (Bibl. No. 10, p. 95).

Fig. 35. *Struthers' Case.* The two families here given were connected; it is stated that I. 2 and I. 3 were related to members of a separate family in which deformity existed.

I. 3, "this grandmother herself is not stated to have had the variety." II. 3, six toes on each foot, and very long thumbs. III. 4, J. H., right hand has a thumb with double first phalanx. III. 10, great toes double. III. 11, A. S., double thumbs, supported on unbifurcated metacarpal bones; one additional thumb has three phalanges; *feet*, very similar; on right side an extra metatarsal also. III. 12, W. S., *left hand*, extra finger between digits 1 and 2; it has three phalanges and a metacarpal; *feet*, extra toe between digits 1 and 2 in each. V. 1, J. D., two thumbs of equal size on each hand, each of which has two phalanges and a metacarpal; *left foot*, a double great toe; two sets of phalanges, and the metatarsal is grooved, though it shows no bifurcation. Argyleshire Family (Bibl. No. 10, pp. 93—5).

Fig. 36. *Attlee's Case.* II. 5, the child's (IV. 1) great uncle had a supernumerary thumb. IV. 1, "on each hand there was an extra digit attached to the ulnar side of the little finger by a soft pedicle; each digit had a well-formed nail." (Bibl. No. 1, p. 163.)

Fig. 37 is reported in the section on hereditary split-foot: see p. 10.

SECTION IV α. BRACHYDACTYLISM.

By Thomas Lewis, M.D., D.Sc., M.R.C.P.

(Containing an abstract of four families (Plate VI, Fig. 38; Plate VII, Figs. 39, 40 and 41, and illustrated by Plate D, Figs. 1 and 2).)

Brachydactylism, literally shortened digits, is a term which is not rigidly applied, and is employed for several grades of deformity which may or may not be separate entities. There is no collected review of the malformations, which might be classed under this heading. This account is therefore confined to one variety which has been

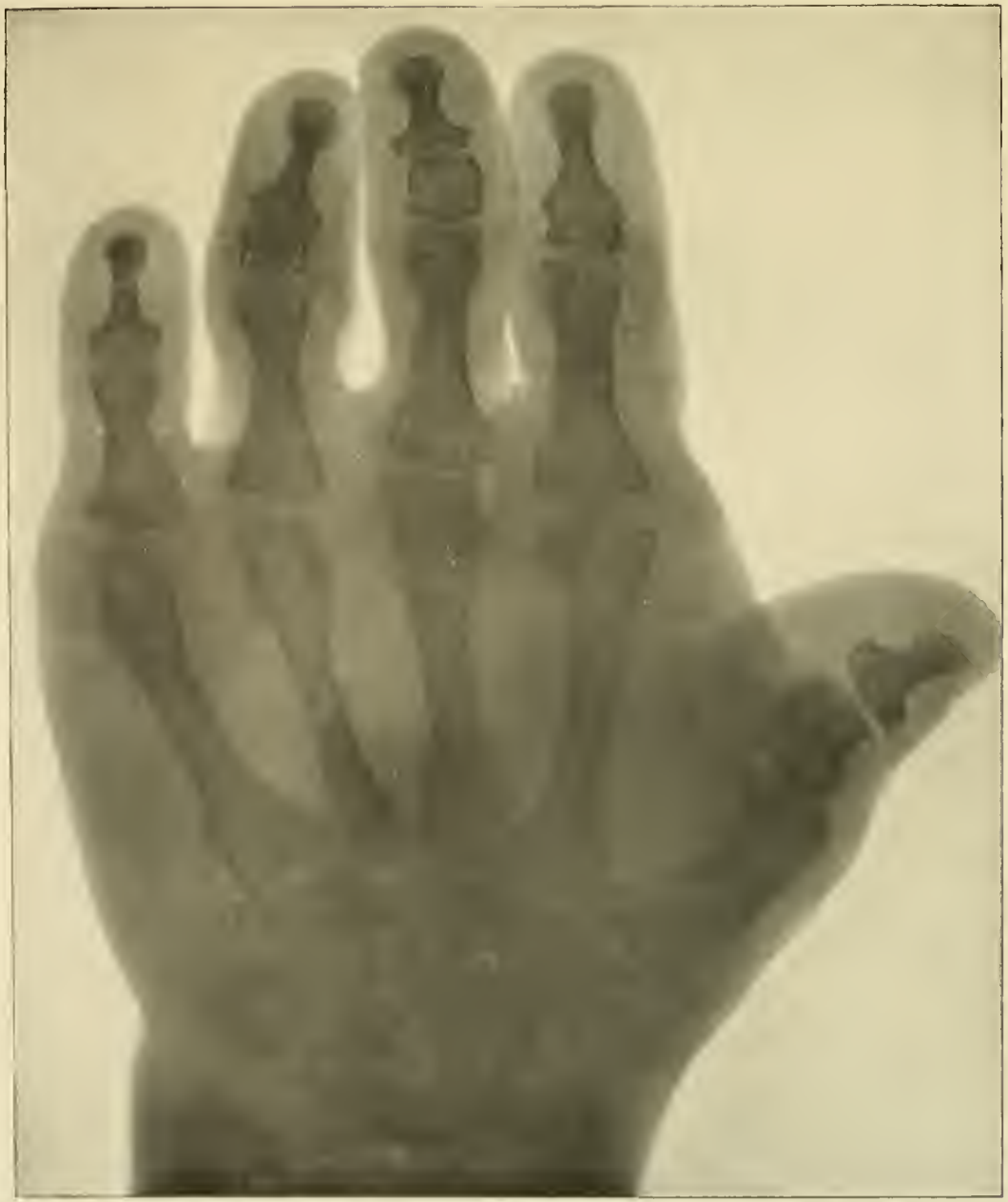

Fig. 1. Skiagram of the hands of a member of the Family 39. It shows brachydactylism in its most complete and most typical form. We are indebted for this as yet unpublished figure to Dr Drinkwater.

Fig. 2. Photograph of a hand from another member of the same family, for which we are also indebted to Dr Drinkwater.

frequently, though wrongly, termed hypophalangia. The essential feature of the deformity seems to lie in an incomplete development of the middle phalanx of all four post-axial digits on hands and feet; the phalanx is represented by a shortened and malformed bone which is more or less welded into the base of the terminal phalanx (Hasselwander), or is altogether unrepresented. Associated with the defect are seen alterations in the lengths of the bones of digit 1 (shortening or lengthening, etc.), and in some instances shortening of the long bones of the limbs, with resultant deficiency of stature. The deformity tends to be remarkably symmetrical and hereditary transmission appears to be the rule. The outward conformation of the extremities is necessarily altered, the lines of the skin are changed, the grip and manipulative power suffer.

There appears to be an interesting relationship between the mode in which shortening takes place in this deformity and the usual condition of the first digit of hand and foot in the normal condition, namely, the presence of two phalanges only and the not infrequent absence of a phalanx in the fifth digit (cp. Hasselwander). So far no family with the complete condition is on record, in which a generation was missed during the transmission.

For further details reference may be made to the original memoirs cited later, and to the well-known works on teratology, including Fort (*Des déformités congénitales*, Paris, 1869). Closely allied deformities are instanced and described by Joachimsthal (*Virchow's Archiv*, 1898, Bd. CLI. S. 429). The accompanying illustration, Plate D, of the bones exhibits very beautifully the various grades of deformity in the different fingers.

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HEREDITARY CASES.

In the pedigrees illustrating the following cases, the mates are inserted as normal, where there is no statement in the original account.

Fig. 38. *Mercier's Case.* All the deformed individuals are reported as having but two phalanges on all the fingers and toes. IV. 9, A. D. (the individual observed), had two bones in each of the fingers and toes. There was little shortening of the digits. The thumbs are stated to have consisted of two phalanges, but from the account it is not certain whether there was not merely an absence of one phalanx. French Case (Bibl. No. 4, p. 35).

Fig. 39. *Drinkwater's Case.* In every instance of deformity, which was observed, the hands were exactly symmetrical, and the feet were probably also symmetrical. The hands and feet were affected in

all instances; all the digits were shortened. "The middle phalanx is practically or virtually absent—though not actually—from each finger and toe. The metacarpal bones are short, but the metatarsus is scarcely, if at all, affected." The middle phalanx was present as a separate bone in finger 3 of V. 27 and 44, VI. 18, 21, 26, 28, 32, 34 and 36; and in finger 4 of VI. 26, 28, 34 and 36; no trace of it was to be seen in the fingers of V. 24, 35 and 43. In other instances it was absent from certain fingers or might be recognised, welded into the base of the terminal phalanx. "The chief change in the thumb consists of a shortening of the first phalanx." With few exceptions, the abnormals were below average stature, the deficiency in growth seemed to take place after the third year. Delicate manipulation was interfered with. The children of normal parents were all normal. The married abnormals have been twice as prolific as the married normals; *moreover no living abnormal individual over 23 years of age has remained unmarried.*

The following table gives the measurements of surviving abnormals:

	Sex	No. in Fig. 39	Age	Hand	Radius	Humerus	Tibia	Femur	Height	Middle Finger	Width of Hand	Span	Reach
									ft. in.			ft. in.	ft. in.
Mrs N. .	f	V. 1	64	$4\frac{3}{4}$	9	$9\frac{3}{4}$	...	...	4 $6\frac{1}{2}$	$1\frac{1}{2}$	$3\frac{3}{4}$	...	...
Mrs M. .	f	V. 24	59	$5\frac{1}{4}$	$7\frac{3}{4}$	$9\frac{1}{2}$	13	16	4 $10\frac{1}{2}$	$1\frac{7}{8}$	$3\frac{3}{4}$	4 6	5 9
W. R. .	m	V. 27	57	$5\frac{1}{2}$	$8\frac{1}{4}$	$12\frac{1}{4}$	$13\frac{1}{4}$	$12\frac{1}{2}$	5 1	2	4	4 10	6 6
T. R. .	m	V. 35	46	$5\frac{3}{4}$	9	$10\frac{5}{8}$	$13\frac{3}{4}$	$16\frac{1}{4}$	5 $3\frac{1}{2}$	$2\frac{1}{8}$	4	5 2	6 9
Mrs H. .	f	V. 44	36	$5\frac{3}{8}$	$8\frac{1}{4}$	$10\frac{1}{4}$	$13\frac{1}{4}$	$15\frac{3}{4}$	5 1	...	...	...	...
Mrs R. .	f	VI. 18	32	$5\frac{1}{4}$	8	$10\frac{1}{2}$	$11\frac{1}{2}$	17	4 $10\frac{1}{2}$	2	$3\frac{3}{4}$	4 7	6 0
E. J. M..	f	VI. 21	22	$5\frac{1}{2}$	$8\frac{1}{2}$	$10\frac{1}{2}$	$14\frac{1}{2}$	$15\frac{3}{4}$	5 2	$2\frac{1}{8}$	$3\frac{3}{4}$	4 $10\frac{1}{2}$	5 6
M. M. .	m	VI. 25	15	$5\frac{1}{2}$	8	10	$13\frac{3}{4}$	16	5 0	$1\frac{3}{4}$	$3\frac{1}{2}$	4 $6\frac{1}{2}$	5 3
J. R. .	m	VI. 26	30	$5\frac{1}{4}$	$8\frac{5}{8}$	$10\frac{5}{8}$	13	15	4 11	...	...	...	...
T. R. .	m	VI. 28	28	5	$8\frac{1}{2}$	10	$12\frac{1}{2}$	$14\frac{1}{2}$	5 2	$1\frac{3}{4}$	$4\frac{1}{4}$	4 $11\frac{1}{2}$	6 $4\frac{1}{2}$
Mrs B. .	f	VI. 32	25	$5\frac{1}{4}$	9	10	$11\frac{1}{4}$	17	4 9	2	4	4 6	6 5
F. R. .	m	VI. 34	21	$4\frac{3}{4}$	8	$10\frac{1}{4}$	$12\frac{1}{2}$	16	5 1	$1\frac{7}{8}$	$3\frac{3}{4}$	4 $9\frac{1}{2}$	6 4
E. R. .	f	VI. 36	18	$4\frac{1}{2}$	8	$9\frac{3}{4}$	$12\frac{3}{4}$	18	5 0	$1\frac{1}{2}$	$3\frac{3}{4}$	4 $4\frac{1}{2}$	6 1
H. R. .	m	VI. 37	15	$4\frac{1}{8}$	$6\frac{3}{4}$	$8\frac{5}{8}$	$11\frac{1}{2}$	14	4 $3\frac{1}{2}$	$1\frac{5}{8}$	$3\frac{1}{2}$	4 0	5 $4\frac{1}{2}$
E. R. .	f	VI. 43	16	5	8	10	$12\frac{1}{2}$	$16\frac{1}{2}$	4 $11\frac{1}{2}$	...	...	...	...
F. R. .	f	VI. 44	6	$3\frac{1}{2}$	$5\frac{1}{4}$	$6\frac{1}{4}$	$6\frac{1}{4}$	$9\frac{1}{2}$	3 $4\frac{1}{2}$	...	...	...	...
W. H. .	m	VI. 52	13	$4\frac{1}{8}$	$7\frac{1}{4}$	9	$11\frac{1}{2}$	$13\frac{3}{4}$	4 7	$1\frac{1}{4}$	3	4 $0\frac{1}{4}$	5 $6\frac{1}{2}$
T. H. .	m	VI. 54	10	4	7	8	11	13	4 2	$1\frac{1}{16}$	$2\frac{5}{8}$	3 $9\frac{1}{2}$	5 3
H. H. .	m	VI. 59	2	...	...	...	...	...	2 $9\frac{3}{4}$	...	...	...	...
M. E. R.	f	VII. 23	5	$3\frac{1}{2}$	5	$6\frac{3}{4}$	$8\frac{1}{2}$	9	3 5	$1\frac{1}{8}$	$2\frac{3}{8}$	3 $0\frac{1}{2}$	4 $0\frac{3}{4}$
G. R. .	f	VII. 29	2	$2\frac{1}{4}$	...	...	...	...	2 $4\frac{1}{2}$	...	...	...	...
W. R. .	m	VII. 30	3	$2\frac{3}{4}$	4	$5\frac{1}{4}$	7	8	2 $10\frac{1}{2}$	$\frac{3}{4}$	2	...	...
T. R. .	m	VII. 31	$\frac{3}{4}$	...	...	...	..	...	2 $4\frac{1}{2}$	...	...	...	...
E. B. .	f	VII. 35	$4\frac{1}{2}$	3	$4\frac{1}{2}$	$4\frac{3}{4}$	$6\frac{5}{8}$	10	2 $11\frac{3}{4}$	$1\frac{1}{2}$	3	2 10	3 9
N. B. .	m	VII. 38	10 wks.	2	3	$3\frac{1}{2}$	3	5	1 $10\frac{1}{2}$	$1\frac{1}{8}$	2	1 6	...

The following measurements serve as a comparison:

Average measurements of four surviving normal *males*, over 21 years of age.

Hand	Radius	Humerus	Tibia	Femur	Height	Middle Finger	Width of hand	Span	Reach
$7\frac{1}{4}$	$10\frac{1}{2}$	$12\frac{4}{5}$	15	$18\frac{1}{4}$	5′ $9\frac{1}{2}$″	$3\frac{1}{2}$	$3\frac{3}{4}$	5′ $10\frac{1}{2}$″	7′ $4\frac{3}{4}$″

Average measurements of six surviving normal *females*, over 21 years of age.

Hand	Radius	Humerus	Tibia	Femur	Height	Middle Finger	Width of hand	Span	Reach
$6\frac{5}{8}$	$8\frac{6}{7}$	$10\frac{1}{2}$	$12\frac{1}{3}$	$16\frac{3}{4}$	5′ $0\frac{1}{2}$″	$2\frac{7}{8}$	$3\frac{1}{8}$	4′ $9\frac{3}{8}$″	6′ $3\frac{5}{8}$″

Family in Mid-England and North Wales (Bibl. No. 1, p. 35).

Fig. 40. *Hasselwander's Case.* II. 1, Josepha K., the metacarpals and metatarsals are all present, and bear first phalanges; each first phalanx articulates with a single terminal bone. III. 3 and IV. 1, Frank K. and Josepha K., similar to II. 1 in both hands and feet. The remaining deformed individuals

had, presumably, similar defects. Further description given deals with minor deficiencies and measurements. In all there was a reduction in the length of the fingers, including the thumb (from 1 to 3 cm.). The reduction was due to deficiency of the second (middle) phalanx in each. In some there was a tendency to lengthening of the metacarpals. The feet showed analogous changes. Toes shortened ·5 to 1·5 cm.; metatarsals lengthened. The middle phalanx of fingers and toes was frequently traced, welded into the base of the terminal phalanx in varying degree. German Family (Bibl. No. 3, p. 511, *corrected*).

Fig. 41. *Farabee's Case.* The family tree is given in two separate figures, from only one of which details are obtainable, and this is the tree of the offspring of abnormal parents[1]. The two trees have been so far as possible combined. The deformity affected the hands and feet, and consisted of a reduction of the number of phalanges to two in all digits with the exception of the first. The abnormality was associated with dwarfism. American Family (Bibl. No. 2, p. 70).

SECTION V α. TUBERCULOSIS.

By W. Bulloch, M.D.

Tuberculosis is an infective disease caused by the *Bacillus tuberculosis* recognised first by R. Koch in 1882. The disease occurs naturally in man and in several domestic animals, and few, if any, mammals prove to be immune when artificially infected with cultures of the living bacillus.

Even prior to Koch's discovery tuberculosis had been the subject of a vast amount of enquiry as it apparently occurs in many different forms, the relation of which was not and is not apparent. According to the overwhelming testimony of all modern investigators the only thing which is common to all forms of tuberculosis is the tubercle bacillus of Koch, which produces in the first instance the so-called miliary tubercle—a small mass of poorly vascularised granulation tissue which is prone to necrose and break-down, as a consequence of which the bacilli are able to involve fresh areas of unaffected tissues. Any part of the body may be the seat of a primary tuberculosis, but observation shows that of all tissues, the lung is the organ most frequently involved. Indeed, pulmonary tuberculosis is far more common than all the other forms of tuberculosis taken together, and in the lung the disease runs a particularly rapid, and in many cases, fatal course.

The portals by which the bacillus may enter the body are admittedly diverse, but considerable difference of opinion prevails as to which is the usual route selected. Some hold that this lies directly in the respiratory tract, giving rise to primary aerogenic tuberculosis. Another accepted route is that by which the bacilli penetrate the mucous membranes of the mouth and nasopharynx, being carried thence to the regional lymphatic glands of the neck, where they may lodge for a time. At a subsequent period they invade other lymphatic channels and become lymph- or blood-borne to the lungs.

Bacilli may be ingested with food—especially milk—and produce a primary tuberculosis in the intestine, or—and this is the more frequent—they successfully pass

[1] A protest may be well made against the method adopted by Farabee for recording a most important case; it is impossible to construct a proper pedigree from his exhibition of the data. Ed.

the intestinal barrier and become lodged in the mesenteric glands, spreading from thence to the lymph channels by which they enter the blood and are carried to the lung. Bacilli may traverse and infect any part of the skin, and lastly they may pass through the placental vessels from a tuberculous mother and give rise to congenital tuberculosis.

An immense amount has been, and is being, written on the subject of the paths of infection in tuberculosis, and the enquiry has essentially narrowed itself down to the question whether pulmonary consumption is caused by a direct aerogenic infection—a view held by Koch and Flügge—or whether the bacilli reach the lung by the more roundabout way of the lymphatics from the digestive system—a view associated especially with the names of v. Behring and Calmette. The path of infection also raises the question of the nature of the infecting material; for those who hold that pulmonary tuberculosis is due to direct inhalation of tubercle bacilli ascribe the source of these bacilli to human tuberculous sputum which is disseminated into the atmosphere, especially in the neighbourhood of uncleanly consumptives. Those, on the other hand, who hold with v. Behring that the disease reaches the lungs or other parts through the food conceive that an important *rôle* must be ascribed to bacilli of bovine origin. Indeed, Behring goes so far as to assert that the main source of consumption is the tuberculous milk consumed by the suckling, and to account for the fact that consumption is rare in young children he presupposes a very lengthy latent period, amounting to months, years, or even decades, the bacilli lying dormant, so to speak, all the while. As is well known, however, Koch at the Tuberculosis Congress in London in 1901 propounded the view that human and bovine tubercle bacilli are not identical, and that the danger of infection from bovine bacilli may, from a practical point of view, be discarded. This opinion has given rise to a vast amount of controversy, and the time is not yet ripe for the expression of definite views on the subject. It seems to the writer, however, that Koch has not been driven from his standpoint by the researches published on this point since 1901, for by far the largest number of bacillary strains isolated from human cases of tuberculosis up to the present time have proved to be of the human type, and no direct evidence has been brought forward to prove that the two types are mutually convertible. It appears impossible to doubt that the main source of infection in man is tuberculous sputum from other human beings. The chief arguments in favour of a primary aerogenic infection of the lungs are (1) the enormous dissemination of tuberculous sputum, (2) the early anatomical lesions found in the lungs of persons dead from other causes, (3) the fact that the inhalation of minimal doses induces pulmonary tuberculosis in susceptible animals. The importance of a human source of infection is also seen in countries like Greenland, Turkey, Roumania, Japan, and the Gold Coast, where tuberculosis in all its forms is very rife, and where cows' milk is drunk sparingly, or not at all.

It cannot, as we have seen, be doubted that tubercle is caused by Koch's bacillus. It is, according to the modern usage of the term, an infective disease. This doctrine is not a novel result of Koch's discovery. The medical historians of tubercle, like

Waldenburg and Predöhl, are agreed that prior to the beginning of the nineteenth century tubercle was believed to be contagious. Galen believed it; Morgagni believed it. About the beginning of the nineteenth century however, and largely through the influence of the teaching of Laennec, the contagion hypothesis began to be doubted, and in its place was set up the belief that consumption depends on some constitutional diathesis, people exhibiting this diathesis or disposition being prone to contract the disease. Laennec's influence on medicine was so great that within a quarter of a century of his death Sir Thomas Watson in his admirable *Principles and Practice of Physic* writes that the disease "cannot in my opinion be imparted even by one scrofulous individual to another. The disease I am convinced is not spread by contagion." In 1867, however, an acute and original observer, Dr William Budd of North Tawton, brought forward evidence that tuberculosis is a zymotic disease of specific nature and "the tuberculous matter is (or includes) the specific morbid matter of the disease and constitutes the material by which phthisis is propagated"; a view in complete harmony with the discoveries of Villemin and Koch. The essential factor which led Budd to this view was the geographical distribution of consumption in former as compared with present times, and especially its present great fatality in countries which when first discovered were free from the disease (e.g. South Sea Islands, North America). He also drew attention to the prevalence of consumption in convents, harems, barracks and penitentiaries in which the inmates live in relatively close contact. Koch's discovery apparently confirmed this, and in the early eighties enquiries were set afoot on a very large scale in Germany, Austria, England and America to determine from practical medical men what they had witnessed to support infection. In the main the results of these enquiries were very disappointing, for of the immense number of doctors referred to, relatively few were able to answer the question one way or another. This is perhaps not so surprising when one remembers that the date of the infection cannot be determined. It is not always possible to determine with exactitude the early stages of the disease. The endemic character of tuberculosis over the civilised globe also renders the tracing of any individual case to its source of infection almost an impossibility. Lastly, there is the question of predisposition or diathesis, for it cannot be doubted that in all infections there are two factors, the exogenic virus and the soil in which it grows. Predisposition and immunity are terms which are relative, not absolute. That a certain degree of predisposition to tuberculosis is present in almost all mankind is seen from the results of *post mortem* records published by Naegeli, Burckhardt and Schlenker. The vast majority of adult cadavera show tuberculous changes in a latent or quiescent form. From this it is justly inferred that some men are either born with or acquire a certain degree of immunity in virtue of which they are able successfully to resist the infection. Other individuals, either from an excessive dose of the virus, or an excessive degree of virulence of the virus, or from congenital or acquired disposition in unusual degree, are unable to resist, and suffer a spreading infection of active tuberculosis which carries them off. In all probability a prolonged exposure to the virus of tubercle in many cases is necessary before it makes a suc-

cessful attack. In the present state of knowledge predisposition cannot accurately be defined, although many have sought its basis in excessive vulnerability of the lymphatic system or in abnormalities, physical or otherwise, of the thorax. It must be remembered that the lung is a peculiar organ as regards its relation to the rest of the body, for all the blood of the body passes through it in the lesser circulation; still the essential cause of its predilection to tubercle remains at present unknown.

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FAMILY PHTHISIS.

By W. C. Rivers, M.R.C.S., D.P.H.

The first two (Figs. 42 and 43) of the appended pedigrees are from a German source (Klebs, *Münch. Med. Wochenschrift*, No. 4, S. 130, 1901). Any pedigrees of the tuberculous are rare; but these two have additional value because in many instances exact medical details are given, and because the record goes back for five or six

generations. It must be remembered, however, that Klebs gives them as selected instances of tuberculosis occurring in subjects whose ascendants did not suffer from the disease, or had it only in a latent form. Figs. 44—50, 52—56 give pedigrees which were recorded (at two large public sanatoria) by the present writer, and refer only to *pulmonary* tubercle. They represent, perhaps, about the maximum of information practically obtainable when a long series of cases is being gathered. Figs. 47—48 are chosen to show how family incidence of some non-tuberculous abnormal condition may seem to have a bearing on the problem of inheritance of a phthisical predisposition. Thus ichthyosis, which both these patients had—antecedent, and in addition, to their pulmonary disease—is known to be sometimes hereditary. Gassmann (*Untersuch. über Ichthy.*, 1904), from a review of many recorded cases, states that the assertion that ichthyotics come of ichthyotic stocks in 25 per cent. of the cases is a proportion probably well under the truth. Several writers (Tommasolli, Strauss, Du Mesnil) say that ichthyotics are frequently tuberculous. Pedigrees 47 and 48 show families in which in each generation consumption pitches in a remarkable way upon those affected with ichthyosis. All eight patients were "sputum-positive" cases, and care was taken to check their statements by reference to their relations. Needless to say, however, the value of such statements is only a relative and a comparative one. With a view to investigation of the recently described elder born incidence of consumption (see Pearson, *A First Study of the Statistics of Pulmonary Tuberculosis*, Dulau, 1907) it would seem advisable to put down separately the age of the parents at the patient's birth, when this information is not implicit in the record of details. Fig. 51 is taken from a series of pedigrees (F. R., No. 19) belonging to Professor Pearson, others of which will be issued later.

It is essential that the pedigrees should be read in conjunction with the descriptions before any interpretation is placed upon the hereditary factors. Pulmonary tuberculosis being a disease of young adult life, it is important to note (*a*) the ages of death of relatives not marked as phthisical, (*b*) to note that in many such cases the cause of death is unknown, and (*c*) that many individuals not marked as phthisical have not yet reached, or not yet passed through the "danger zone." For example, in Pedigree Fig. 44, in the sibship of the tuberculous II. 3, three siblings died as infants, II. 2, 4 and 5, and the remaining one, II. 1, died as a young man of "brain-fever." It is of course not reasonable to treat such a sibship as a case of one affected and four normals; the only individual who entered the danger zone died of tuberculosis at 43. Again in the same figure, III. 11 appears to have four "normal children"; IV. 11, however, died of croup at 6 months, IV. 13 of "debility" at 10 months, and IV. 10 and 12 were at date of the record only aged 15 and 12 years respectively. Such a sibship cannot be included when considering the affected offspring of tuberculous individuals. Again in the sibship II. 6—16, the four eldest were unaffected, II. 12, 14 and 15 definitely affected, but II. 13 and 16 died at 12 and in infancy of unknown causes, and certainly cannot be counted as "normals." Again in Fig. 47 nothing is known of ages at or causes of death of II. 3, 4 and 5, and we must not assume that they were free of tuberculosis, although they appear unmarked

in the pedigree. In every case the nature of contact with affected relatives as far as was ascertainable is stated.

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FAMILY PHTHISIS, CASES.

PLATE VIII. Fig. 42. I. 1, no positive trace of tuberculosis, died aet. 81. Four tuberculous (II. 2, 4, 5 and 6) and one doubtful (II. 1) in a sibship of five; marriage record probably incomplete; II. 2, a hard drinker, died aet. 64; his wife (II. 3) died aet. 35; II. 5, died aet. 33; II. 6, died aet. 36. Five tuberculous (III. 6, 8, 11, 12, 13) and three doubtful cases (III. 2, 3, 9) in a sibship of eight. III. 2, died aet. 76, had no children; III. 6, died aet. 20; III. 8, died aet. 25; III. 9, age of death given variously as 35 and 55; III. 11, died aet. 11; III. 12, died aet. 21; III. 13, died aet. 16. Three tuberculous (IV. 4, 6, 8) and two normal (IV. 1, 5) in a sibship of five; IV. 1, no children; IV. 6, sex not given; IV. 8 married and has five apparently healthy children (V. 1). Two tuberculous children in a sibship of two (IV. 9, 10), died young. Two tuberculous children in a sibship of two (IV. 11, 12), died young. Two normal children

in a sibship of two (IV. 13, 15); IV. 14, was a widow when she married and is IV. 13 in Fig. 40; she died of cancer of the breast. (Dr Klebs, Case 1.)

Fig. 43. I. 1, died aet. 83. One tuberculous (II. 2) and one normal (II. 1) in a sibship of two. II. 2, alcoholic, died aet. 51, married a normal wife (II. 3). Two tuberculous (III. 2, 4) in a sibship of two; III. 2, died aet. 67, married two normal wives (III. 1, 3); III. 4, alcoholic, died aet. 38, married tuberculous woman (III. 5), she died aet. 39; tuberculosis in her family. Six tuberculous children in sibship of six; IV. 1, 2 by first wife of III. 2; IV. 3, 6, 8, 9 by second wife of III. 2. IV. 3, married a normal wife and had three healthy children (V. 1); IV. 6 married a normal husband and had three healthy children (V. 2); IV. 8, married a healthy husband, aborted, eclampsia, one still-born child (V. 3); IV. 9, died aet. 15. Four tuberculous (IV. 11, 12, 14, 18) and one normal (IV. 16) in a sibship of five; IV. 11, died aet. 19, after birth of first child (V. 4), which she suckled; she had married a man of healthy family (IV. 10), who became tuberculous (circumscribed focus middle lobe right lung) after marriage; said to have been infected by wife; cured by staying on the Riviera. IV. 12, died aet. 21, married a normal wife and had one tuberculous child (V. 6); IV. 13 is the same woman as IV. 14 in Fig. 39; IV. 14, died aet. 17 of tuberculous peritonitis; IV. 16, alcoholic, married IV. 15, who died of apoplexy; they had no children; IV. 18, alcoholic, psychical disturbances, recovered from tuberculous peritonitis in girlhood, married a normal man (IV. 17) and had a badly developed child (V. 7), sex not stated. V. 4 treated at some time for tuberculosis, now aet. 31, and apparently healthy, married a normal woman and has one child (VI. 1). (Dr Klebs, Case 2.)

Fig. 44. I. 1, died aet. 44, brain fever; I. 2, died aet. 70, paralysis; I. 3, died aet. 77, erysipelas; I. 4, died aet. 87, senility. One tuberculous (II. 3) in a sibship of five, of whom three died in infancy (II. 2, 4, 5), cause unknown, and one (II. 1) died of brain fever as a young man. Three tuberculous (II. 12, 14, 15) in a sibship of nine. II. 6, aged 81, married II. 3; II. 7, died unmarried aet. 73, of cancer; II. 8, died aet. 77 of paralysis, she married and has a normal son (III. 13) who is married, but has no children; II. 10, aet. 76, married, and has four children (III. 15, 17, 19, 21); II. 12, died aet. 34 unmarried; II. 13, died aet. 12, cause unknown; II. 14, died aet. 28, unmarried; II. 15, died aet. about 20; II. 16, died in infancy, cause unknown. One tuberculous (III. 11) in a sibship of six; III. 1, aet. 53, married and had five children, one of whom died aet. 21 of tuberculosis; III. 3, aet. 50, married for 20 years and no children; III. 5, married for 18 years and no children; III. 7, aet. 46, married and has five children (IV. 6, 7); III. 9, married and has two children (IV. 8 and 9); III. 11, aet. 40, symptoms of tuberculosis noticed at age 37; contact with father (II. 3) until two years old, no contact with uncles (II. 12, 14, 15); he married, and has four children, the eldest is aet. 15 (IV. 10); IV. 11, died, aet. 6 months of croup; IV. 12, alive, aet. 12; IV. 13, died, aet. 10 months, of debility; III. 15, 17, 19, 20, sibship of four normals; III. 15, aet. 47, has one child aet. 15 (IV. 14); III. 17, married and has three or four children; III. 19, aet. 25—30, unmarried; III. 20, married and has three children, the eldest aet. 16. (Dr Rivers, Case 1.)

Fig. 45. I. 1, died, age unknown, tuberculous; he married a normal wife (I. 2) who died aet. 84, cause unknown; they had six children; II. 1, died, aet. 21, of tuberculosis; II. 2, died in childbed, age unknown; II. 4, over 50, married and has a normal son (III. 1), who has two normal children (IV. 1 and 2); II. 6, married only for a short time, but had no children, wife dead, age and cause unknown; II. 8, died, aet. 54, chronic bronchitis, suspected consumption; he married II. 16, aet. 59, who was one of a sibship of five, all normal, and had seven children, two died in infancy (III. 3, 4), cause unknown; III. 6, one of twins, married III. 5 who died insane, age unknown, they had two children, aet. six and four (IV. 3, 4); III. 7, died aet. 17; III. 8, aet. 28, married and has four children (IV. 5), one of them died aet. 2, cause unknown; III. 10, aet. 23; III. 11, aged 19, symptoms of tuberculosis appeared about age 18, contact with father and sister, father fell ill when he was 15, sister when he was four; II. 9, married and has two normal children, aet. 17 and 16 (III. 12, 13); II. 11 may be married; II. 12 is married and may have children; II. 14 is married and has three children, aet. 24, 20, 17 (III. 14, 15, 16). (Dr Rivers, Case 2.)

Fig. 46. I. 1, died aet. 84, gout; I. 2, died aet. 82, cause unknown; they had eight children, all normal; II. 1, aet. 69, married II. 2, aet. 63, and they have five normal children (III. 1, 3, 5, 7, 8), the eldest aet. 34 and the youngest aet. 19; II. 3, aet. 60, is married and has five normal children, aet. 38 to 25; III. 12, married, firstly a tuberculous woman (III. 11), who died aet. 35, and by her had one normal daughter aet. 16; II. 5, died aet. 59, of gout; II. 6, died aet. 54, cause unknown; II. 7, aet. 52, married II. 22, one of a sibship of eight, two of whom tuberculous; II. 7 has two children (III. 41, 42), the elder, aet. 25, is tuberculous, symptoms appeared when 24, there was no contact with the affected relatives; II. 8, aet 49, has seven children; II. 10, aet. 40; II. 11, died aet. 38, of pleurisy and heart failure. I. 3, died aet. 86, cause unknown; I. 4, died aet. 72, cause unknown; II. 12, died over 50, cause unknown; II. 13, died over 50, cause unknown, married II. 14 who died, age and cause unknown, they had three normal children, aet 38, 28, 22 (III. 25, 27, 28); II. 15, died, aet. 62, of a "broken heart" and brain disease; her

husband (II. 16), died aet. 68, alcoholism; they had one son (III. 29) who died, aet. 28, of brain disease; II. 17, dead, age and cause unknown, his wife also dead, age and cause unknown; they had two children, one, aet. 38 (III. 30), is married and has three children, the other died in childbirth, age unknown, she had three children, the eldest now aet. 18; II. 19, tuberculous, died over 50, he married and had four children; III. 34, aet. 40, has been six years married, no children; III. 36, died aet. 20, cause unknown; III. 37, aet. 30; III. 39, aet. 27, married and has three children; II. 21, died, aet. 58, of paralysis; II. 23, tuberculous, died aet. 38. (Dr Rivers, Case 3.)

Fig. 47. I. 1 and 2, both dead, age and cause unknown. It is stated that there is no consumption or rough skin in II. 1, 2, 3, 4, 5 and III. 1, 2, 3. II. 3, aet. 69, married II. 7, who had tuberculosis and ichthyosis, they had nine children; III. 7, aet. 37, married, four children, eldest 15, one (IV. 5) died, age and cause unknown, but not consumption; III. 9, aet. 35; III. 11, aet. 33, suffering from tuberculosis and ichthyosis, married three years, no children; III. 13, aet 31, suffering from tuberculosis (symptoms appeared aet. 29) and ichthyosis, no contact, married for seven years, no children; III. 15, died aet. 24, married, one child who died from an accident; III. 17, aet. 27, married, one child; III. 19, about 20; III. 20, died aet. 17, tuberculosis and ichthyosis, contact with mother; III. 21, died aet. 8 from an accident, he suffered from ichthyosis. (Dr Rivers, Case 4.)

Fig. 48. I. 2, died aet. 75, cause unknown, there is uncertainty (except in II. 3) as to the presence or absence of ichthyosis in his family; his first wife (I. 1) died when her youngest son was 14, cause unknown; they had two sons; II. 1, consumptive, dead, age unknown, married and had two sons (III. 1, 2); II. 3, died aet. 50, stroke, he married II. 4, she knows of no consumption or rough skin on her side of the family, her mother (I. 4) died, cause unknown, when II. 4 was an infant; II. 3 and 4 had seven children; III. 3, aet. 34; III. 4, aet. 32; III. 5, aet. 29, symptoms of tuberculosis appeared when 26, no contact with relatives affected; his wife (III. 6) died aet. 31, puerperal fever; their child (IV. 1) died aet. two weeks of blood poisoning; III. 7, died aet. 28, consumption did not appear till after his marriage; his wife (III. 8) died aet. 25 of consumption, they were married 18 months, there were no children; III. 9, died in infancy of teething; III. 10, died in infancy of inflammation of brain; III. 11, aet. 20. (Dr Rivers, Case 5.)

PLATE IX. Fig. 49. I. 1, died aet. 77, dropsy, his wife (I. 2) died aet. 76, stroke; they had nine normal children; II. 1, died aet. 87, cause unknown; his wife died aet. 50—60 from cancer; II. 3, aet. 87, married II. 29 who died, aet. 57, of apoplexy; they had 10 children, III. 39 aet. 55, III. 42 aet. 48, III. 47 died aet. 8 of diphtheria, III. 51, aet. 35; III. 52, aet. 34, symptoms of tuberculosis appeared when aet. 33, no contact with affected relatives; II. 4, died aet. 74, cause unknown, his wife may be dead; II. 7, died aet. 71, cause unknown, his wife died aet. 60, cause unknown; II. 9, died aet. 77, cause unknown, his wife dead, cause and age unknown; they had 10 children, of whom seven are dead and none married; II. 11, died aet. 60, dropsy, her husband dead, age and cause unknown; they had seven children; III. 3, aet. 70, his wife, who was 50 at marriage now 75, they have no children; III. 7, aet. 60, was 50 at marriage, no children; III. 9, has eight children, two of whom are married; II. 13, died, aet. 40, from an accident; II. 15 died aet. 50, stroke, his wife dead, age and cause unknown; they had four children, III. 20, died, aet. 30, of pneumonia; III. 21, died suddenly, age and cause unknown; III. 22 and 23, died in infancy; II. 17, died over 80, cause unknown, her husband (II. 18) dead, cause unknown; they had more than seven children, of whom at least three married and had children; II. 19, died, aet. 70, stroke; II. 20, died 50—60, tuberculous, his wife (II. 21) died aet. 76, cause unknown; they had seven children, of whom three are dead, one of pneumonia, aet. 25, the other two died aet. 27 and aet. 45 of unknown causes; II. 22, died aet. 77, heart disease, her husband died over 80, cause unknown; they had eight children; III. 26, over 60; III. 28 is 40; III. 29 is over 50; III. 30 has four daughters and two sons, one daughter dead, age and cause unknown; III. 32, died of liver disease, age unknown; III. 33, died of cancer of womb, age unknown; II. 24, aet. 82; II. 25, died, aet. 56, of diabetes, of her five children (III. 37), two died in infancy, cause unknown; II. 27, died aet. 35, tuberculous; II. 30, died aet. 50—60, cause unknown. (Dr Rivers, Case 6.)

Fig. 50. I. 1, died aet. 42, accident, his wife died aet. 77, senility; they had nine children; II. 1, aet. 64, his wife (II. 2) dead, age and cause unknown; their elder daughter (III. 1) died aet. 20, cause unknown; II. 3, died, aet. 27, of tuberculosis; II. 4, aet. 61, married II. 24 aet. 64, her father (I. 3) died aet. 63 of congestion of the lungs, and her mother (I. 4) died aet. 76, senility; II. 4 and 24 had four children, III. 4 died aet. 3 of scarlatina, III. 5 died aet. 4 of croup, III. 6 aet. 28, III. 8 aet. 20, tuberculosis developed when 19, no contact with affected relatives; II. 6, aet. 57, his wife (II. 7) died of Bright's disease, age unknown, they had seven children (III. 9, 11 and 3 to 17); II. 8, aet. 55, has two sons, the elder aet. 21; II. 10, died, aet. 53, of Bright's disease and diabetes; II. 12, died, aet. 18, of tuberculosis; II. 14 died in infancy, cause unknown; II. 15, died, aet. 60, of heart disease; his wife (II. 16) dead, age and cause unknown, they had two children, III. 24, aet. 40—50, whose eldest son (IV. 1) died suddenly of inflammation of the bowels, age unknown, the younger son (IV. 2) is aet. 21; III. 26, aet. 35, has two children under 10 years; II. 17, died aet. 18, cause unknown; II. 18, died, aet. 32, cause unknown;

II. 20, died aet. 54, cancer; her husband dead, age and cause unknown; they had three children; III. 28, aet. 44, her husband (III. 29) dead, age and cause unknown; III. 30, aet. 34; III. 32, aet. 29; II. 23, died aet. 22, accident; II. 25, aet. 62, has seven children, the eldest (III. 34) aet. 33; II. 27, aet. 60, has been married 13 years and has no children. (Dr Rivers, Case 7.)

Fig. 51. I. 1, died aet. 78, senility, suffered during life from nasal polypus and hypochondria; his wife (I. 2) died aet. 83, senility; II. 2, died, aet. 51, of asthma and bronchitis, he was one of a sibship of nine, of whom one was insane, one an epileptic, one eccentric, one suffered from hysteria and one from chorea; II. 2 married II. 3 who died aet. 98 of senility, her father (I. 3) died when elderly, cause unknown, her mother (I. 4) died in childbirth, age unknown; II. 2 and 3 had six children, III. 1 died, aet. $\frac{5}{12}$, probably hydrocephalus; III. 2, aet. 67; III. 3, aet. 66, suffers from gout and hysteria; III. 4, aet. 60, scrofulous; III. 5, died, aet. 53, of chronic bronchitis, had scrofula; III. 6, died, aet. $\frac{11}{12}$, of scrofula; II. 4, died in middle age of tuberculosis; II. 5, died, aet. 60, of tuberculosis; II. 6, died in middle life of tuberculosis; II. 7, died in middle life probably of dropsy; II. 8, died aet. 75, cause unknown. (Pearson's Family Records, Schedule 19.)

Fig. 52. I. 1, died over 80, cause unknown; his wife (I. 2) died over 90, cause unknown; they had six children; II. 1, married II. 10; her father (I. 3) died, aet. 70, of cancer, and her mother (I. 4), aet. 40, of heart disease; II. 1 and 10, had nine children; III. 5, died aet. 29, tuberculosis; his wife died, aet. 25, of tuberculosis; they had three children, the youngest (IV. 3) died, aet. seven months, of tuberculosis; III. 7, aet. 32; III. 9, died, aet. 24, of tuberculosis, had one child who died, aet. two months, of convulsions; III. 11, died, aet. 21, of tuberculosis, contact with elder brothers; III. 12, aet. 21, symptoms of tuberculosis showed when 20, contact with elder brothers; III. 13, aet. 19; III. 14, aet. 17; III. 15, aet. 13; III. 16, aet. 9; II. 2, died aet. 30 of fever; II. 3, aet. 46, married five years, no children; III. 2, died aet. 11, of influenza; II. 9, died, aet. 57, cause unknown; II. 11, aet. 51, has four children, the eldest (III. 17) died, aet. 10, of disease of the spine; III. 18, aet. 19; II. 13, died, aet. 36, of dropsy; II. 15, died, aet. 18, of small-pox; II. 16, died, aet. 16, of small-pox. (Dr Rivers, Case 8.)

Fig. 53. I. 1, died, age not given, tuberculosis; he had six children; II. 1, died, aet. 27, tuberculosis; II. 4, died, aet. 45, tuberculosis, he had one daughter (III. 1) who has been married for two years to a tuberculous man, they have no children; II. 6, aet. 59, has three children; III. 3, aet. 23, married and has one child (IV. 1); III. 5, died when teething; III. 6, aet. 16; II. 8, aet. 40, tuberculous; II. 10, aet. 49; he married II. 14, aet. over 50, whose father (I. 3) died, aet. 79, of old age, and mother (I. 4), aet. 82, of a stroke; II. 10 and 14, had 10 children; III. 11, died, aet. 4, of enteric; III. 12, aet. 25; phthisis developed aet. 24, no contact with affected relatives, had atrophic rhinitis; III. 13, aet. 23; III. 14, died, aet. 5, from an accident; III. 15, aet. 21; III. 17, aet. 18; III. 19, aet. 13; III. 26, a sibship of six, aet. 25 to 2, one girl dead, aet. 21, married, left one child. (Dr Rivers, Case 9.)

Fig. 54. I. 2, died of heart disease, aet. 56; she had 10 children, no phthisis in the family; II. 1, about the fifth child, aet. 39, married II. 5, aet. 40, daughter of I. 3 and 4; I. 4, died, aet. 29, of tuberculosis; I. 3, died seven years after his wife; II. 1 and 5, had five children; III. 1, aet. 21; III. 2, aet 19, symptoms of tuberculosis at 17, no contact with affected relatives; III. 3, aet. 18; III. 4, aet. 13; III. 5, aet. 5; II. 5 aet. 40; II. 6, has 12 children, aet. 23—$\frac{3}{12}$, the eldest is married, has no children; II. 8, aet. about 30; II. 10, died, aet. 5, of tuberculosis. (Dr Rivers, Case 10.)

Fig. 55. I. 1, died, aet. over 70, stroke, his wife (I. 2) died, aet. over 60; they had six children; II. 1, died from an accident; II. 3, died of heart failure; II. 7, aet. 45, married II. 11, aet. 47, who is the son of I. 3 who died, aet. 65, stroke, and I. 4 who died, aet. 56, of phthisis; one of the sisters of I. 4 also died of phthisis; III. 4 is a daughter of II. 7 and 11, aet. 24, symptoms of phthisis when she was 20, she, aet. 13, nursed her grandmother (I. 4); III. 5, aet. 21; III. 6, aet. 20; II. 12, aet. 46; her husband (II. 13), aet. 45, two of their six children died in infancy; III. 9, died of convulsions; III. 10, dead, cause not given; II. 14, aet. 44, and II. 15, aet. 46, had four children. (Dr Rivers, Case 11.)

Fig. 56. I. 1, died, aet. 45, of heart disease; I. 2, died, aet. 67, of rupture; II. 1, aet. 52, married II. 3, aet. 48, whose father, I. 3, died aet. 50, "natural death," and mother, I. 4, aet. 70—80, died of dropsy; II. 5, the brother of II. 3, died aet. 44, of phthisis; two of the sibship (III. 1 to 6) died in infancy (III. 1, 3), cause not given; III. 2, died, aet. 24, of phthisis, no contact with uncle (II. 5); III. 4, aet. 22; III. 5, aet. 18, symptoms of phthisis appeared when 17, contact with brother (III. 2). (Dr Rivers, Case 12.)

SECTION VI α. DEAF-MUTISM.

INTRODUCTORY. By Jobson Horne, M.A., M.D., B.C. Cantab.

The acquisition of speech is dependent upon the possession of the sense of hearing. Dumbness resulting from impairment of hearing is termed *deaf-mutism*.

The number of deaf-mutes in Europe has been estimated at about 1 in 1350 of the population. In England the number is 1 in 2043, in Scotland 1 in 1860, in Ireland 1 in 1398 of the population[1].

Deaf-mutism is either *congenital* or *acquired*, that is to say the defects of the organ of hearing are either developed before birth and in the embryonic stage, or result from disease, general or local, acquired during the first few years of life. Pedigrees collected by the Eugenics Laboratory show that direct heredity from the parents and the grandparents is not so very rare as some authorities[3] have stated. Deaf-mutism among sisters and brothers is of frequent occurrence. Consanguineous marriages and the intermarrying of the congenitally deaf are largely responsible for the congenital cases, while the infectious diseases of children account for the acquired cases of deaf-mutism. Meningitis (especially epidemic cerebro-spinal meningitis) and scarlet fever, are more frequently the cause than all other infectious diseases of childhood put together. Hitherto statistics do not seem to have taken into account sufficiently mumps and hereditary syphilis as causative factors. Although within recent years a considerable amount of valuable material has been collected for statistical purposes, it is difficult to deduce from this the true ratio of the congenital to the acquired cases of deaf-mutism. The figures given vary between 33 and 174 cases of acquired deaf-mutism to every 100 congenitally deaf[2]. Many of the congenital cases do not come under observation during the first two or three years of life. A child need not be born stone-deaf in order to develop into a deaf-mute. It may be that by the age of three or four years the child has acquired by means of lip reading a few sounds such as Pa-pa, Ma-ma, and makes no further progress. The mother does not know the child is dumb and regards the speech only as backward. The age of school-life is next reached and the difficulty has to be faced. The parents are naturally reluctant to disclose a family defect and the tendency is then to attribute to some illness or accident in the first years of life what is really an inheritance. Furthermore it would seem that a weakness or tendency to deafness is at times hereditary, and may become patent with disease; hence the record of cases in which deaf-mutism has been discovered in ancestry and collateral lines, but is attributed in other members of the family, and probably in some cases correctly, to non-congenital sources. These are some of the difficulties to be contended with in arriving at a true estimate of the ratio of the acquired to the congenital cases, with the result that there is a liability for the latter to be understated.

A large number of deaf-mutes have remnants of hearing upon which it is possible

to base a method of education enabling them to use their ears in conjunction with their eyes[4]. A congenitally deaf child not uncommonly has more hearing than one who has lost the hearing since birth, but the former has more difficulty in learning how to speak than the child who acquired deaf-mutism at an age after some speech had been developed. The method of education has to be varied accordingly. It is the work of the otologist to investigate carefully the hearing of each pupil and to decide to what extent the child can be relieved of the ills of its inheritance.

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Cases of Hereditary Deaf-Mutism.

PLATE X. Fig. 57. *Stephenson's Case.* I. 1, an uneducated deaf-mute woman seduced by a normal man, I. 2, had a deaf-mute daughter, II. 1, who married a normal man and had four children, one normal and three deaf-mutes. The normal child, III. 3, married a normal husband, III. 2, and had four normal children, IV. 1; the first deaf-mute child, III. 3, married a deaf-mute wife, III. 4, and had nine children, of whom one only, IV. 4, was a deaf-mute, five of the normal children, IV. 2, died in infancy; the second deaf-mute, III. 5, married a deaf-mute husband, III. 6, and had four children, IV. 5 a deaf-mute, IV. 8 an idiot and two normal children, IV. 6 and 7; the third deaf-mute, III. 7, also married a deaf-mute husband and had three children, two normal, IV. 9 and 10, and one deaf-mute, IV. 11. (Communicated by Mr Stephenson to Dr Kerr Love and published by him in his book, Bibl. No. 14, p. 81.)

Fig. 58. *Moos' Case.* I. 1, a deaf-mute, I. 2, normal; they had two children, II. 1, a deaf-mute and, II. 2, normal who married a normal man, II. 3; both II. 2 and 3 are said to have been healthy and their senses perfect. They had three children, III. 2 and 3, deaf-mutes, and III. 4, normal. III. 2, married a deaf-mute and had a deaf-mute son, IV. 1. III. 4, married a woman, III. 5, like himself healthy and with perfect senses, and had a deaf-mute son. (Cited from the *Wiener med. Wochenschrift* by Dr A. Hartmann, Bibl. No. 7, p. 55.)

Fig. 59. *Dahl's Case.* Not much more data are given than are indicated in figure. Idiocy and insanity seem to have appeared in the stock in the third recorded generation; III. 1, being an idiot and III. 14 insane. The brother, III. 2, of III. 1 had two grandchildren only, one, V. 1, epileptic and V. 2, his cousin, an idiot; the brother, III. 4, had one grandchild only, V. 3, insane, and this insane grandson, marrying his second cousin once removed, IV. 15, had an idiot daughter, III. 1. Among first cousins once removed of III. 1 and 14, are: IV. 7 and 8 insane, IV. 10 an idiot, and IV. 11 and 14 deaf-mutes, the brother, IV. 13, of one of these deaf-mutes has two deaf-mute sons, V. 5 and 6. The stock is a marked illustration of general degeneracy. (See Bibl. No. 4, Pedigree III., p. 80.)

Fig. 60. *Townsend's Case.* Nothing known of I. 1 and 2, except that they had three deaf-mute offspring, II. 2, 3 and 5, and probably other offspring. II. 2 married a normal wife, II. 1, and had an only daughter, III. 1, deaf-mute; she married a normal man, III. 2, and had three normals (IV. 1—3) and two deaf-mute (IV. 4—5) children. The second deaf-mute member, II. 3, of the original sibship cohabited with a deaf-mute man, II. 4, and had seven illegitimate children by him, of whom three, III. 4—6, were deaf-mutes, the third original deaf-mute, II. 5, married a normal husband, II. 6, and a single daughter, deaf-mute, III. 8, who in her turn married a normal husband, III. 8, the result being a deaf-mute son IV. 6. (Townsend's Case, communicated to and published by Dr Kerr Love, Bibl., No. 14, p. 80.)

Fig. 61. *Hartmann's Case.* Nothing reported of I. 1, 2 and 3, but I. 4 was a deaf-mute. II. 2, a deaf-mute the son of I. 1 and 2 married the deaf-mute daughter, II. 3, of I. 3 and the deaf-mute, I. 4; the result of this marriage was four deaf-mute daughters, III. 1—4 and a son, III. 5, with perfect senses. There may have been other offspring of I. 1—4, and the birth order of III. 1—5 is not recorded. (See Bibl. No. 7, p. 54.)

Fig. 62. *Howard's Case.* I. 3 and 4, two deaf-mutes on marriage had four deaf-mute children, of whom II. 3—5 left no descendants, II. 2, however, married the deaf-mute son, II. 1, of two normal parents, and gave birth to five deaf-mute children, III. 1—5. (Published by Dr Kerr Love, Bibl. No. 14, p. 81.)

Fig. 63. *Dahl's Case.* An extensive pedigree showing general degeneracy in the later descendants of a single pair, I. 1 and 2. One of their daughters, II. 2, leads to a stock in which idiocy and insanity are rife, and a second to a stock in which deaf-mutism, idiocy, blindness and leprosy appear. Of the pair, II. 1 and 2, one son, III. 9, no grandchildren, five great grandchildren, V. 3, 12, 15, 29, 33, eight great great grandchildren VI. 5, 9, 15, 16, 23, 24, 25, 26 and four great great great grandchildren, VII. 1, 2, 3, 7, were insane. Besides this two great great grandchildren, VI. 18 and 22, and four great great great grandchildren were idiots; three great great great grandchildren, VII. 5, 6 and 9 were feeble-minded, the latter being also epileptic. In the other branch of the family from II. 4 and 5, the grandchildren were all normal, two of the great grandchildren, V. 54 and 55 were deaf-mutes, the first being also an idiot and the second blind; of the great great grandchildren, VI. 43, 44, 49, 50, 52, were deaf-mute, VI. 50, being also blind, VI. 38 was insane, VI. 32 probably, and VI. 51, certainly lepers. Of the great great great grandchildren, VII. 13, 18 and 19 were deaf-mutes, VII. 23 an idiot, and VII. 14, 15, and 21 lepers. In the eighth generation there are only five great great great great grandchildren and all of them are said to be lepers. Thus in the seventh generation of one branch all the offspring are mentally abnormal and in the eighth generation of the other branch all are leprous. (See Bibl. No. 4, Pedigrees IV. and V., p. 82.)

SECTION VII α. INHERITANCE OF ABILITY.

(From the Galton Eugenics Laboratory.)

When we pass from the subject of human deformity, or of human disease capable of definite diagnosis to the question of the inheritance of ability in man, it will at once be admitted that the transition from the physical to the psychical has introduced new difficulties of classification and interpretation. The problem, however, of classifying ability and of determining whether it does or does not exist is really not so widely divergent from that which faces the inquirer into the medical aspect of physical inheritance. With the latter the fundamental idea is the inheritance of the diathesis or constitution which, given the suitable environment, leads to the development of disease. With the former the investigation turns on the presence of a certain mental constitution, a psychical aptitude—not wholly severed however from physique—which, given the suitable environment, training or opportunity, leads to achievement. The use of the term "psychical diathesis" might be justified as a legitimate extension of a familiar idea. Thus in both cases we should be concerned with the inheritance of the diathesis, but alike in both cases we can only infer it from its product—the appearance of disease or the record of achievement. In both cases we must accompany our investigation by recording, if possible, whether the conditions for the appearance of the disease, or for achievement have been present. For this reason the inheritance of ability is easier to follow in the middle or upper social strata, than in the lower ones; for in the former the physical nurture and the mental training which provide the primary conditions of achievement are present as a matter of course. The school, the university, the professions, political, literary, mercantile, scientific or administrative life, are more or less open to the individual and ability, if present, has power to show itself in achievement. *Marked* ability from the lower ranks can fight its way to achievement to-day, but it is not obvious that it was as well able to do so in the past, and accordingly the record of achievement is likely to be at fault when we approach the ancestry of the able but so-called "self-made" man. Thus our first pedigrees are drawn largely from families whose histories are more or less public property. In the two cases given in the first instalment of this section, we have to thank distinguished members of the stock for much aid. The pedigrees were drafted in the Galton Eugenics Laboratory and then presented to them with a request for revision, which was most courteously granted. The responsibility for determining the grade of achievement which justified the use of the symbols ● or ◒ lies with the Laboratory. In general the standard taken was that appearance in the *Dictionary of National Biography* (D. N. B.), or an equivalent public estimate justified the use of the symbol ●; cases of less distinction or of great promise of future achievement were marked by ◒. In the families selected an attempt was made to illustrate ability of a special kind. Thus in Fig. 64 the chief characteristic kept in view was legal ability turning in the administrative direction.

In Fig. 65 we have again legal ability but accompained also by literary and executive capacity. It is more than possible that we have passed over cases in which ability has led to achievement unknown to us. Further, in the youngest generations attention must be paid to the age, which in many instances we have recorded; it will be seen at once that the individual has frequently not reached, only just reached or is still within the period of possible achievement. Whatever be the defects of such omissions or incompletenesses in the pedigrees of human ability appearing in this *Treasury*, these pedigrees will, we believe, suffice to show that the power of achieving distinction in special directions undoubtedly runs in certain stocks. The curious—but not necessarily scientific—reader of these pages may succeed, without much trouble possibly, in identifying the stocks whose pedigrees are given. As the only information provided tends in these cases to the honour of the family, there is no reason for the suppression of the names, beyond the rule of this publication, that, as it is intended for purely scientific purposes, no names will be given throughout. Each individual sinks into a classified unit, and appears only as a counter in the final statistical discussion.

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PLATES XI. and XII. Fig. 64. II. 6, died aet. 90. II. 8, died aet. 33, distinguished himself as Captain in the R. Navy. III. 9, died aet. 26, Secretary, Interpreter and Commissary to Lord Clive. III. 15, died aet. 63, Director East India Company, Chairman, created Bt. IV. 3, died aet. 73, Bencher, successively Reader and Treasurer of the Inner Temple, Judge in Ceylon, Chief Commissioner of Colonial Board of Audit, Master of the Crown Office. IV. 10, died aet. 92, P.C., M.P., D.C.L., Joint Secretary to the Treasury, Chairman of Committees, House of Commons, Governor of Madras (D. N. B.). IV. 12, died aet. 80, G.C.B., M.P., General, Chairman East India Co. (D. N. B.). IV. 23, died aet. 91, P.C., D.C.L., M.P., Fellow of All Souls Coll., Oxford, Judge of High Court of Admiralty, Dean of Arches (D. N. B.). IV. 26, died aet. 71, M.P., Chief Secretary to the Govt. of India (published two works of importance). V. 3, died aet. 32, D.C.L. Oxon., Prof. of Greek at Glasgow Univ., Lord Rector of Glasgow Univ. (D. N. B.). V. 4, died aet. 43, Fellow of Trin. Coll. Camb., Secretary of State at Malta, brought forward new Maltese Code (D. N. B.). V. 12, died aet. 78, Barrister Inner Temple, Member of Supreme Council of Ionian Islands, Metropolitan Police Magistrate. V. 17, died aet. 28, considered a man of considerable ability, Private Secretary to Governor of Madras and Govt.-Agent to the Durbar of H.H. the Nabob of the Carnatic. V. 25, died aet. 62, Financial Sec. to the Govt. of India. V. 31, died aet. c. 40, considered an able man, First Assistant to Chief Commissioner at Delhi, Officiating Magistrate and Collector at Bareilly, Commissioner at Kumaon. V. 35, died aet. 45, Fellow of All Souls, Oxford, M.P. for Canterbury for three years. V. 52, died aet. 95, a man of considerable

ability in Bengal Civil Service, Magistrate and Collector of Northern Division of Moorshedabad, etc. V. 54, died aet. 74, G.C.B., Admiral Superintendent of Indian Navy, commanded South American Station, commanded Naval Brigade at Sebastopol (D. N. B.). V. 68, died aet. 82, Financial Sec. to Govt. of India for six years, Treasurer of Guy's Hospital for 20 years (D. I. B.[1]). V. 74, died aet. 49, Lady Principal of Women's College. V. 75, died aet. 30, distinguished school career, in the Bengal Civil Service, Joint Magistrate and Deputy Collector at Dacca. V. 77, living, aet. 76, K.C., distinguished career. V. 78, died aet. 75, G.C.M.G., Barrister Inner Temple, Counsel to the Home Office, Permanent Under-Sec. Home Office. V. 82, died aet. 68, D.D., Fellow of Trin. Coll. Camb., Canon of Bristol, writer (D. N. B.)[2].

Fig. 65. II. 2, died aet. 67, Chief Justice of the Supreme Court, Bombay (D. N. B.). II. 6, died aet. 87, Chief Baron of the Exchequer, Attorney General (D. N. B.). II. 10, died aet. 86, Field Marshal, Govt. Director of the East India Co. (D. N. B.). III. 2, an able woman, author of literary reviews and one published memoir. III. 3, died aet. 73, Queen's Remembrancer, author (D. N. B.). III. 7, living, aet. 87, very distinguished legal career. III. 10, died aet. 74, Baron of the Exchequer, received status of Justice of the High Court, author of legal works (D. N. B.). III. 15, died aet. 72, K.C.S.I., Commisioner of Peshawar, Seistan Boundary Commissioner (D. I. B.), III. 18, died aet. 82, Hon. LL.D., Q.C., M.P., Baron of the Exchequer (D. N. B.). III. 33, died aet. 71, Q.C., M.P., Lord Justice of Appeal (D. N. B.). IV. 2, living, aet. 63, LL.D., D.C.L., Fellow of his College, University Professor. IV. 3, living, aet. 58, editor of a weekly review for eleven years, author. IV. 10, living, aet. 59, K.C., Bencher of his Inn, Deputy-Chairman of Quarter Sessions. IV. 21, living, aet. 49, F.R.C.P., well-known obstetric physician. IV. 23, living, aet. 47, K.C. IV. 25, living, aet. 45, D.D., M.V.O., headmaster of a well-known public school. IV. 28, living, aet. 56, Select Preacher, Cambridge. IV. 46, living, F.R.S. IV. 57, living, aet. 78, K.C., P.C., G.C.M.G., Bencher, Hon. Fellow of his College, Lord of Appeal in Ordinary. IV. 59, died young, distinguished officer. V. I, living, aet. 30, Fellow of his College. V. 17, living, aet. 49, K.C. V. 20, living, aet. 39, rising Junior at the Common Law Bar, editor of several law books[2].

SECTION VIII *a*. CHRONIC HEREDITARY TROPHOEDEMA.

(Milroy's Disease, Meige's Disease, Congenital hereditary Elephantiasis.)

By W. Bulloch, M.D.

In 1891 Desnos[(1)], in a communication to the Société médicale des hôpitaux Paris, drew attention to the existence of a peculiar, hard, painless oedema of the skin, of chronic character, occasionally hereditary, and more frequent in females than males. He proposed to call it "oedème rhumatismal chronique pseudo-elephantiasique." In the same year under the name of congenital hereditary elephantiasis, Nonne[(17)] of Hamburg described a similar condition. In the following year Milroy[(11)] a physician of Omaha published an account of a remarkable family in which twenty-two cases of chronic oedema had occurred. Seven years later Henri Meige[(10)] in publishing the history of another family with eight affected members gave an excellent account of the disease which has been recognised by a large number of subsequent observers in different parts of the world.

The disease is characterised by the presence of persistent hard painless swelling of one or more segments of the limbs. In the great majority of cases, after it has once developed, the disease is progressive and persists throughout life. In a number of cases it has appeared at birth (Milroy[(11)], Tobiesen[(15)], Nonne[(17)], Lortat-Jacob[(18)], Jopson[(13)], Sutherland[(20)]), whereas in others (Meige[(10)], Lannois[(12)], Rolleston[(16)], Hope

[1] *Dictionary of Indian Biography.*
[2] In the last generation the ages when known are given on the pedigrees.

Fig. 1. Son.

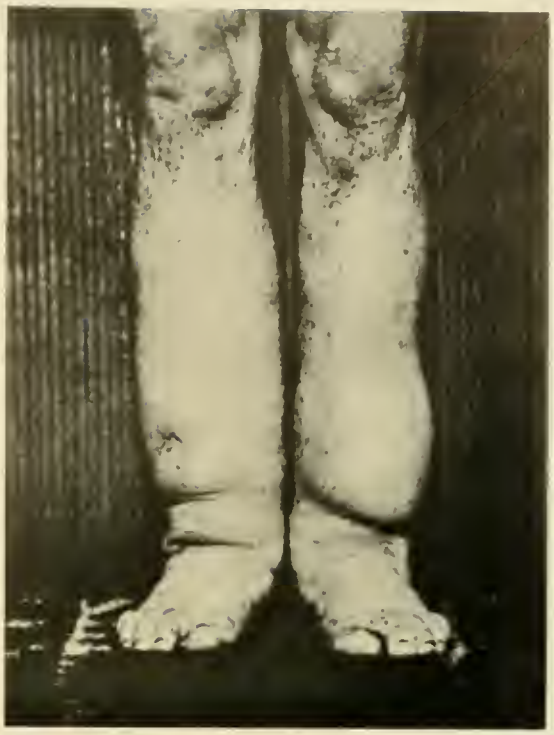

Fig. 2. Father.

Chronic Trophoedema. Nonne's Cases. Reproduced, by kind permission of the Publishers, from *Virchow's Archiv*, Bd. 125.

Fig. 3. Daughter.

Fig. 4. Father.

Chronic Trophoedema. Hope and French's Cases. Reproduced, by kind permission of the Publishers, from the *Quarterly Journal of Medicine*, Vol. I.

and French[14]), its onset has been delayed till about puberty or in some instances later. Occasionally sporadic as in the cases reported by Mabille[4], Sicard and Laignel-Lavastine[5], Parhon and Florian[6], Hertoghe[7], Rapin[8], Sainton and Voisin[9] and others, a number of cases have been published in which heredity is an important factor. Such cases have been reported by Milroy[11], Nonne[17], Meige[10], Tobiesen[15], Rolleston[16], Lortat-Jacob[18], Lannois[12], Jopson[13], Hope and French[14], Moyer[19] and Sutherland[20]. In the majority of instances the heredity is direct but instances in which a normal individual has transmitted the disease are seen in the pedigrees published by Milroy (III. 7, III. 4, IV. 11), Hope and French (III. 14), Tobiesen (III. 2), Lortat-Jacob (III. 2) and Lannois (II. 4).

In Hope and French's case and in Meige's case (IV. 1) in addition to the chronic persistent oedema several of the affected individuals suffered from "acute attacks" ushered in with violent pain, shivering, rise of temperature, and redness and swelling of the oedematous parts. The table below shows the sex incidence.

Observer	Male	Female	Sex not stated
Hope and French	5	8	
Tobiesen	3	1	
Rolleston	1	2	
Nonne	3	5	
Lortat-Jacob	1	5	
Meige	4	4	
Milroy	12	8	2
Lannois	—	4	
Jopson	3	—	
Moyer	—	2	
Sutherland	—	2	
Totals: ...	32	41	2

With reference to the cause of Trophoedema nothing is known with certainty. Meige considered that the evidence was in favour of some congenital or acquired defect of the trophic centres in the spinal cord for the cellular connective tissue—hence his name Trophoedema. The segmental distribution of the disease is held to support this theory. Plate E illustrates the appearance of the disease.

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PLATE XIII. Fig. 66. *Hope and French's Case.* Hope and French (1908) have published the history of a family (T—— W——), some of the members of which reside at Caversham near Reading. In five generations 13 members were affected with persistent oedema of the legs. Five were ♂ and eight ♀. In addition to the chronic trophoedema acute "attacks" in the affected parts were frequent. A number of those afflicted were also subject to epilepsy and other nervous disorders. It will be noted that our pedigree differs in several respects from that given in Hope and French's paper. This is to be explained by several gaps being filled up or corrections made, as the result of fresh information which has been sent us by Dr Hope (of Caversham) to whom we are greatly indebted. I. 1, William T——, ♂, "had very swollen legs all his life," no other data available. He died in 1854 at a very advanced age. I. 2, his wife, no particulars. II. 1, William T——, Jun., had "swollen legs" and "attacks," died in 1870. II. 3, sister of II. 1, also affected, died in 1864 at the age of 70. Married II. 4 (W——). III. 1, had "swollen legs" and "attacks," died in 1860 of scarlet fever. It is stated in Hope and French's paper that she married but had no children. Dr Hope's recent inquiries however show that she had two boys and a girl all normal, likewise four grandchildren normal. III. 3 and III. 5, not mentioned in Hope and French's paper, normal. III. 7, alive, normal. III. 9, ♀, "had swollen legs," went to America and is thought to have been drowned. III. 10, ♂, alive aged 75, first troubled with swollen legs at the age of nine. At 20 years of age had his first acute attack and during the next 30 years had frequent seizures. Is able to keep down the swelling of his legs by bandages. III. 12, III. 14 and III. 19, normal females. III. 14 is the only instance of transmission through an individual not affected. III. 16, no oedema, died of phthisis. III. 17, suffered from girlhood with "swollen legs" and "acute attacks," died in her first confinement. IV. 1—23, T—— descendants in fourth generation all free from oedema. Living in Wiltshire. IV. 20—21 and IV. 22—23, twins. IV. 24, ♂, alive and in good bodily health, no oedema. Dull of intellect and barely able to earn his living. IV. 26, ♂, aged 48, has "swollen legs" to a moderate extent and has had "acute attacks."

Both legs affected up to the knee but more so on right than on left side. Intellect fairly good but he lacks enterprise and energy. In the last two years has had three fits, in one of which he fell down and injured himself, probably epileptic. IV. 30, no oedema, had convulsions in infancy, slow in speech and movement. IV. 34, ♀, began to have swollen legs when nine years old, the swelling gradually increasing till it reached the hips; also suffered from acute attacks. She married and took to drink; died in 1901. IV. 43, ♀, aged 39, was normal up to age of 21 when her legs began to swell first at the knees and then spread down to feet and toes. Had her first "acute attack" at the age of 36, since then they occur frequently either just before or just after the monthly period. With one exception the acute "attacks" have been unilateral. At the present time both legs are greatly enlarged up to the groins but not involving the vulva. IV. 46, child still-born, sex not stated. IV. 47—50, four children of III. 19. In Hope and French's paper it is said there is only one child. V. 7, ♀, aged 20, was normal at birth but at the end of three months had swollen feet without apparent cause. By the age of 11 oedema was very pronounced extending up to the knees. In 1904 when 16 years old she had her first "acute attack" and in the course of the next two years had 10 acute seizures, mostly ushered in with violent pain, shivering, rise of temperature, redness and swelling of the affected parts. The "attack" was usually more marked on one side than on the other. In 1906 had an attack of mental derangement (mania) lasting about four months. V. 10, ♀, 12 years old, has begun to develope swollen legs. Like her father and sister she has also had acute attacks. V. 11, no oedema so far, but is epileptic. V. 14 and 15 not mentioned in Hope and French's paper. V. 16—21, living in Egypt, four reported to be normal, the fifth, a boy (V. 16), has already swollen legs, which began without apparent cause. It is since reported that the swelling has disappeared (Dr Hope). V. 22, in Hope and French's paper said to be a ♂. Names in original. (See Bibl. No. 14.)

Fig. 67. *Tobiesen's Case.* Four cases of trophoedema in three generations and occurring congenitally. I. 1, I. 2 and II. 1, all stated to be normal. II. 2, woman, aged 49. Affected with oedema of legs at time of birth. As she grew older the oedema diminished and by the time she had her first child at the age of 21 it had almost entirely disappeared. When seen by Tobiesen the oedema was very slight. II. 3 her husband, healthy. III. 1, aged 28, affected from birth. At first both legs were affected, especially the left which continued large. The right leg at the time the patient was seen by Tobiesen was normal. III. 2, 3, 4, 5, normal. IV. 1, ♂, brought for examination when 14 days old. Oedema of legs had existed from time of birth and was independent of any vascular or osseous change (X-ray test). IV. 2, brought for examination two years later, affected in like manner but not so severely. (See Bibl. No. 15.)

Fig. 68. *Rolleston's Case.* Three cases of hereditary trophoedema in a family; names in original. They were patients of Dr Hollis of Wellingborough who has personally supplied a great deal of the information on which the pedigree is built. I. 1 and 2 said to have been normal. II. 1 and 2, English, normal. II. 3, Italian, first wife of II. 4, also an Italian. II. 5, his second wife an Italian, alive, normal. II. 6, three brothers and sisters of II. 5, no oedema. III. 1, normal, only child. III. 2, Italian family by first marriage of II. 4, nothing known with reference to them. III. 4, mother of Dr Rolleston's patients. When seen by him was 45 years of age and had suffered from oedema for 35 years. III. 3, III. 5, III. 6, normal, but all died in infancy before five years. All the fourth generation are dark, three however, viz. IV. 4, IV. 5 and IV. 7 being much lighter than the remaining four. IV. 1, aged 27, normal, IV. 2, died at age of five. IV. 3, aged 24, normal. IV. 4, ♀, aged 22, affected with oedema since the age of 14 or perhaps earlier, oedema disappears after rest in bed for some days. IV. 5, legs oedematous, died of phthisis at the age of 16. IV. 6, now (1908) aged 14, normal. IV. 7, aged 12, has enlarged legs, which however IV. 4 says are not due to oedema. (See Bibl. No. 16.)

Fig. 69. *Nonne's Case.* Eight individuals affected with congenital elephantiasis in three generations. The affection involved the legs and feet, in one case the hands and legs. The pedigree is wanting in details and the names and ages of certain individuals in the text do not correspond with those in Nonne's table. I. 2, died at age of 64, is stated to have received a fright, at the sight of a dropsical woman, while carrying II. 1. I. 1, husband of I. 2, not mentioned in text, presumably healthy. II. 1, ♀, died at age of 64, unmarried, more severely affected with elephantiasis than any other member of the family. II. 2, II. 4, II. 6, II. 8, II. 10 free from oedema, all married. II. 2 died at age of 60. II. 4 aged 52. II. 10 is mentioned in Nonne's text but not in his table. II. 11, affected with elephantiasis all her life. She married II. 12 who was healthy, died at age of 66 of pneumonia. III. 1, 2, 3, 4, normal families, number of individuals and sex unknown. III. 5, three normal individuals, sex and order of birth not given (not mentioned at all in Nonne's pedigree although in text). III. 6, aged 41. III. 8, 39, no mention of III. 7 or III. 9, but both presumably healthy. III. 10, ♂, ship stoker, affected from birth with elephantiasis in both feet and legs. In text his name is given as Heinrich Hansen, aged 34, whereas in pedigree he is called Heinrich Mandt, aged 26. The swelling begins below the knees and extends to the feet, is hard and brawny and pits easily on pressure. Circumference of R. calf 41 cm., left 38. Bones normal. III. 11, sister of III. 10 in text called Helene Mandt and 30 years of age, in pedigree Emilie Mandt, aged 28. Married one Gehl. In the text her children by him are called Mandt, in pedigree Gehl. Her left leg

and foot noticed to be affected at birth, and this has increased in size, R. leg unaffected. IV. 1 and IV. 2, families of III. 6 and III. 8, normal, number and sex not stated. IV. 3, ♂, born with "thick legs" in high degree, dead. IV. 4, ♀, born at term but acephalic. Hands, feet and legs affected. IV. 5, six years old, ♀, healthy and strong, but affected from birth with thickening on R. leg and foot. IV. 6, ♂, aged one year, born at term, healthy and strong but showing the rudiments of the family trouble in the feet and toes. (See Bibl. No. 17. This case was also the subject of a communication by Nonne entitled Elephantiasis congenita. *Deutsche med. Wochenschr.* Leipzig, 1890, XVI. p. 1124.)

Fig. 70. *Lortat-Jacob's Case.* I. 1 and 2, nothing stated. II. 2, congenital oedema of feet, no other data given. II. 3, sister of II. 2, also affected. II. 4, brother with oedema. III. 1 and 2, normal although III. 2 had two children affected. III. 3, first cousin of III. 2, feet oedematous from birth onwards. III. 4, no oedema, but has congenital luxation of thigh. IV. 1, aged five, oedema of legs and feet since birth. IV. 2, aged 10 months, born at term with oedematous foot, the oedema extending up to the knee. (See Bibl. No. 18.)

Fig. 71. *Meige's Case.* In Meige's Case eight people were affected in four generations. I. 1, both legs up to the knees, the seat of chronic trophoedema. I. 2, wife of I. 1, presumably normal. II. 2, aged 60, affected with trophoedema on both sides up to the knee but not higher. II. 1, husband of II. 2 presumed to be unaffected. III. 2, aged 40, affected like her mother II. 2 on both sides up to the level of the knee. She is quite well otherwise. Two of her brothers, III. 3 and III. 4, were also affected. The one who died at the age of 25 had oedema of both lower limbs involving feet, ankles, calves and *thighs*. He died after 48 hours illness, the family alleging that the oedema had "spread to his heart." The other brother, III. 4, died at the age of 27 of some unknown cause. He was affected like his sister, III 2, on both sides but only up to the knees. IV. 1, aged 21, extensive chronic oedema affecting both lower limbs up to the groins. It began at the age of 13 in the feet, involving in succession ankles, calves, and legs up to the knees where it remained stationary till she was 17. After this the thighs became affected. There was no pain in the swollen limbs but the swelling was increased at menstrual periods. At five different times she has had "acute attacks" accompanied by great swelling and pain in the limbs. IV. 2, aged 17, had suffered from chronic oedema for five years. It began in the R. foot and ankle, the calf becoming involved later. The oedema did not extend for a time beyond the knee. The left leg was perfectly normal. At a later period the oedema involved the whole of the R. lower limb up to the groin. No organic disease could be detected but there was a condition of hypaesthesia on the right side, probably hysterical in character. A comparison of the lower limbs of III. 1 and III. 2 showed the measurements given below. IV. 3, aged 13, shows signs of oedema in the right foot and lower half of R. calf. Left foot normal. (See Bibl. No. 10.)

	III. 1		III. 2	
	R.	L.	R.	L.
Circumference at level of malleolus	29 cm.	28 cm.	32 cm.	23 cm.
,, of calf (average) ..	42 ,,	39 ,,	42 ,,	31 ,,
,, at knee	43 ,,	38 ,,	44 ,,	32 ,,
,, thigh (average) ...	52 ,,	48 ,,	55 ,,	45 ,,

Fig. 72. *Milroy's Case.* Milroy's cases occurred in an old American family "H," the pedigree of which on the side of I. 1 can be traced back two hundred and fifty years. The oedematous condition entered the family by the marriage of I. 1 and I. 2. Although I. 2 was not the subject of oedema a near relative—probably a sister, I. 3, had the disease. II. 2, the son of I. 1 and I. 2, had both legs enormously enlarged. He was born in 1784 and died of enteric at the age of 78. He was twice married. By his two wives he had nine children, namely one son and eight daughters (generation III. in pedigree). III. 1 died at the age of four but nothing is reported about him being oedematous or otherwise. III. 2, aged 82, at the time of Milroy's publication, had one leg the seat of oedema. Her husband, III. 3, presumably normal. III. 4, aged 80, no oedema. III. 5, her husband. One of their sons, IV. 8 was affected. III. 6 and 7, twin sisters. III. 6, died in infancy but already had one oedematous leg. III. 7, died at the age of 32, no oedema. III. 8, husband of III. 7. IV. 14, their child, affected. III. 9, alive, aged 75, born with one enlarged foot. When between 20 and 30 years of age her other leg was injured in a carriage accident and thereafter it began to enlarge till it attained enormous dimensions. III. 10, her husband, presumably normal. III. 11, alive, aged 73, has one enlarged foot and ankle, married III. 12. III. 13, died young, no information. III. 15, alive, aged 66. Until 12 years of age both her lower extremities were of normal size but in that year one ankle became enlarged without apparent cause and remained so. The other leg was of normal dimensions. III. 14 and III. 16 her two husbands. Fourth

Generation. IV. 1—6, children of III. 2 and III. 3. The three sons had each one foot enlarged, the limbs of the three daughters being normal. The order of birth of these children is not stated. IV. 7 and IV. 8, the eight children of III. 4 and III. 5. One of the eight, a boy, has one enlarged foot, the other seven being normal, three of the seven are dead. IV. 9—IV. 14, the five children of III. 7 and III. 8, the youngest—sex?—has an enlarged oedematous foot, the others being normal. IV. 15 and 16, the four children of III. 9 and III. 10. The youngest, sex not stated, has an oedematous leg. IV. 17, IV. 18, IV. 19, three children of III. 11 and III. 12. Only the boy, III. 19, was affected and suffered from enlarged foot. When he reached maturity his testicles began to swell and progressed to such an extent that one of them had to be removed. Strange to say as the testicle increased in size the oedematous foot decreased and after the operation became and remained normal. IV. 20 and IV. 21, children of III. 15. By her first husband the three children (sex not stated) were normal, whereas by her second husband she had a son, IV. 21, with great oedema of both feet and both legs up to the level of the knees. The left side was more oedematous however than the right. IV. 21, an American clergyman, was the patient who consulted Milroy and whose condition led to Milroy's enquiry into the family history. V. 1, 2, and 3, the 11 grandchildren of III. 2 and III. 3. Of them is stated that two, viz. a son and a daughter, were affected with the family disease. In both cases the foot alone on one side was affected. The respective parents of these children are not given. V. 4, nine grandchildren of III. 4 and III. 5, sex and order of birth not stated, none of them were oedematous. V. 5, V. 6, V. 7, nine grandchildren of III. 7 and III. 8. Their order of birth and in seven cases, V. 5, the sex not stated. V. 6, the son of the eldest daughter of III. 7 has both legs enlarged, while V. 7 the son of the eldest son of III. 8 has one foot enlarged. The exact parentage of V. 5, V. 6 and V. 7 is not clear from Milroy's account. V. 9, V. 10, V. 11, V. 13, grandchildren of III. 9 and III. 10. One, a son, V. 10 has an enlarged foot, the other children being unaffected. V. 8 wife of V. 9, and V. 12 the wife of V. 11, presumably normal. V. 14 and 15, two normal grandchildren (sex?) of III. 11 and III. 12. V. 16, 13 normal grandchildren of III. 15. The exact parentage of these 13 children not stated. VI. 1, three normal children (sex?) of V. 8 and V. 9. VI. 2, has one enlarged foot. VI. 3, sex not stated but alleged not to be oedematous. VI. 4, has both feet enlarged. VI. 5, two great grandchildren of III. 15, age and sex not given, but stated not to suffer from oedema. In Milroy's family of 97 individuals there were 22 cases of oedema or about 23 % of the whole number. With three exceptions the disease was congenital and progressive, the exceptions being: (1) III. 9, whose normal leg began to enlarge after a carriage accident. (2) III. 15, who was normal till the age of 12. (3) IV. 19, whose oedema disappeared after the removal of a testicle. With reference to the sex incidence there were twelve affected males, eight females and two the sex of which was not determined. (See Bibl. No. 11.)

Fig. 73. *Lannois' Case.* Trophoedema in three generations and limited to female sex. I. 1, affected for a long time with epilepsy, died ultimately from heart disease. I. 2, aged 55, alive and well, but with oedema of left leg especially dorsum of foot. The oedema began insidiously and painlessly during her first pregnancy at 21. It was the subject of exacerbations especially in connection with pregnancy. II. 1, sex not stated, but normal. II. 2, ♀, aged 38, born at full time. About the age of 10 she began to show oedema of feet and ankles but this went away, to recur again at 18 when she had an attack of bronchitis. At this time her right calf suddenly became swollen and red but not very painful, and it has remained swollen. Married at 24 and in one and a half years had a daughter (III. 1), no other pregnancies. During her pregnancy she became very fat and signs of oedema began to show themselves in left leg and this gradually increased. Otherwise she is well. II. 4, healthy, II. 6, ♂, aged 28, thin and has a cough. II. 7, ♀, aged 21, at age of 13 feet began to swell at night. At the age of 19 an attack of scarlet fever with swollen feet and legs. This has continued. II. 8, four children died in childhood. II. 9, three alive and well, sex not stated. III. 1, died at age of one and a half from pneumonia following measles. III. 2, aged five, has convulsions, sex not stated. III. 3, aged three, developed normally up to 14 months when without apparent cause development was arrested, oedema of both feet. III. 4, normal child. (See Bibl. No. 12.)

Fig. 74. *Jopson's Case.* Congenital elephantiasis in two brothers. II. 1, suffered from similar condition in youth but it disappeared as he grew up. II. 2, presumably normal. III. 1, two healthy children, alive, sex not stated. III. 2, two healthy children, dead, sex not stated. III. 3, 4, miscarriages. III. 5, ♂, aged four, marked swelling of both lower limbs involving feet and legs up almost to knees. The swelling is hard and pits on prolonged pressure. Over the dorsum of the feet the oedema is very marked and the skin white. At birth the prepuce was also involved and had to be circumcised. Bones not enlarged. III. 6, ♂, one and a half years old, condition also congenital but less marked than in III. 4, in so far that the swelling only extends up to the ankles. Prepuce not affected. III. 4 and III. 5 are otherwise healthy and intelligent. (See Bibl. No. 13.)

PLATE XIV. Fig. 75. *Sutherland's Case.* V. 10 is a child of seven months. In December, 1908, the child was shown at the Royal Society of Medicine. She was accompanied by her sister (V. 7), who was there

FIG. 1

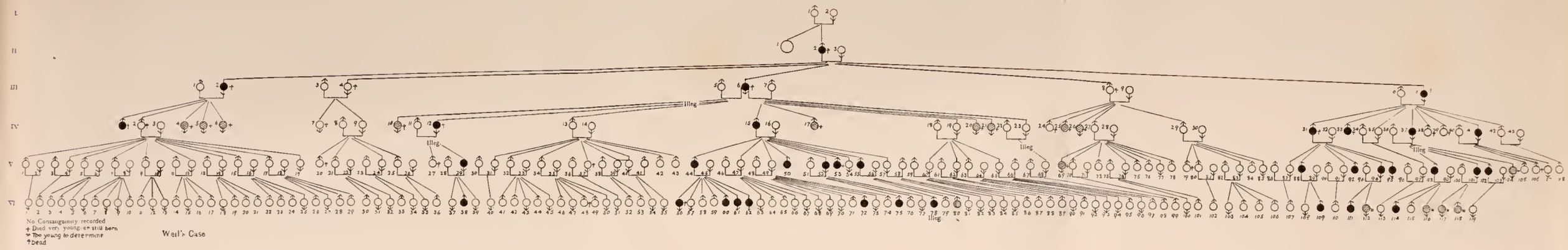


FIG. 2

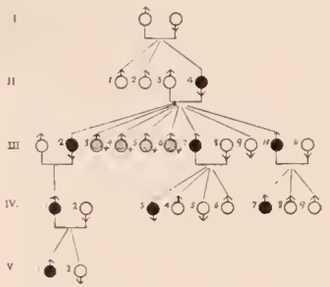


FIG. 3

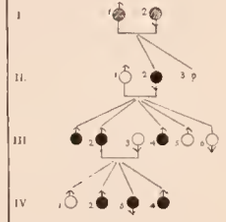


FIG. 4

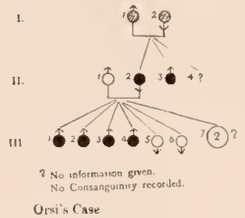


FIG. 5

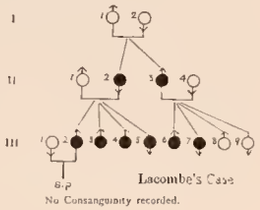


FIG. 6

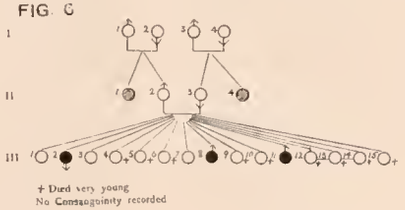


FIG. 7

FIG. 8

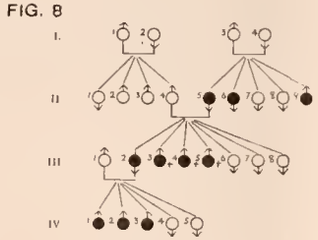


FIG. 9

FIG. 10

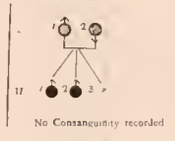


FIG. 11

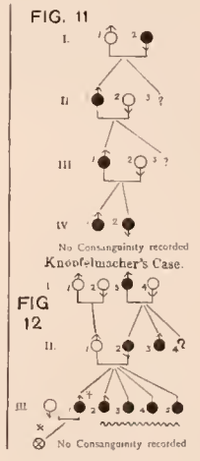


FIG 12

FIG. 13

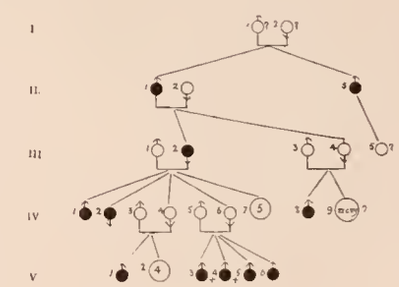
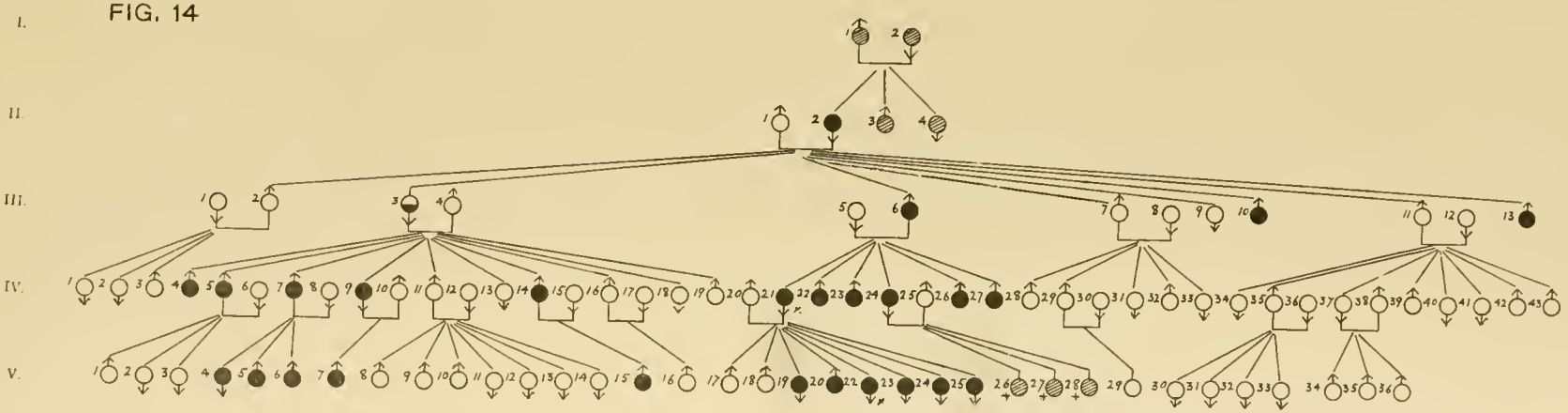


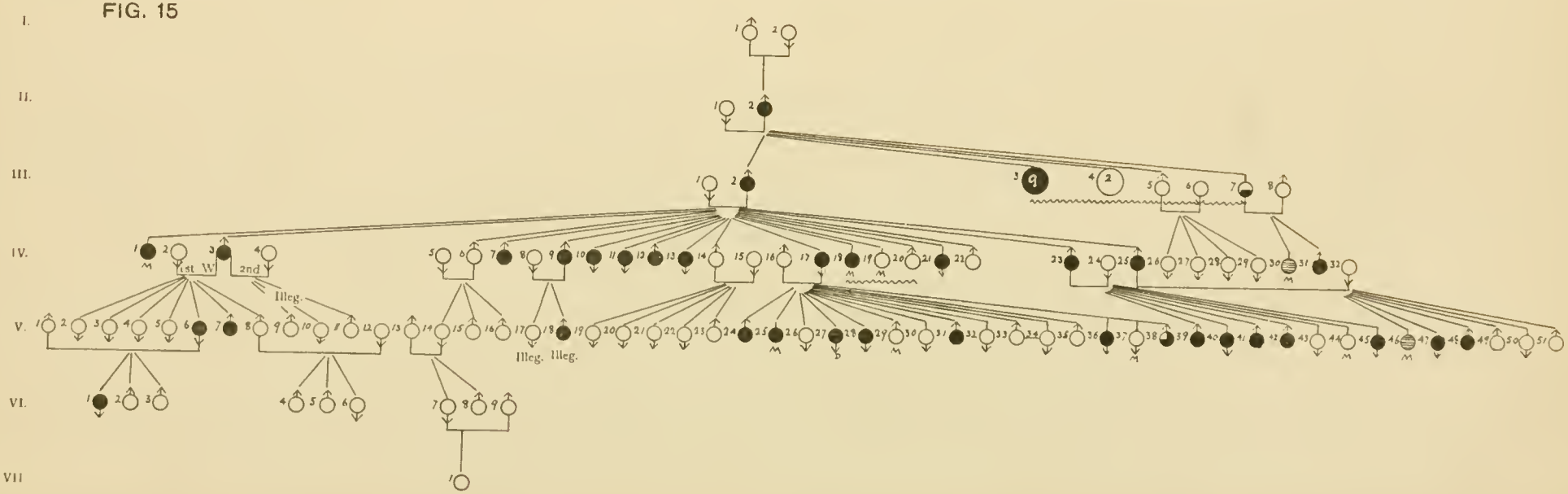
FIG. 14



No Consanguinity.
+Died at birth, no record
× *Subject to fits.*

Pearson's Case.

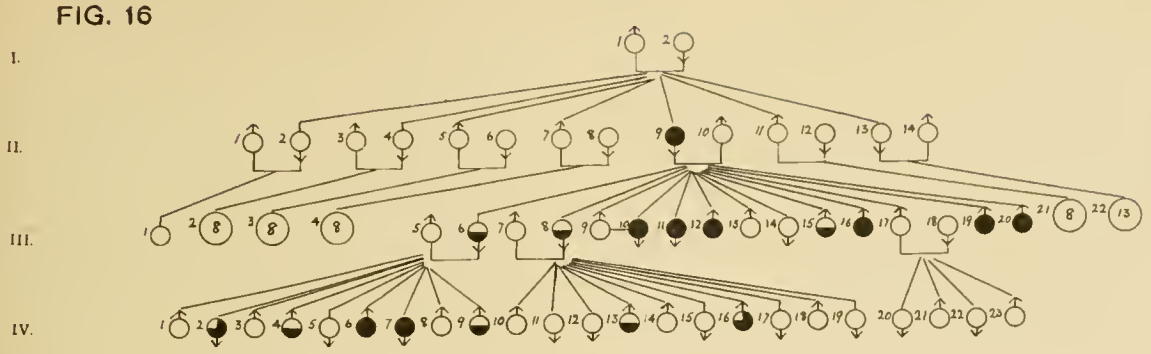
FIG. 15



No Consanguinity.
M=Miscarriage.

Lewis and Embleton's Case.

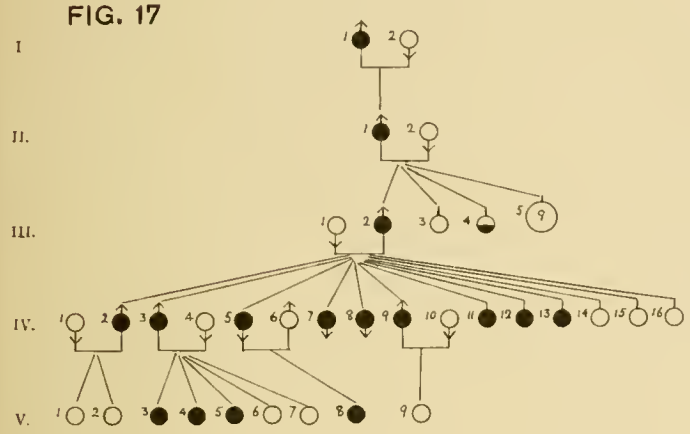
FIG. 16



No Consanguinity recorded.

Parker and Robinson's Case.

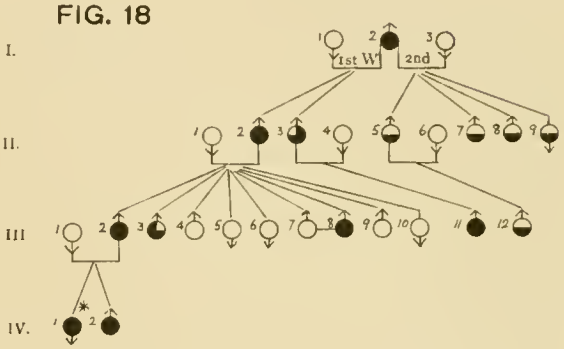
FIG. 17



No Consanguinity recorded.

Fotherby's Case.

FIG. 18



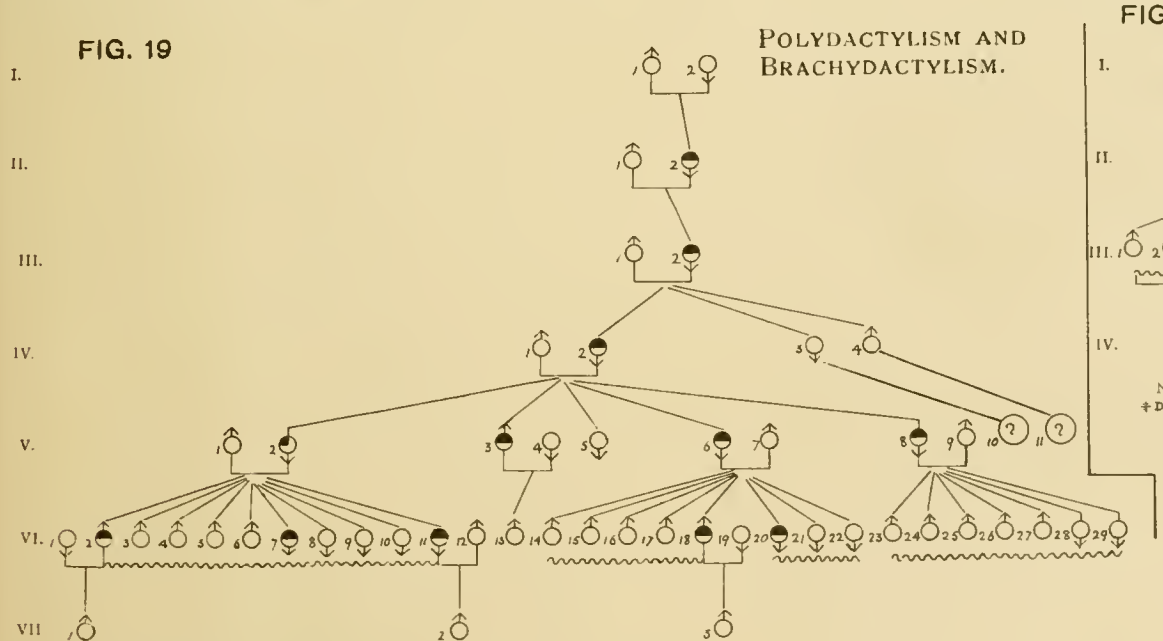
No Consanguinity recorded.

Mayer's Case.

* *Syndactylism alone*

FIG. 19

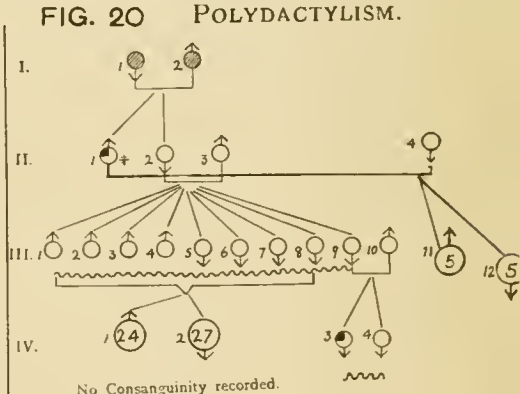
POLYDACTYLISM AND BRACHYDACTYLISM.



No Consanguinity.

Mathews' Case.

FIG. 20 POLYDACTYLISM.

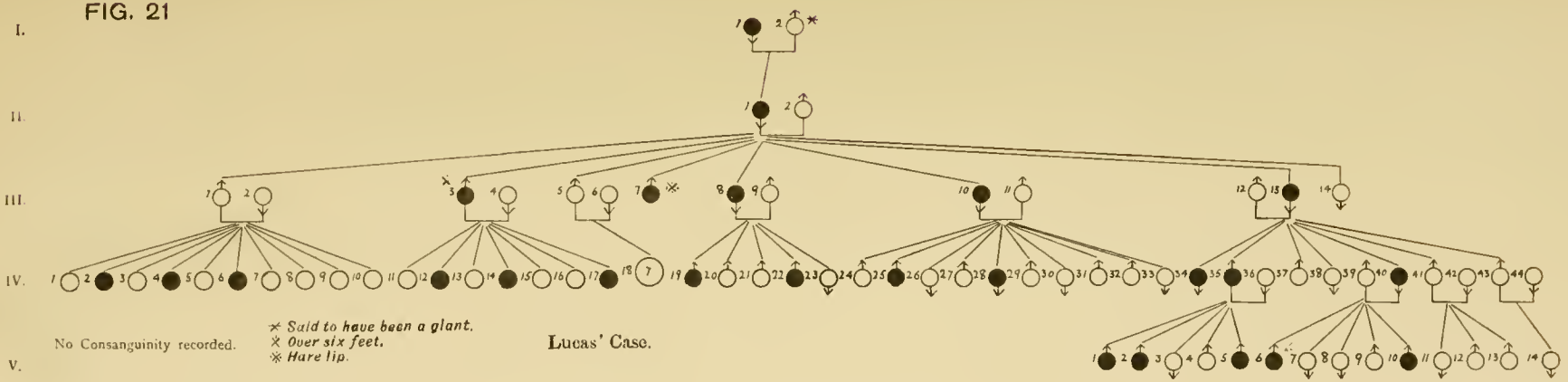


No Consanguinity recorded.

* Doubtful left or right

Struther's Case.

FIG. 21

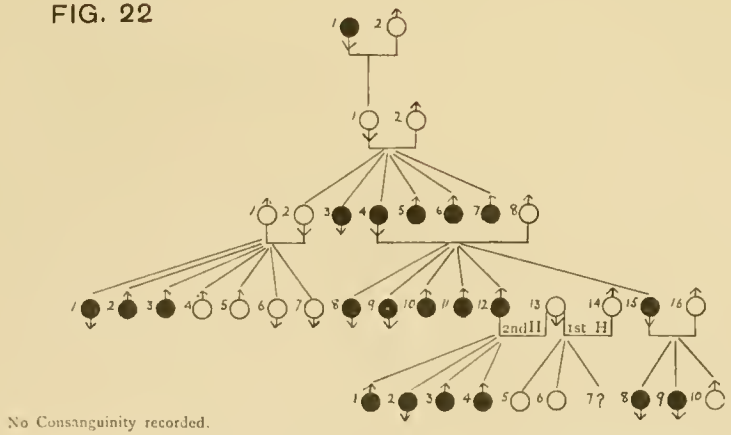


No Consanguinity recorded.

* Said to have been a giant.
※ Over six feet.
※ Hare lip.

Lucas' Case.

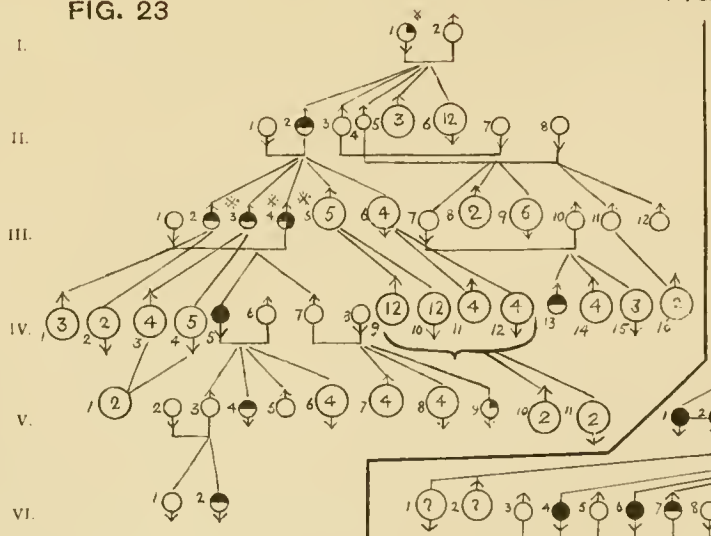
FIG. 22



No Consanguinity recorded.

Smith and Norwell's Case.

FIG. 23

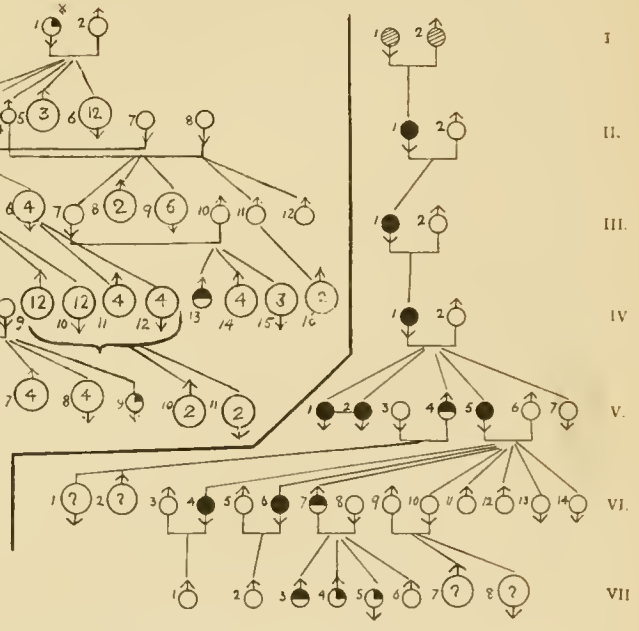


No Consanguinity recorded.

No birth order given.
*·Side of unilateral affection uncertain.

Struther and Wilson's Case.

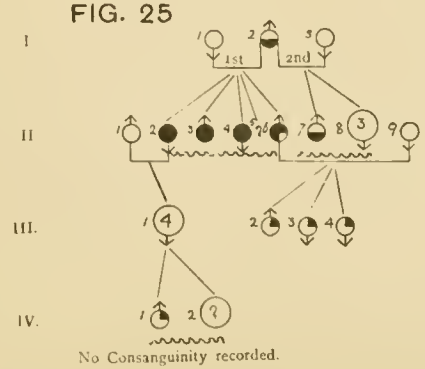
FIG. 24



No Consanguinity recorded.

McKellar's Case.

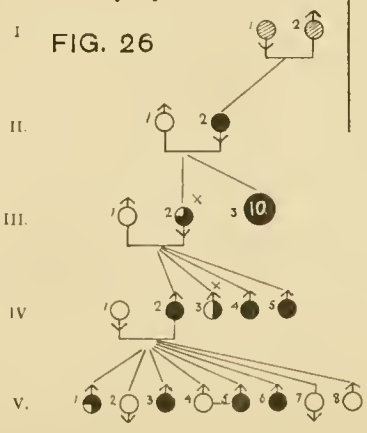
FIG. 25



No Consanguinity recorded.

Wilson's Case

FIG. 26

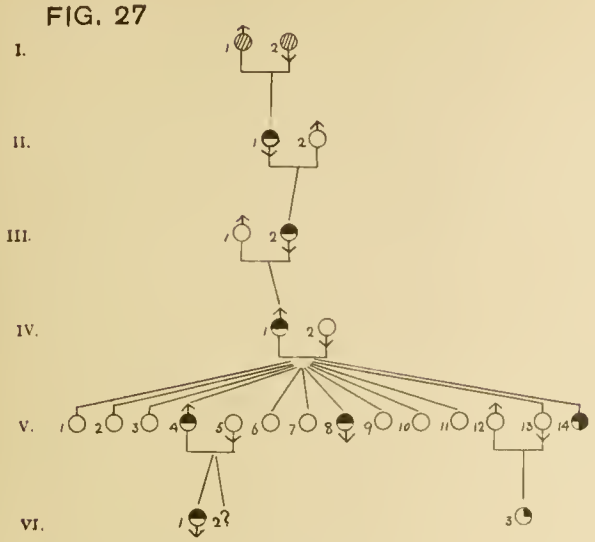


No Consanguinity recorded.

※ Unaffected side unknown.

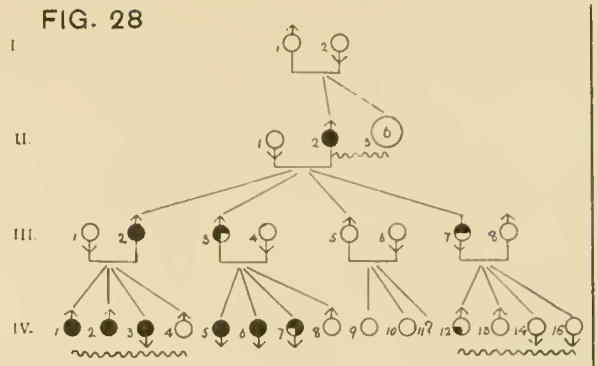
Carlisle's Case.

FIG. 27



No Consanguinity recorded. Greene's Case.

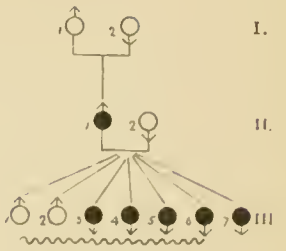
FIG. 28



No Consanguinity recorded.

Riville's Case.

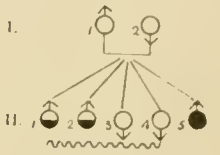
FIG. 29



No Consanguinity.

Morrish's Case

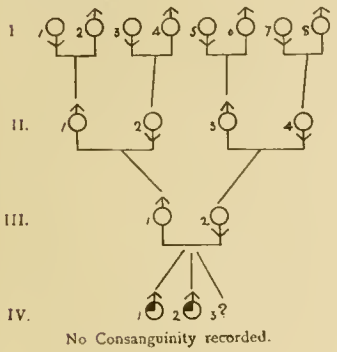
FIG. 30



No Consanguinity.

Withrow's Case.

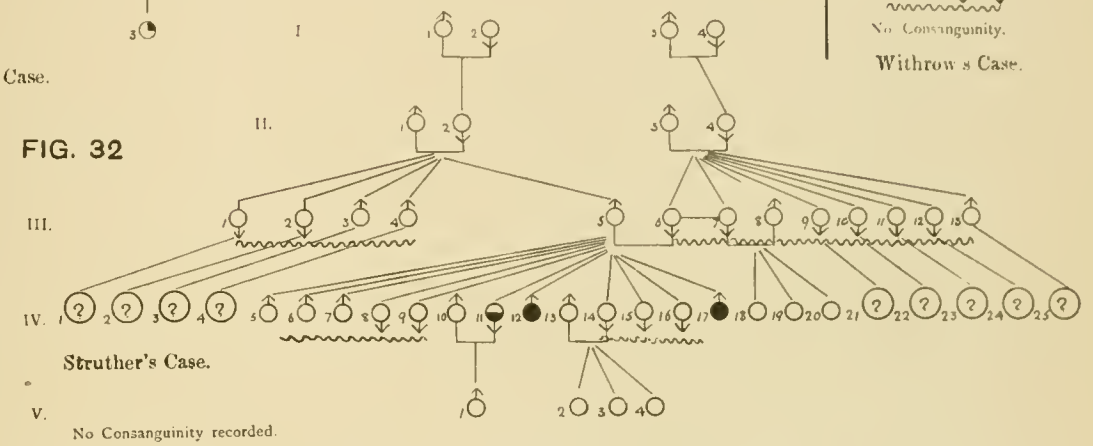
FIG. 31



No Consanguinity recorded.

Struther's Case

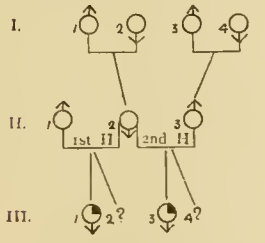
FIG. 32



Struther's Case.

No Consanguinity recorded.

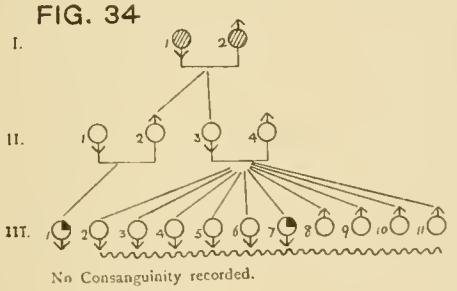
FIG. 33



No Consanguinity recorded.

Struther's Case

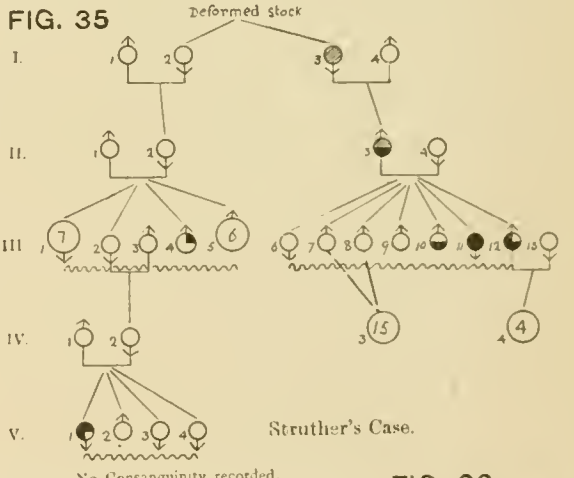
FIG. 34



No Consanguinity recorded.

Struther's Case.

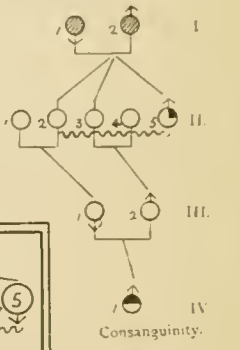
FIG. 35



Struther's Case.

No Consanguinity recorded.

FIG. 36

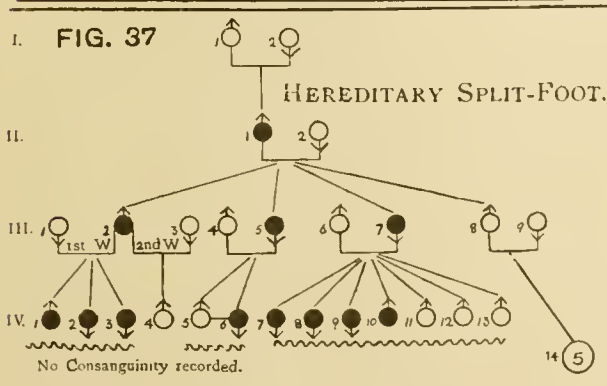


Consanguinity.

Attlee's Case.

FIG. 37

HEREDITARY SPLIT-FOOT.

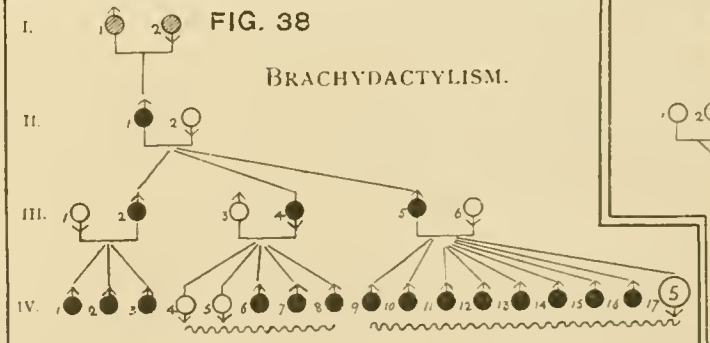


No Consanguinity recorded.

Bédart's Case.

FIG. 38

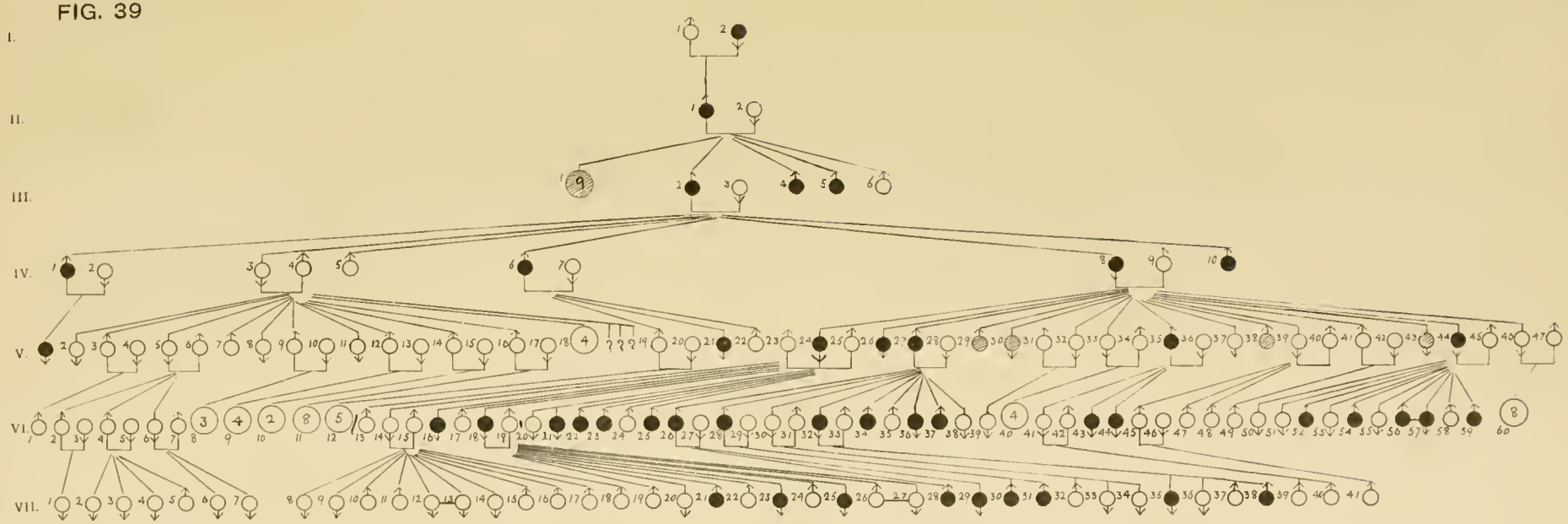
BRACHYDACTYLISM.



No Consanguinity recorded.

Mercier's Case.

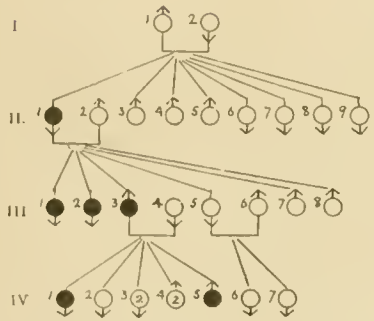
FIG. 39



No Consanguinity recorded. *Dwarfism present*

Drinkwater's Case.

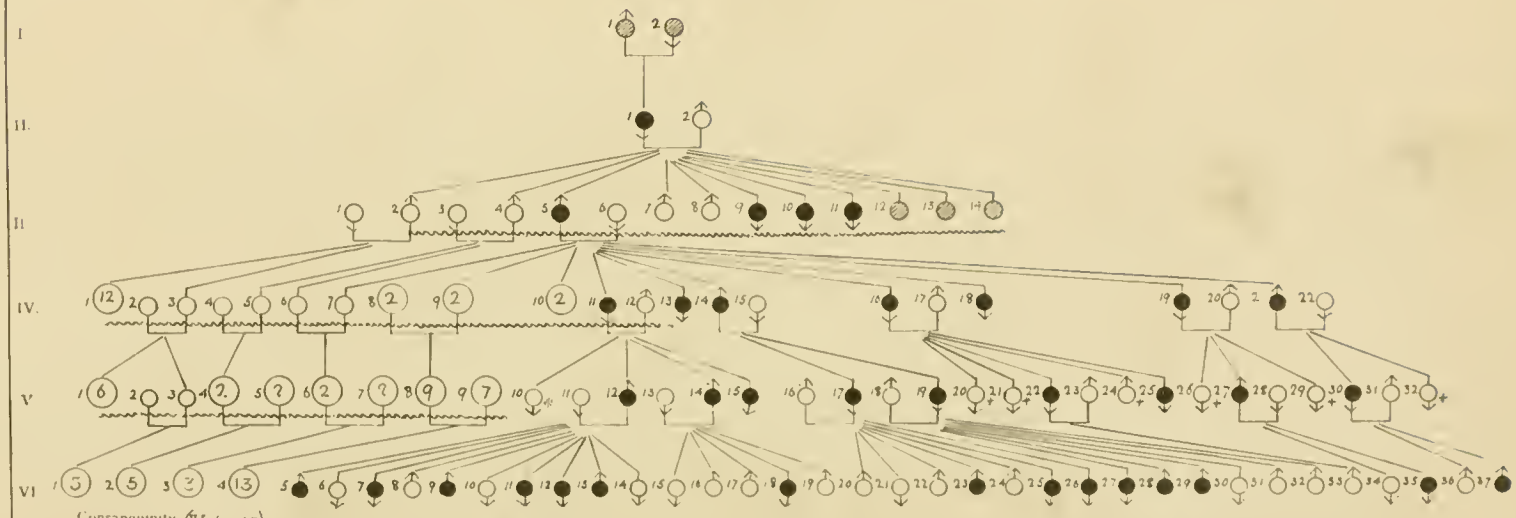
FIG. 40



No Consanguinity recorded.

Hasselwander's Case.

FIG. 41



Consanguinity. (IV 6 and 7)
+ 2 of these 7 normals had 2 normal children

Dwarfism present

Farabee's Case.

FIG. 42 Klebs' Case (i.)

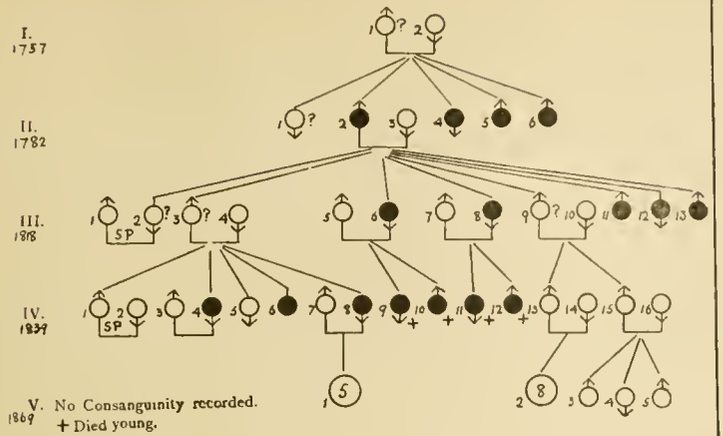


FIG. 43 Klebs' Case (ii.)

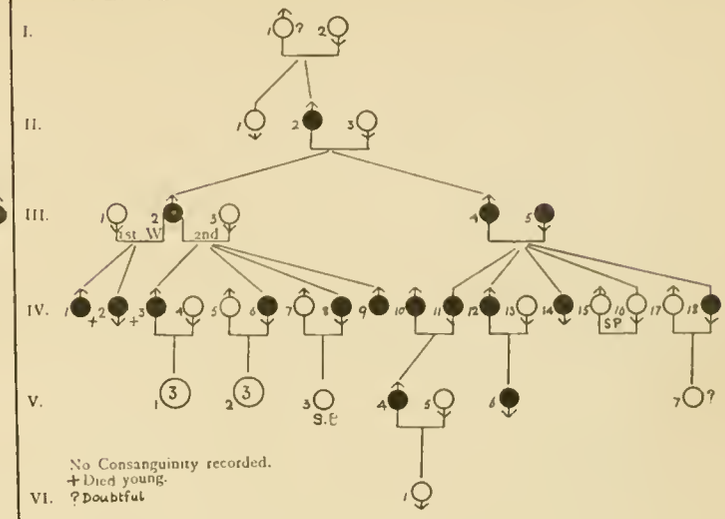


FIG. 44 Rivers' Case (i.)

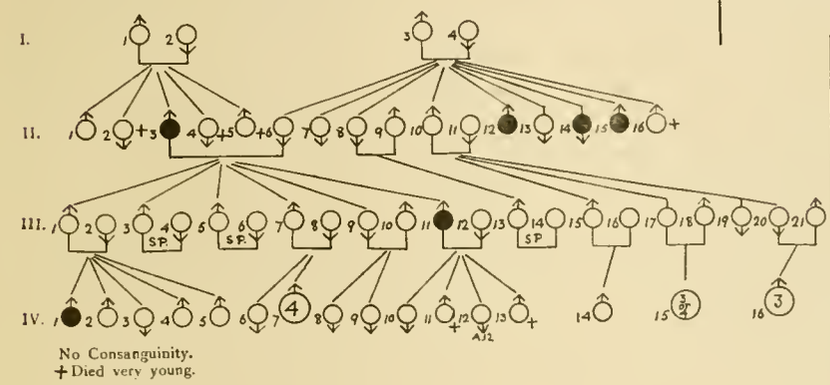


FIG. 45 Rivers' Case (ii.)

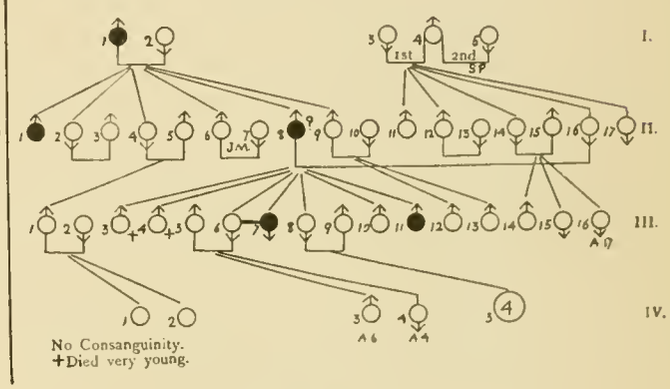


FIG. 46 Rivers' Case (iii.)

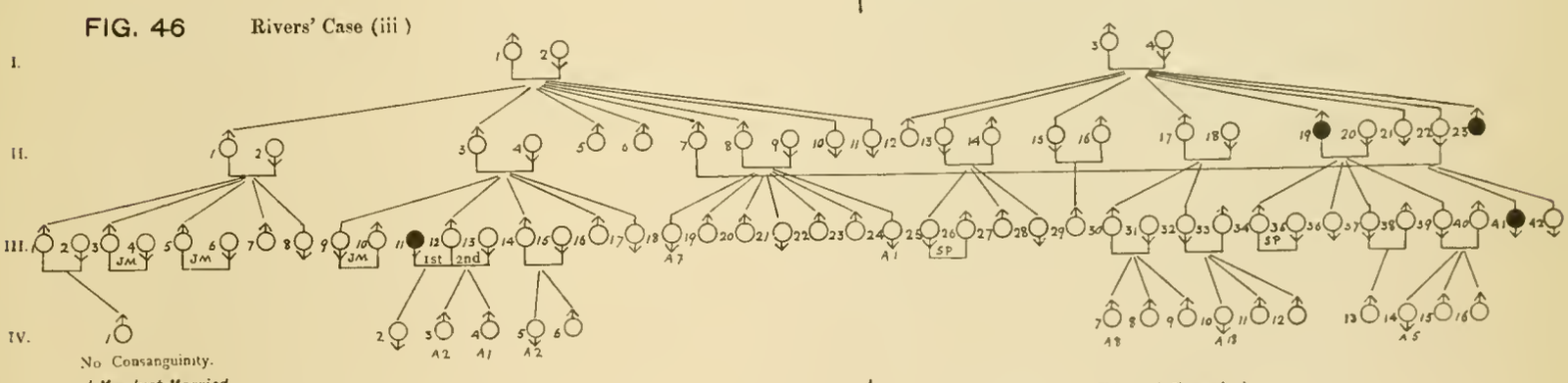


FIG. 47 Rivers' Case (iv.)

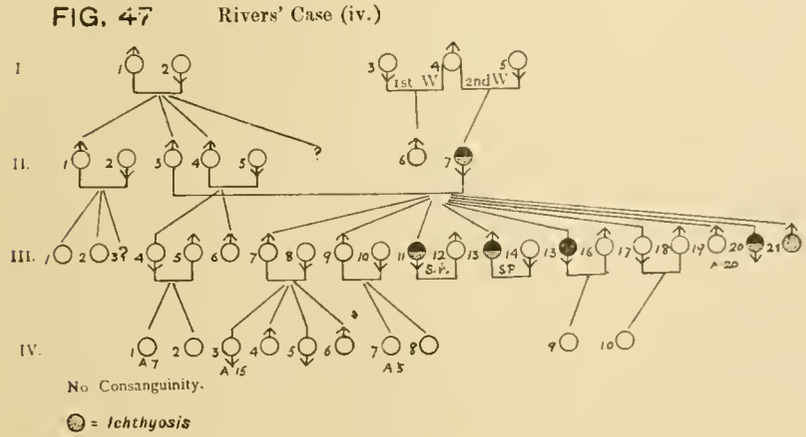


FIG. 48 Rivers' Case (v.)

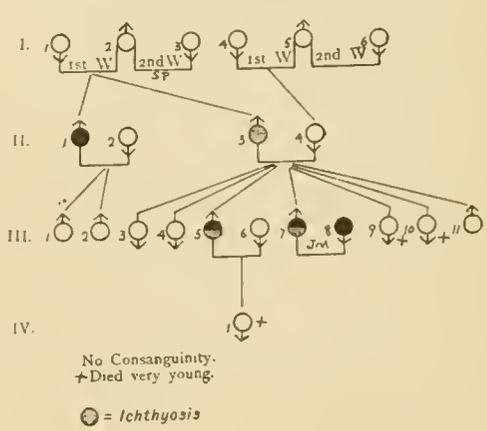
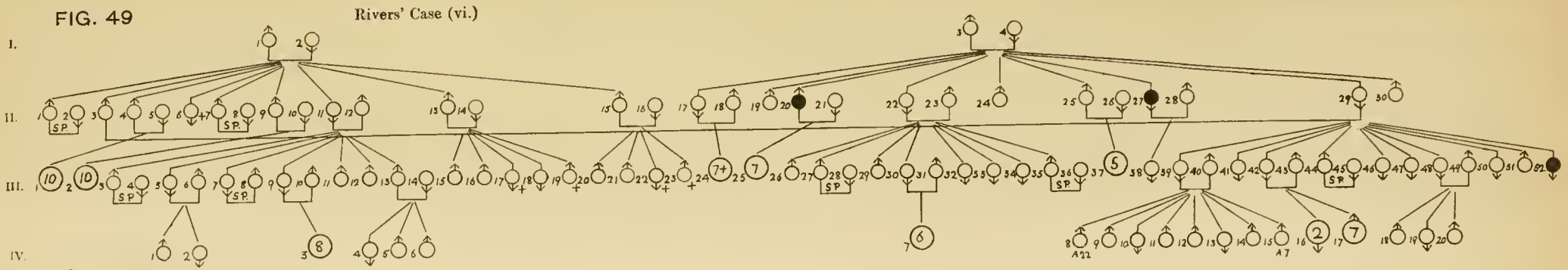
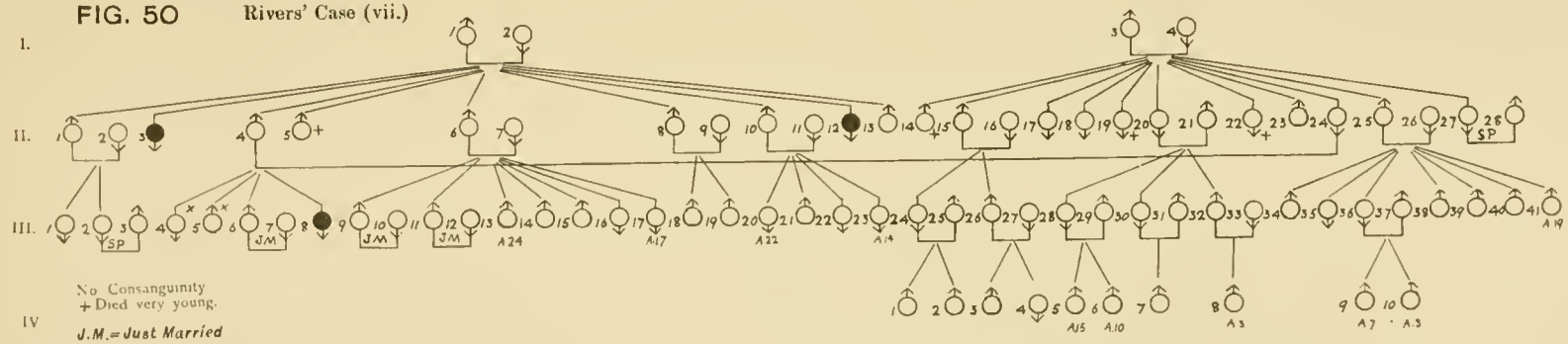


FIG. 49 Rivers' Case (vi.)



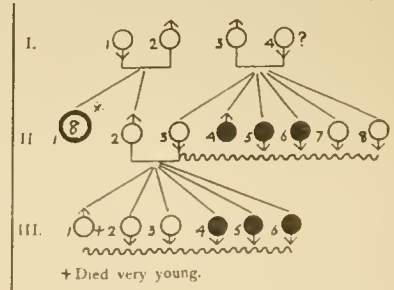
No Consanguinity.
+ Died very young.

FIG. 50 Rivers' Case (vii.)



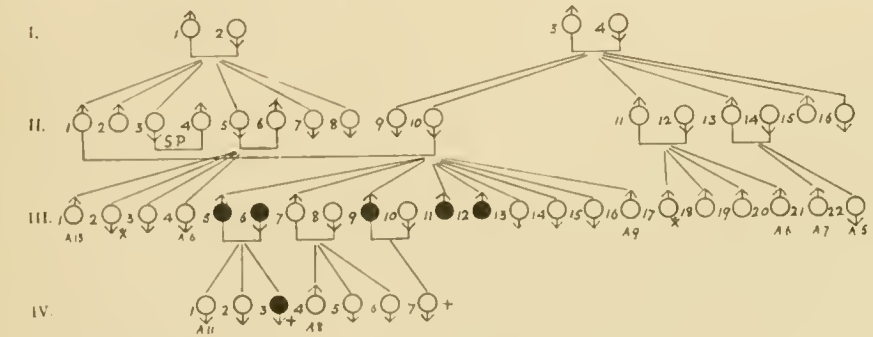
No Consanguinity
+ Died very young.
J.M. = Just Married

FIG. 51 Pearson's Case (i.)



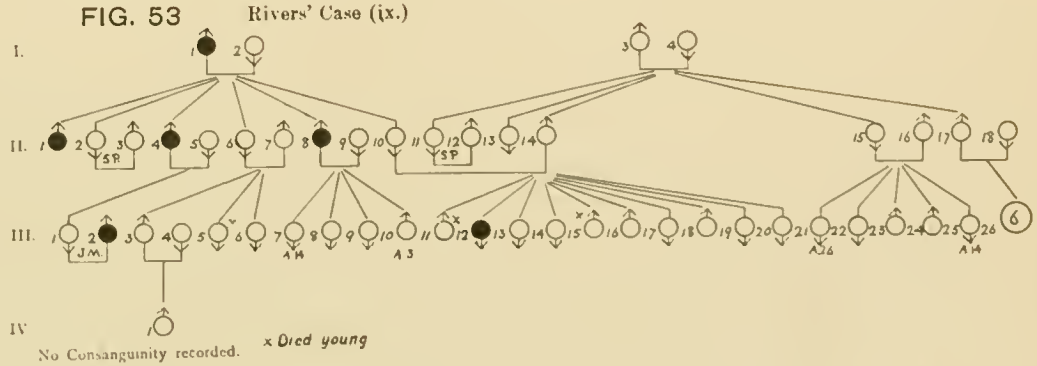
+ Died very young.
☆ Epileptics and neurotics

FIG. 52 Rivers' Case (viii.)



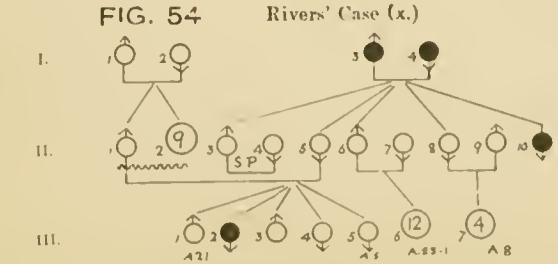
No Consanguinity.
+ Died very young.
x Died young.

FIG. 53 Rivers' Case (ix.)



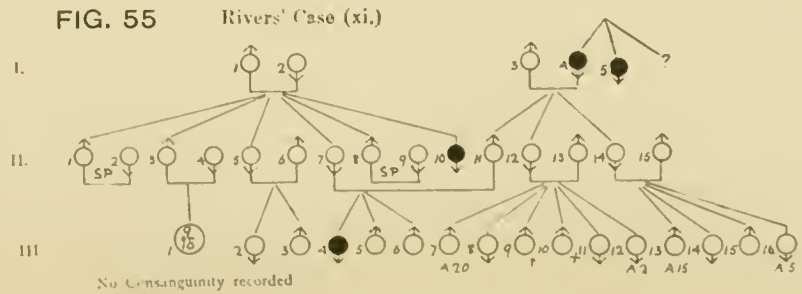
No Consanguinity recorded. x Died young

FIG. 54 Rivers' Case (x.)



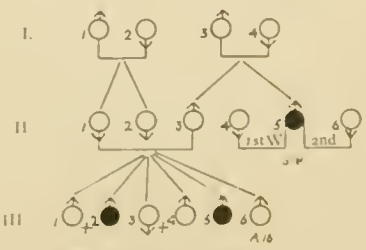
No Consanguinity recorded

FIG. 55 Rivers' Case (xi.)



No Consanguinity recorded
+ Died very young.

FIG. 56 Rivers' Case (xii.)



No Consanguinity recorded
+ Died very young

FIG. 57 Stephenson's Case.

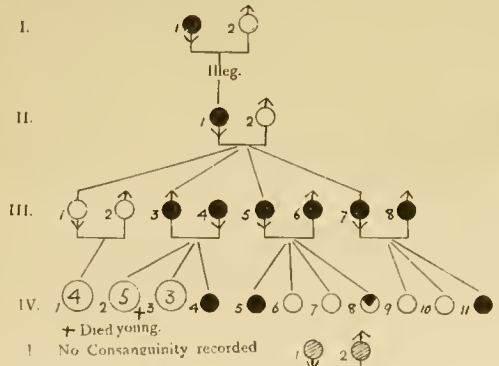


FIG. 58 Moos' Case.

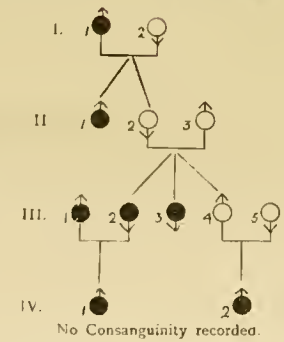


FIG. 59 Dahl's Case.

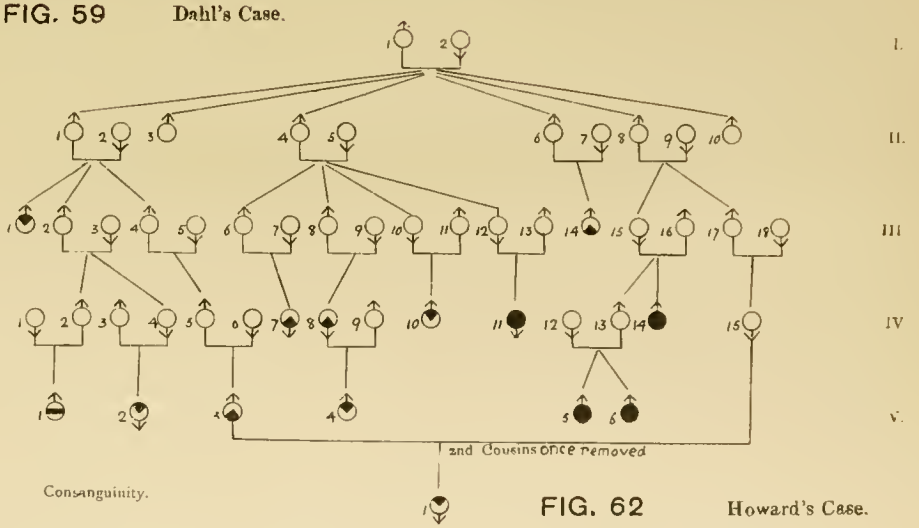


FIG. 60 Townsend's Case.

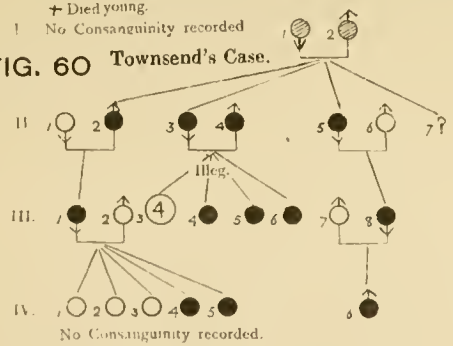


FIG. 61 Hartmann's Case.

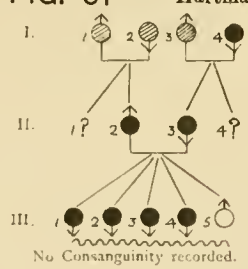


FIG. 62 Howard's Case.

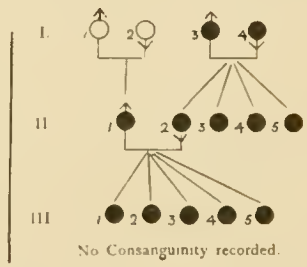


FIG. 63 Dahl's Case.

For this Plate ● = Deaf Mutism ◔ = Insanity ◒ = Idiot ⊖ = Epileptic ◑ = Feeble minded

FIG. 64 LEGAL AND ADMINISTRATIVE.

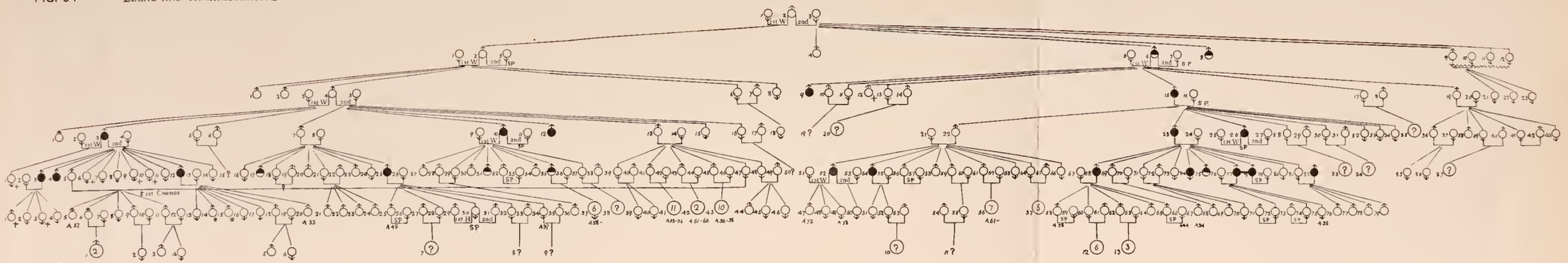


FIG. 65 LEGAL AND LITERARY.

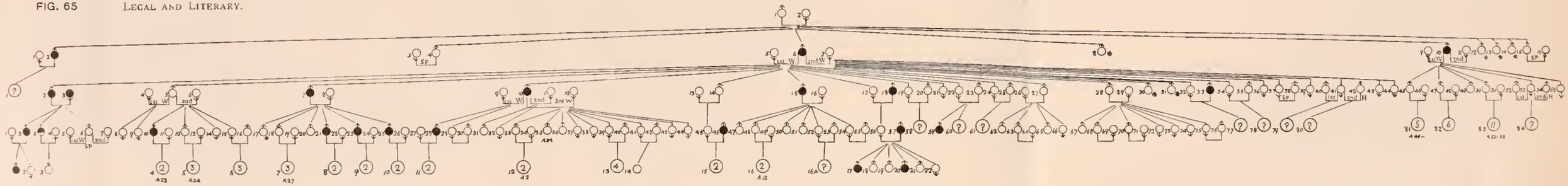
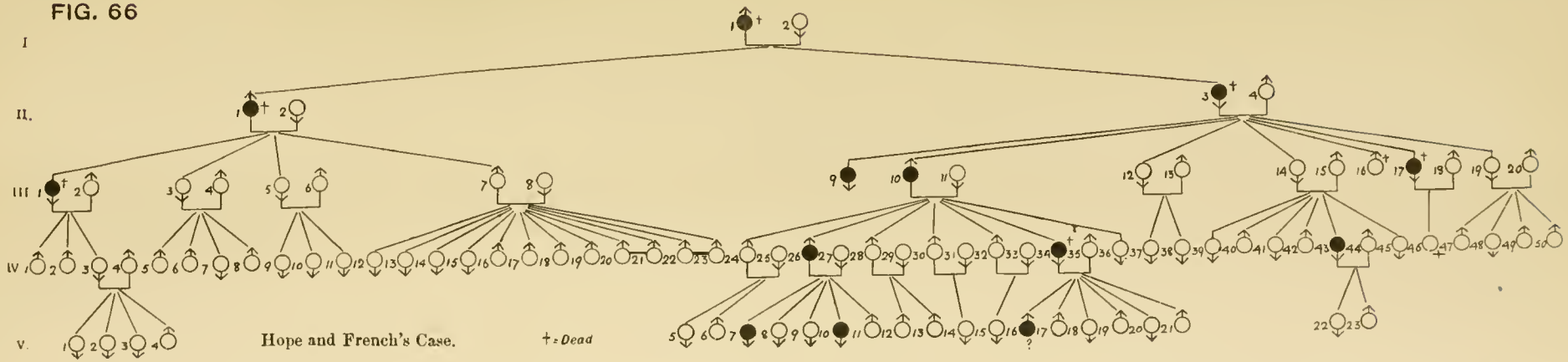


FIG. 66

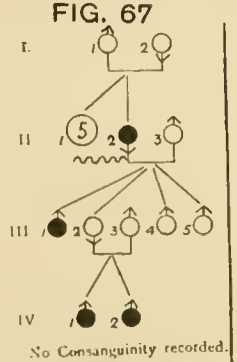


Hope and French's Case.

†=Dead

No Consanguinity recorded.

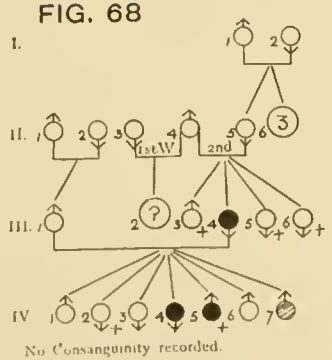
FIG. 67



No Consanguinity recorded.

Tobiesen's Case.

FIG. 68

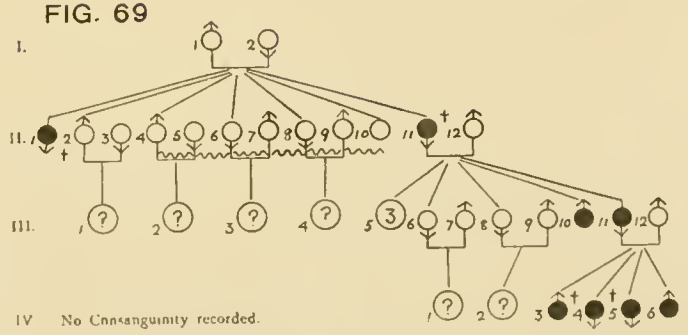


No Consanguinity recorded.

+ Died young.

Rolleston's Case.

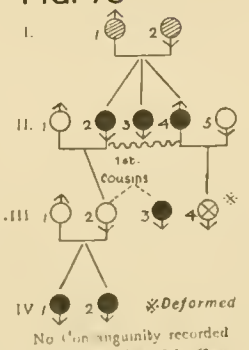
FIG. 69



No Consanguinity recorded.

Nonne's Case. †=Dead

FIG. 70

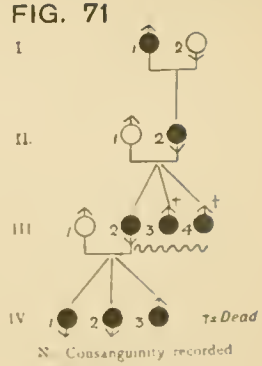


*Deformed

No Consanguinity recorded

Lortat-Jacob's Case.

FIG. 71

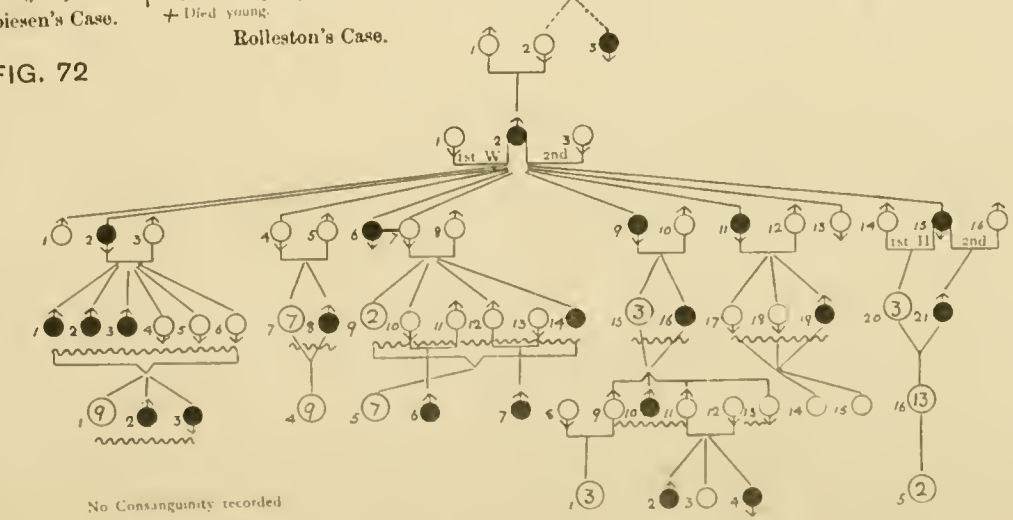


†=Dead

No Consanguinity recorded

Meige's Case.

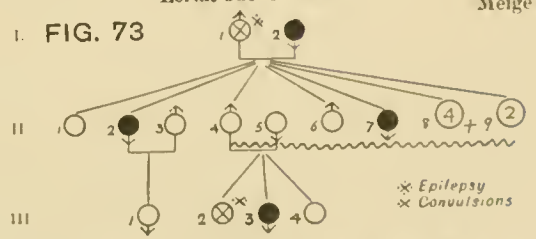
FIG. 72



No Consanguinity recorded

Milroy's Case.

FIG. 73



*Epilepsy

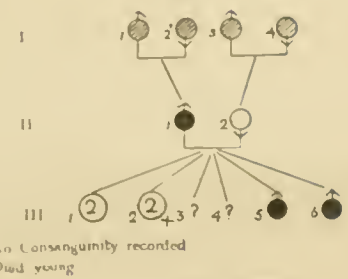
*Convulsions

No Consanguinity recorded.

+ Died young.

Lannois' Case.

FIG. 74



No Consanguinity recorded

+ Died young

Jopson's Case

UNIVERSITY OF LONDON

FRANCIS GALTON LABORATORY FOR NATIONAL EUGENICS

THE TREASURY OF HUMAN INHERITANCE

PART III

WITH 11 PLATES OF PEDIGREES AND 1 PLATE IN COLOURS

PLATES XIV—XXIV PLATE F

PEDIGREES 73—192

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1909

CONTENTS OF PART III.

(This will be replaced by a complete list of contents when the first volume is finished.)

SECTION IX α. *Angioneurotic Oedema.* By W. BULLOCH, M.D.

SECTION X α. *Hereditary Malformation of the Genital Organs. Hermaphroditism.* By W. BULLOCH, M.D.

SECTION XI *α*. *Insanity*. By A. R. URQUHART, M.D.

SECTION VI *β*. *Deaf-Mutism* (EUGENICS LABORATORY).

SECTION VII *β*. *Ability* (see Vol. I. p. 30). (EUGENICS LABORATORY.)

ILLUSTRATIVE PLATE.

The following Sections and continuations of Sections are in an advanced state: Hare Lip and Cleft Palate, Haemophilia, Dwarfism, Cataract, Unpublished Deaf-Mute Pedigrees, Tuberculosis, Insanity and Allied States (unpublished), Polydactyly, Congenital Defects of Bones, Dental Defects, etc., etc. Subscribers and others are earnestly requested (*a*) to contribute unpublished material, (*b*) to draw the Editor's attention to any family histories recently published in British or Foreign Journals. One or two "omnibus plates" will shortly be issued giving pedigrees of rare family characters too infrequent for, or incapable of, sectional classification.

Generation. IV. 1—6, children of III. 2 and III. 3. The three sons had each one foot enlarged, the limbs of the three daughters being normal. The order of birth of these children is not stated. IV. 7 and IV. 8, the eight children of III. 4 and III. 5. One of the eight, a boy, has one enlarged foot, the other seven being normal, three of the seven are dead. IV. 9—IV. 14, the five children of III. 7 and III. 8, the youngest—sex?—has an enlarged oedematous foot, the others being normal. IV. 15 and 16, the four children of III. 9 and III. 10. The youngest, sex not stated, has an oedematous leg. IV. 17, IV. 18, IV. 19, three children of III. 11 and III. 12. Only the boy, IV. 19, was affected and suffered from enlarged foot. When he reached maturity his testicles began to swell and progressed to such an extent that one of them had to be removed. Strange to say as the testicle increased in size the oedematous foot decreased and after the operation became and remained normal. IV. 20 and IV. 21, children of III. 15. By her first husband the three children (sex not stated) were normal, whereas by her second husband she had a son, IV. 21, with great oedema of both feet and both legs up to the level of the knees. The left side was more oedematous however than the right. IV. 21, an American clergyman, was the patient who consulted Milroy and whose condition led to Milroy's enquiry into the family history. V. 1, 2, and 3, the 11 grandchildren of III. 2 and III. 3. Of them is stated that two, viz. a son and a daughter, were affected with the family disease. In both cases the foot alone on one side was affected. The respective parents of these children are not given. V. 4, nine grandchildren of III. 4 and III. 5, sex and order of birth not stated, none of them were oedematous. V. 5, V. 6, V. 7, nine grandchildren of III. 7 and III. 8. Their order of birth and in seven cases, V. 5, the sex not stated. V. 6, the son of the eldest daughter of III. 7 has both legs enlarged, while V. 7 the son of the eldest son of III. 8 has one foot enlarged. The exact parentage of V. 5, V. 6 and V. 7 is not clear from Milroy's account. V. 9, V. 10, V. 11, V. 13, grandchildren of III. 9 and III. 10. One, a son, V. 10 has an enlarged foot, the other children being unaffected. V. 8 wife of V. 9, and V. 12 the wife of V. 11, presumably normal. V. 14 and 15, two normal grandchildren (sex?) of III. 11 and III. 12. V. 16, 13 normal grandchildren of III. 15. The exact parentage of these 13 children not stated. VI. 1, three normal children (sex?) of V. 8 and V. 9. VI. 2, has one enlarged foot. VI. 3, sex not stated but alleged not to be oedematous. VI. 4, has both feet enlarged. VI. 5, two great grandchildren of III. 15, age and sex not given, but stated not to suffer from oedema. In Milroy's family of 97 individuals there were 22 cases of oedema or about 23 % of the whole number. With three exceptions the disease was congenital and progressive, the exceptions being: (1) III. 9, whose normal leg began to enlarge after a carriage accident. (2) III. 15, who was normal till the age of 12. (3) IV. 19, whose oedema disappeared after the removal of a testicle. With reference to the sex incidence there were twelve affected males, eight females and two the sex of which was not determined. (See Bibl. No. 11.)

Fig. 73. *Lannois' Case.* Trophoedema in three generations and limited to female sex. I. 1, affected for a long time with epilepsy, died ultimately from heart disease. I. 2, aged 55, alive and well, but with oedema of left leg especially dorsum of foot. The oedema began insidiously and painlessly during her first pregnancy at 21. It was the subject of exacerbations especially in connection with pregnancy. II. 1, sex not stated, but normal. II. 2, ♀, aged 38, born at full time. About the age of 10 she began to show oedema of feet and ankles but this went away, to recur again at 18 when she had an attack of bronchitis. At this time her right calf suddenly became swollen and red but not very painful, and it has remained swollen. Married at 24 and in one and a half years had a daughter (III. 1), no other pregnancies. During her pregnancy she became very fat and signs of oedema began to show themselves in left leg and this gradually increased. Otherwise she is well. II. 4, healthy, II. 6, ♂, aged 28, thin and has a cough. II. 7, ♀, aged 21, at age of 13 feet began to swell at night. At the age of 19 an attack of scarlet fever with swollen feet and legs. This has continued. II. 8, four children died in childhood. II. 9, three alive and well, sex not stated. III. 1, died at age of one and a half from pneumonia following measles. III. 2, aged five, has convulsions, sex not stated. III. 3, aged three, developed normally up to 14 months when without apparent cause development was arrested, oedema of both feet. III. 4, normal child. (See Bibl. No. 12.)

Fig. 74. *Jopson's Case.* Congenital elephantiasis in two brothers. II. 1, suffered from similar condition in youth but it disappeared as he grew up. II. 2, presumably normal. III. 1, two healthy children, alive, sex not stated. III. 2, two healthy children, dead, sex not stated. III. 3, 4, miscarriages. III. 5, ♂, aged four, marked swelling of both lower limbs involving feet and legs up almost to knees. The swelling is hard and pits on prolonged pressure. Over the dorsum of the feet the oedema is very marked and the skin white. At birth the prepuce was also involved and had to be circumcised. Bones not enlarged. III. 6, ♂, one and a half years old, condition also congenital but less marked than in III. 4, in so far that the swelling only extends up to the ankles. Prepuce not affected. III. 4 and III. 5 are otherwise healthy and intelligent. (See Bibl. No. 13.)

PLATE XIV. Fig. 75. *Sutherland's Case.* V. 10 is a child of nineteen months. In December, 1908, the child was shown at the Royal Society of Medicine. She was accompanied by her sister (V. 7), who was there

found to be affected with trophoedema. The pedigree of the family was then worked out by Mr P. Fildes from information supplied by IV. 5 and III. 17, the latter aged 76 and living in the same house as the affected children. As both his son-in-law (IV. 5) and he gave the same facts at different times, there is reason to believe that the pedigree is correct. No history of trophoedema could be found in the ascendants. I. 1 died aged 84, in 1837. I. 2, no information. II. 1 and 2, no information. II. 3 died aged 97. II. 5 and 6, no information. II. 8 died aged 65. II. 9, no information. III. 1—8 stated by IV. 5 to have been unaffected—all dead. III. 4 had "paralysis" from birth. II. 7 represents 1 or 2, which IV. 5 could not account for. III. 8 died from heart disease. III. 9, 10, 11 unaffected with oedema. III. 11 suffered from some form of paralysis. III. 12—15, no information. III. 16 died of cancer (1874). III. 17 alive, aged 76, in excellent preservation mentally and bodily; does not suffer from oedema. III. 18, not affected. III. 19 was, according to III. 17, a "bad lot." IV. 1 died young. IV. 2 died from heart disease. IV. 3 died of consumption. IV. 4 died young. IV. 5 is not oedematous, but suffers from a form of paralysis which is stated to have been a sequel to measles. Considerable wasting below knee, foot drop and some *talipes Equino-varus*. IV. 6 died aged 41. According to IV. 5 and III. 17 she was not affected with swollen feet. IV. 7, not affected. IV. 9, a soldier. IV. 10, 11, 12, dead. IV. 14, alive; not affected. IV. 16, no information. IV. 18 and 20 stated to be unaffected. V. 1, aged 18. V. 2, a few days old. V. 3—6, normal. V. 7, aged seven, has trophoedema of left lower extremity extending from below the knee to the tips of the toes; began at birth. No other symptoms. V. 8 and 9, normal. V. 10, one year and seven months; affected with oedema in both feet since birth. Dorsum of foot, sole and toes affected. Oedema ends abruptly at ankle, and is of a pinkish red colour. Child has suffered from lichen urticatus of severe degree on various occasions. Heart normal. The child's foster-mother states that when the rash improves the swelling of feet becomes greater, and *vice versa*. The oedema is persistent. V. 11—19, all normal. V. 20, 21, twins; sex not known to IV. 6. V. 22—24, healthy. (See Bibl. No. 20, supplemented by information gathered by P. Fildes.)

Fig. 76. *Moyer's Case.* II. 1, a young lady of 32 years of age, of good history; never had any sickness since the usual children's diseases. The enlargement began when II. 1 was aged 20, just above the shoe top. Never had been any pain or discomfort. It occurred after a sprain, but she did not know that the sprain caused it. The same thing began in her mother four years afterwards, and was like the daughter's case except that it did not extend above the knee. The patient had been active all the time, attending to her duties as school teacher, and able to walk long distances. There was a difference in the calf measurements of five inches. The skin seemed perfectly normal. It was probably not an oedema, but a trophic disturbance. There was no pitting of the skin and no fluid. There was a uniform enlargement of the whole lower extremity from the crest of the ilium to the foot. There seemed to be an increase of body of the entire muscular system. It was not, in a certain sense, a disease. All that was complained of was muscular hindrance. The leg was big and hence uncomfortable and in the way. There was no central disturbance. The thyroid was present, but it was impossible to tell its condition. No further information as to other relatives, etc., has been received in answer to our letter of enquiry sent to Dr Moyer. (See Bibl. No. 19.)

SECTION IX α. ANGIONEUROTIC OEDEMA. (ACUTE CIRCUMSCRIBED OEDEMA, QUINCKE'S DISEASE.)

By William Bulloch, M.D.

The attention of the medical profession was first prominently directed to this condition by the publication of Professor Quincke[1] of Kiel in 1882. In his short paper he described the occurrence of circumscribed pale oedematous swellings of the limbs, trunk and face, and he emphasised the fact that the mucous membranes of the lips, palate, pharynx and intestine might also be involved. The swellings come and go quickly and often show a remarkable periodicity. More than one member of a family may be affected. The views of Quincke were further extended in Dinkelacker's[2] thesis, carried out under Quincke's direction, and since then a large number of cases have been recorded from different parts of the world, so that the condition is now well known. Of the many degrees in which this malady occurs the most severe are those with the periodic recurrence of acute circumscribed hard swellings lasting

usually a short time and disappearing entirely. Added to this, abdominal symptoms comprising anorexia, nausea, feeling of tightness, very severe colic and vomiting are almost constantly observed. Some individuals however are affected with the swellings only, others with the abdominal symptoms only. There exist, indeed, all grades of the affection even in one and the same family. The condition may appear at any time of life from infancy onwards and both sexes are affected. In some instances it is troublesome rather than dangerous, but a large number of cases have now been published where sudden death has occurred from oedema of the glottis and larynx. In the few instances which have come to autopsy the oedema has been found to involve not only the mucous membrane but also the deeper structures (Wardrop Griffith[32]). The abdominal crises have been referred to oedematous swellings in the mucous membrane of the stomach and bowel and in at least one case (Harrington[13]) this was directly proved by operation, a cylindrical enlargement of the ileum $2\frac{1}{2}$ inches in length being brought to light through an exploratory abdominal wound. By all authorities the condition is looked upon as a neurosis of vasomotor origin and akin to other angioneuroses such as acroparaesthesia, Raynaud's disease, and erythromelalgia. The causes are unknown, but in a number of instances direct inheritance has been

Observer	No. of Gen.	Cases				Deaths of affected				Deaths from oedema of glottis			
		♂	♀	○?	T.	♂	♀	○?	T.	♂	♀	○?	T.
Ensor	7	28	21		49	16	6		22	8	4		12
Osler	5	9	11	2	22	2	2		4	1	1		2
Schlesinger	4	4	1		5	1	—	—	1	—	—		—
Yarian	4	5+	3		8+	2	1		3	1	—		1
Mendel	4	6	3		9	4	2		6	4	2		6
Ricochon	3	3	4		7	1	—		1	—	—		—
Fritz	3	3	3	2	8	3	1	2	6	2	1	2	5
Prior	3	3	1+1?		4+1?	2	—	—	2	2	—	—	2
Halsted (Case i) ...	3	2+1?	2+		4+1?	—	—	—	—	—	—	—	—
Bramwell	3	4	3		7	1	1		2	1	1		2
Wardrop Griffith ...	3	2	1		3	1	1		2	1	1		2
Quincke-Dinkelacker-Valentin	2	3			3	—	—		—	—	—		—
Quincke-Dinkelacker	2	1	2		3	—	—		—	—	—		—
Falcone	2	2			2	—	—		—	—	—		—
Morris	2	1	2	1?	3+1?	1		1?	1+1?	1	—	—	1
Strübing	2	2	1		3	—	—		—	—	—		—
Smith	2	1	1		2	—	—		—	—			—
Joseph	2	1		1	2	—	—						—
Sträussler	2	3			3	3			3	3			3
Wagner	2	2			2	—			—	—			—
Apert and Delille ...	2	5			5	—			—	—			—
Courtade	2	5			5	—			—	—			—
Mosler	2	3	1		4	—			—	—			—
Cuntz	2	1	1?		1+1?	—			—	—			—
Roy	2		2		2	—			—	—			—
Ross	2	1	1		2	—			—	—			—
Halsted (Case iii) ...	2	1+1?			1+1?	1?			1?	1?			1?
Halsted (Case ii) ...	1	1+1?	1?		1+2?	—			—	—			—
		102+3?	63+3?	5+1?	170+6?	37+1?	14	2+1?	53+2?	24+1?	10	2	36+1?

Taking the undoubted cases at 170 and the deaths from oedema of the glottis at 36 the percentage of deaths from oedema to the total number of cases is 21·1 %, but taking the total number of deaths from all causes of those afflicted by the disease 67·9 % died of oedema of the glottis.

determined, or the disease has occurred as a family complaint, the principal cases showing this being those of Ensor[14], Osler[15], Schlesinger[16], Yarian[17], Mendel[18], Ricochon[19], Fritz[20], Prior[21], Halsted[22], Byrom Bramwell[23], Quincke-Dinkelacker[2], Valentin[24], Falcone[25], Strübing[26], Smith[27], Joseph[28], Sträussler[29], Wagner[30], Apert and Delille[31], Wardrop Griffith[32], Courtade[33], Mosler[34], Cuntz[35], Roy[36], Morris[37], Ross[38]. The preceding table shows the number, sex, and generations known to be affected. As in many cases the oedema has not manifested itself till the age of 20 or even later, attention must be paid in the later generations to the probable age of the individual when considering whether they may fairly be classed as normal. We should probably have been justified in hatching many of these apparent "normals" in the last generation of the pedigrees as "unknown."

Our pedigrees and the accounts of them have all been taken from the original sources.

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HEREDITARY CASES OF ANGIONEUROTIC OEDEMA.

PLATE XIV. Fig. 77. *Wardrop Griffith's Case.* Three cases of angioneurotic oedema occurring in three generations. I. 1, died at age of 72. I. 2, died at 70—a very intelligent woman. I. 3 and I. 4, nothing stated. II. 1 and II. 3, not affected. II. 3, unmarried. II. 4, according to I. 2, was well until five, when he had an attack of scarlet fever; after this had repeated swellings. I. 2, frequently referred to II. 4's ailment and remarked she had never heard of anything of the kind in the family; from information supplied by II. 6 (wife of II. 4) he was known to have repeatedly suffered from swellings from earliest infancy; on three occasions between 21 and 29 years these seem to have affected the throat, the last attack carrying him off; he felt his throat swelling and a medical man was sent for, but before he arrived II. 4 got blue in the face and died asphyxiated. II. 5, not affected, unmarried. II. 6, not affected. II. 8, 10, 12, normal. III. 1 and III. 2, unaffected. III. 4, unaffected. III. 5, man[1] by whom III. 6 had an illegitimate daughter, IV. 7. III. 6, female, affected, repeatedly seen by Professor Griffith during 16 years. II. 6, her mother alleged that she had been subject to swellings on various parts of the body since infancy; shortly after birth one arm swelled so much that the clothing had to be cut; swellings frequent but not periodic on hands, feet, shoulders, chest, vulva; when about 20 she struck her head on the lock of a door, causing a small lump on her forehead; next day the swelling had affected the eyelids and face and then her throat, but it

[1] Erroneously marked "1st H." by a slip of engraver.

passed off quickly; about 16 months later had a severe attack in the face with great swelling of lips, tongue and glottis; married at age of 25, and had a girl and two boys; swellings uninfluenced by pregnancy; on Feb. 20th, 1902, at 10.30 a.m. she said her throat was swollen; she was found by her husband at noon, sitting on the floor playing with one of her children, and apparently more comfortable; she suddenly started up, clutched at her neck, got blue in the face, and died almost at once; autopsy: mucous membrane of larynx very oedematous, tense and pale, and the sides of the laryngeal cavity came in contact with each other a little below its superior aperture and remained so until just below the level of the true cords; transverse sections showed that the oedema involved not only the mucous membrane but the deeper connective tissue and even the substance of the muscles; the oedematous fluid was of the serous variety. III. 8 and 9, unaffected, likewise III. 10—30, cousins of III. 6. IV. 1—6, normal. IV 7, female illegitimate child of III. 6, died suddenly at age of 15 months: she had been playing at the knees of her grandmother, II. 6 (Dr Griffith's informant), who struck her lightly on the hand; she flew into a passion, went blue and died. II. 6, did not consider that she suffered from oedema, as there had been no swellings; a doctor said she died of rupture of the heart (!); no autopsy. IV. 8, unaffected. IV. 9, male, now 12 years old (1909); has frequent swellings of face. IV. 10, male, never had swellings; died Jan. 1909, with what was apparently diphtheria or some infection of throat; his grandmother, II. 6, was present when he died, but considered that his death was not due to suffocation from swelling of throat. (Bibl. No. 32. Our pedigree is constructed from additional data kindly supplied by Professor Wardrop Griffith in February, 1909.)

Fig. 78. *Falcone's Case.* Two cases of Quincke's oedema occurring in a man and his grandson. I. 1, aged 55, was first affected when about 24; after exposure he got oedema of eyelids and a considerable swelling near R. knee joint; the attack lasted 24 hours; from this time, however, he was frequently affected with swellings lasting about a day. II. 1, died from lung trouble, did not suffer from oedema. II. 2, presumably unaffected. III. 1, aged seven; had diphtheria at two, but was otherwise well and strong; at seven was attacked with oedema of R. cheek, diarrhoea and headache, anorexia and a great desire to sleep; vomited five or six times; this was followed in two days by swelling of R. upper arm and hand; no fever, urine of low specific gravity and of pale colour (no mention of albumen test). (See Bibl. No. 25, p. 125.)

Fig. 79. *Osler's Case.* A series of 22 cases occurring in five generations. The cases conform to the typical picture of Quincke's oedema and in two or three instances death took place from oedema of the glottis. Gastro-intestinal crises were very marked in this family. Osler's patient was IV. 12, but he obtained most of his records from II. 5, a hale and vigorous old man of 92, whose mental faculties were perfect; according to him the condition probably started with I. 2, his mother (1762—1834); he thinks that if it had been in her father's or mother's family she would have known the fact and mentioned it; she suffered with oedematous attacks from youth onwards, and II. 5 had frequently seen her when affected; in one crisis she was nearly suffocated; she had gastric crises; after the age of 45 she was not so much troubled but her constitution had been weakened by drugs; she was twice married, having two sons by the first husband and three sons by the second; of these four grew up. II. 2, not affected, but his children suffer, and one (♂) died of the disease. II. 3, not affected. II. 5, aged 92, in good health; he was healthy as a child, but began to suffer at age of 18 or 19 and attacks have recurred at intervals of 4—6 weeks; in recent years he has suffered less frequently but had an attack at 92; his oedema has been chiefly in his hands and privates but never in the face; gastric crises very marked, followed by vomiting; he was twice married, and has had 14 children (in Osler's pedigree it gives 10), of whom only one ♂ by first marriage and two ♀ by second are affected. II. 7, suffered frequently from youth onwards; has four children living, of whom one is affected. II. 9, died young. III. 1, died of oedema; no details. III. 2 and 3, suffered from oedema, sex not stated. III. 5, began to "swell" at age of 20 and had very many bad attacks; died of Bright's disease at age of 60; of his nine children all suffered except IV. 7; information supplied by III. 4, his wife. III. 7, began to be affected at 10 or 12 years, with swellings of feet, hands, face; attacks recur every few weeks, unmarried; she cannot eat apples and certain vegetables. III. 8, had very severe attacks especially of colic; married, no children. III. 10, three children, sex not stated; unaffected. III. 11, ♂ (? = "Angey"), affected, no data. IV. 1, ♀, affected, no data. IV. 4, always suffered with attacks of cramps in the stomach and of late has been attacked with swellings. IV. 5, began to swell at four or five years; attacks much more frequent after marriage; had three children, all premature as a result of her malady; she died in an attack at 5 a.m., evidently oedema of larynx. IV. 7, never affected. IV. 8, always had cramps, but first "swelled" in 1888; age not stated. IV. 9, subject to swellings since childhood. IV. 10, affected, but not so frequently as the others. IV. 11, within the last few years has had bad spells of cramp and swellings. IV. 12, age 24, seen by Dr Osler; medium sized, brunette; married, two years; no children; has had transient swellings on hands, fingers, knee caps, elbows, buttocks, thighs, face, lips; often affected with red spots or red lines without oedema; gastric crises marked; periodicity of attacks about every two weeks. IV. 14, has always had bad spells of cramps but swelled last summer (age not stated) for first time. V. 2, badly affected, married (one year before Osler's publication). V. 3, has bad attacks. V. 4, born at seventh month, dead. V. 5, born at

seventh month, now 17 years, has had one attack. V. 6, born at eighth month, aged 11, unaffected so far. (See Bibl. No. 15, p. 362.)

Fig. 80. *Yarian's Case.* Angioneurotic oedema in four out of six generations. I. 1, suffered from oedema but had no gastric crises. II. 2, and some of his brothers, II. 3, had oedema, but no gastric crises. III. 2, had oedema from childhood but not during gestation; died of dropsy (cause?) at 63. III. 5, had oedema and died of closure of glottis, aged 42. IV. 2, ♀, Yarian's patient, aged 54 and seen by him in severe gastric attacks; she has suffered as long as she can remember; swellings of hands, face, arms, shoulders; from 24 hours to two days after the swelling begins she is seized with severe pain in the bowels or stomach; these attacks being succeeded by nausea and vomiting; periodicity once every two weeks since her child-bearing period, but before that every week for several weeks; longest period of freedom from attacks was nine weeks. IV. 6, ♀, affected with swelling, but no gastric crises. IV. 7, ♂, suffered from oedema; died of scarlatina at age of seven. V. 1, died of tuberculosis, aged 21; was not affected with oedema but of irritable disposition. V. 2, aged 25, unaffected; her daughter, VI. 1, unaffected. (See Bibl. No. 17, p. 238.)

Fig. 81. *Prior's Case.* A case, III. 1, of angioneurotic oedema, fatal from swelling of glottis; information with regard to affection in other members supplied by II. 2. I. 1, was subject to large white swellings after trivial injuries or without any apparent cause; he died suddenly from swelling of throat. II. 2, ♀, on one occasion had her tongue greatly swollen without any apparent cause; in the pedigree a query is put against her as she is a doubtful case of Quincke's oedema. II. 3, sister of II. 2, stated to have been subject to oedema, no details. III. 1, aged 38, an imbecile with constant tremor of hands, and exaggerated knee jerks; suffered from typical attacks of acute circumscribed or diffused oedema on trunk, arms, feet, genitals; no periodicity in attacks; on June 5th, 1905, lower lip found to be swollen at 3 a.m., with rapid extension to face and neck by 7.30 and death from syncope at 10 o'clock; autopsy showed great swelling of face, lips, neck, tongue, epiglottis and mucous membrane of larynx. III. 2, an epileptic. III. 4, stated to have suffered from oedema—no details. (See Bibl. No. 21, p. 116.)

Fig. 82. *Apert and Delille's Case.* Typical acute circumscribed oedema in all, five, of the male members of a family in two generations. The condition showed marked "anticipation" in each generation. In II. 1, 3, 7 the affection first manifested itself in adult life, whereas in III. 1, 2 it developed in infancy. A further "anticipation" was also witnessed according to the order of birth of the siblings. In the second generation II. 1 was first attacked at the age of 30, II. 3 at 28, and II. 7 at 20, all being affected for the first time in 1894. In the third generation III. 1 was attacked for the first time at seven and III. 2 at five years of age, both attacks occurring in 1900. I. 1 and 2, stated in Apert and Delille's pedigree to have been normal—not mentioned in text. II. 1, aged 40; for the last ten years had been affected every 8—15 days with partial oedema of face, limbs, genital organs, pharynx or larynx; oedema of fugitive character and lasting for about a day; during the oedematous "crises" the most trivial traumata suffice to determine the oedema to the seat of injury; in one of the laryngeal attacks three years ago tracheotomy was necessary; after the attack subsided the canula was taken out, but a fresh operation was required three months later during another crisis, since which time he has permanently retained the canula; it is stated that he has no children. II. 2, nervous and choleric subject; died at age of 26. II. 3, brother of II. 1, aged 38, a wood turner, strong and sober; always well up to the age of 28, at which time he began to suffer attacks of oedema; every one or two months or even at shorter intervals; the oedema usually affects one or both hands; when the attack is at an end general malaise, nausea and vomiting supervene and last for several hours; has been twice married. II. 4, healthy and strong. II. 5 and 6, stated to be normal. II. 7, aged 30; affected from the age of 20 onwards; oedematous attacks of feet and hands every two or three months and lasting for about a day. III. 1, aged 11; from the age of seven has had oedema of upper limbs every two or three months and lasting several hours; as in the case of his father the attacks are accompanied by gastric pains and nausea. III. 2, aged nine; began at the age of five to suffer from attacks of acute oedema, at first rarely then frequently; oedema circumscribed and affecting upper limbs, cheeks, and more rarely the lower limbs. Four months ago the oedematous area of face spread to the tongue and uvula for the first time; since then, however, there has been an almost exact alternation (every 15 days) of the oedema of limbs and face; as in the case of the father and brother, pains in the stomach and nausea accompany the attacks of oedema; in one of these he was brought to the Hôpital Bretonneau in an asphyxiated condition with oedema of soft palate, uvula and glottis; he was accompanied by II. 1, whose tracheotomy canula led to enquiries and the establishment of the diagnosis of Quincke's oedema. III. 3, 4, 5, 6, 7, stated in Apert and Delille's pedigree to be normal. (See Bibl. No. 31, p. 1022.)

Fig. 83. *Joseph's Case.* Incomplete case. IV. 1, aged five years, seen by Joseph in 1888; at 2½ his mother noticed swellings on his hands resembling bug bites; swellings also appeared on his cheeks, forehead and behind ears; these occurred almost exclusively during winter and were in some way connected with exposure to cold as no swellings appeared in parts of the body covered by clothes; at age

of four he also had five attacks of haemoglobinuria; no syphilis or skin or nervous trouble in the ascendants; a cousin, III. 3, of the mother, III. 2, was however also affected with swellings in the face; no specific mention is made of any other members of the family. (See Bibl. No. 28, p. 76.)

Fig. 84. *Mendel's Case.* Mendel reported a typical case in a ♀, aged 18, IV. 1. Her mother volunteered the information that it was a family disease and known to be so in the village where they lived; it was called by the surname of the patients, even by the doctors; most of the information obtained from III. 6. I. 1, said to have had the family malady and died of suffocation (oedema glottidis?). II. 1, suffered all his life and died of suffocation (at 40), the apparent cause being the injury following tooth extraction. II. 3, affected; had swelling of face one morning; she became cyanosed and suddenly died—aged 66. III. 1, died of oedema at the age of 22. III. 2, seen by Mendel when 51 years old; he had been afflicted since youth, the attacks coming on every eight days; in youth they were separated by long intervals, and between 1876—1892 he was free altogether; both he and his brother, III. 3, who was also afflicted, were melancholic and anxious, especially over the risk of swelling in the throat. III. 2 also had abdominal symptoms; when seen by Mendel he was not affected, but showed a movement of the head resembling paralysis agitans. III. 4, not affected. III. 5, ♂, affected, died of suffocation at the age of 33. III. 7, ♀, died of suffocation at 22. IV. 1, aged 18, had suffered from earliest youth with colossal swellings on different parts of the body and recurring at varied intervals; mouth and eyes affected; the swellings came on with or without previous trauma. IV. 2, normal. IV. 3, unaffected with oedema but suffers from rheumatism, morbus cordis and chorea. (See Bibl. No. 18, p. 1126.)

Fig. 85. *Quincke-Dinkelacker's Case.* I. 1 and I. 2, nothing stated. II. 2, ♀, suffered in youth from urticaria, especially on arms. II. 3, her sister, not stated whether affected. III. 1, ♂, oedema in uncovered parts, eyelids, lips, hands; he suffered from earliest period he can remember, but it increased up to 20th year, when eruption occurred almost daily. III. 3, his cousin, gets swellings of lips, especially at night. (See Bibl. No. 2, S. 20.)

Fig. 86. *Schlesinger's Case.* Five cases of acute oedema occurring in four generations. Attacks began about the 20th year, and were ushered in by psychical exaltation or depression followed by a peculiar erythema. The oedematous swellings occurred with great acuteness and gastric crises were present. The condition persisted through life. I. 1, affected from 20th year onwards, although in old age he was less frequently attacked; died at over 80. II. 1—4, stated in Schlesinger's pedigree to be normal —not referred to in text. II. 5, affected from 20th year, but not so frequently in old age; prodromal exanthem well marked. III. 2, aged 44; first attacked at 22; attacks recurred at first every six months, later every 10 or 11 days; commenced with excitement or depression, which was followed by prodromal exanthem of erythematous character on some part of the body, rarely over a large surface; eruption disappeared in 6—8 hours and was rapidly (seconds!) followed by marked oedema, mostly of extremities, penis or scrotum; pain in stomach and vomiting frequent; III. 2 was repeatedly seen by Schlesinger in these attacks. III. 6, aged 37; affected since 20; prodromal rash present, succeeded by oedema; gastric crises intermit with oedema. IV. 3, aged 16; for several months has been affected with the family eruption on the breast, arms, nose, but so far has not suffered from oedema; other members not referred to in text but mentioned in pedigree as normal. (See Bibl. No. 16, S. 334.)

PLATE XV. Fig. 87. *Halsted's Case* (i). Family with one individual, III. 2, affected with typical angioneurotic oedema. Of the other members it is said: "On mother's side grandmother (I. 2) and her sisters (I. 3) had severe *urticaria.* One uncle (II. 3) had an attack of it after eating canned fruit. This uncle's son (III. 3) had the disease so badly that he 'would go into spasms.' Patient's mother (II. 2) had bad urticaria five or six years ago." III. 2, ♂, aged 24, single, blonde; very neurotic, sudden swelling of throat at 24 years of age, then swelling of arm, feet, shoulder; penis and scrotum occasionally the seat of enormous swellings; previously he had suffered for about 12 years; on more than one occasion abdominal crises; he was a director of gymnasium, and afterwards a doctor, and studied his case carefully but was unable to attribute any exciting cause for the attacks. It is stated that the mother's family for years regarded urticaria as a family disease; they are also distinguished among themselves by a peculiar gastric affection which they call the "S— stomach." In all probability a genuine case of angioneurotic oedema. W. B. (See Bibl. No. 22, p. 863.)

Fig. 88. *Halsted's Case* (ii). Case of a male, aged 55, II. 3, with swellings of throat, uvula, soft palate; swellings in eyelids, scrotum, penis, feet, hands; has suffered for 18 years; has two children. III. 1, a sufferer from asthma, but has not had oedema. III. 2 has had throat symptoms, but II. 3 says they are not the same as his. II. 1, brother of II. 2, suffered greatly from "gall stone colic"; two years ago he fell, injuring one of his knees; one month later, while apparently in good health, after being at stool he suddenly fell and died almost immediately; a searching medico-legal *post mortem* was made, but no cause of death could be determined; there was no evidence of gall stones; Halsted inclined to regard the case as oedema arising in brain; II. 1, marked with a ? in chart. (See Bibl. No. 22, p. 863.)

Fig. 89. *Halsted's Case* (iii). III. 1, ♂, aged 35; of highly nervous temperament; had sudden swellings all over body off and on for five years; also throat attacks. I. 1, suffered repeatedly from violent colic—died of uraemia. II. 2, had urticaria, twice in two years, no details. (See Bibl. No. 22, p. 863.)

Fig. 90. *Ricochon's Case.* Typical case of Quincke's oedema occurring in seven members of a family in three generations. Details of individuals affected very incomplete. The symptoms in all were similar, viz. sudden oedema of some part of the body, such as feet, knees, hands, thighs, genitals, neck, face. Duration of oedema 24—36 hours. All suffered from abdominal crises consisting of severe colic, tympanitis, and vomiting. Great thirst and tendency to sleep was also marked. Only the affected individuals are mentioned and of them it is stated that the grandfather died of enteric fever. A son, two daughters, two granddaughters and a grandson are alive. The latter (grandchildren?) were attacked at one or two years and the others at 12, 13, and 15. (See Bibl. No. 19, p. 670.)

Fig. 91. *A. J. Smith's Case.* Two cases of angioneurotic oedema in two generations. I. 1, neurotic subject; affected with oedema in early middle life. II. 1, first attacked between 16 and 18 years; attacks recurred several times a year, without cause, until she was 22; swelling chiefly in the face or on one or both arms and lasting a few hours to a day; married four years and has had three children, III. 1, 2, 3, the first two strong, the third more or less sickly; III. 4 a miscarriage; pregnant with III. 5 when seen by Smith; sex of children not stated. (See Bibl. No. 27, p. 320.)

Fig. 92. *Strübing's Case.* Three cases occurring in two generations. I. 1, aged 70, teacher in a high school; was healthy up to the age of 25; one evening, after a chill, his throat began to swell, and within half an hour his condition was serious but the swelling went down during the night, but was succeeded by swelling of lower lip, cheeks and eyelids; no spontaneous recurrence of swellings for a long time; in later years, however, swelling of throat returned again and was associated with profound dyspnoea; penis and scrotum also affected; even at age of 70 swelling occurs in the limbs; since the age of 26 he has also been the subject of periodic attacks of vomiting and colic every 4—6 weeks. II. 1, ♂, aged 16, affected with oedema and gastro-intestinal disturbance in severe form from the age of three. II. 2, ♀, affected, no details. II. 3, two unaffected, sex not stated. (See Bibl. No. 26, S. 381.)

Fig. 93. *Courtade's Case.* I. 1, had his first attack of acute oedema affecting the scrotum, at the age of 57. I. 2, his wife, presumably normal. II. 1, aged 55, a wood turner by occupation; at age of 12 had convulsions; at 15 had acute rheumatism which lasted two weeks; after this began to be troubled by oedema in acute attacks, lasting about 24 hours; oedema locates itself at the site of the slightest traumata but its periodicity is not marked; rare to begin with, it occurred more frequently as he got older, ultimately coming on about twelve times a year; the oedema is of a wandering character and he has twice had to submit to tracheotomy for oedema of the glottis; abdominal pains and vomiting are frequent in association with the attacks of oedema; Courtade says that the condition existed in all the family, but of his four brothers he refers to only three, viz. II. 2, aged 33, II. 3, aged 32, and II. 4, aged 30, who suffered in the same way. II. 5 not being mentioned, we have marked him unaffected in the pedigree. No other details are given of the family. (See Bibl. No. 33, p. 1340.)

Fig. 94. *Cuntz's Case.* Case of oedema in a ♂ (II. 1), aged 21; his mother, I. 2, suffered from urticaria of unknown origin; this case is reported in Dinkelacker's thesis as a case of acute oedema. II. 1, in his 18th year was suddenly attacked under the eyelids and on dorsum of right hand with swelling and collection of fluid; it disappeared in a few hours; a similar bulla appeared on the prepuce within an hour; swellings recurred in three or four days but disappeared, and he remained free for three years; in his 21st year large swelling appeared without cause in his left palm. (See Bibl. No. 35, S. 63.)

Fig. 95. *Quincke's Case.* In his original communication describing acute circumscribed oedema, Quincke refers to a case in which an individual who was attacked relatively regularly had a son who also showed the characteristic swellings from his first year. No details of either individual are given. (See Bibl. No. 1, S. 129.)

Fig. 96. *Roy's Case.* Case of angioneurotic oedema in mother and daughter. I. 2, aged 45, suddenly attacked with oedema of one foot, accompanied by gastric pain and nausea; on following day the other foot became swollen, then hands and face; she had had previous attacks of similar character but slighter. II. 1, seen by Roy in similar attack, except that swelling was confined to face and hands and was accompanied by nettle-rash; the abdominal crisis was more marked than in I. 2; had suffered on previous occasions. No mention of any other members of family. (See Bibl. No. 36, p. 42.)

Fig. 97. *Sträussler's Case.* Severe fatal case of Quincke's disease occurring in a soldier, II. 2; father and a brother probably succumbed to same disease. I. 1, aged 46, and II. 1, a youth, age (?), both seized with symptoms of suffocation and died suddenly. II. 2, a soldier, aged 23; awoke his comrades in the night between 18th and 19th May, 1903, with his cries; he was profoundly dyspnoeic and rapidly became unconscious and died of suffocation; artificial respiration was attempted as a restorative, but without avail; tracheotomy was also carried out but was also ineffective; during the operation the skin, subcutaneous tissue and the mucous membrane of larynx found to be very oedematous and infiltrated with fluid; the mother, I. 2, alleged that he had had swellings before he entered the army, and that the father and a brother of patient had succumbed suddenly with asphyxia. (See Bibl. No. 29, S. 597.)

Fig. 98. *Mosler's Case.* Four cases occurring in two generations. II. 1, medical student, aged 22, strong and healthy; in youth suffered from scrofula and urticaria, but remained free from the latter for a number of years, when it recurred with a periodicity of 8—14 days; swelling mostly of extensor aspect of forearm and metacarpus of thumb and little finger, but at times also of breast; swelling lasts for half to one hour; he could attribute no cause for the affection till his mother, I. 2, told him she also had suffered for years from the same malady. II. 1 also said his younger brothers, II. 2 and II. 3, were affected but two sisters were free. (See Bibl. 34, S. 605.)

Fig. 99. *Quincke-Dinkelacker-Valentin's Case.* This case is stated by Dinkelacker to be the one referred to briefly by Quincke, "Krankheiten der Gefässe," in v. Ziemssen's *Handbuch d. spec. Pathologie und Therapie*, Leipzig, 1879, 2^te^ Auflage (Dinkelacker says VI. Aufl. evidently a misprint), Bd. VI. p. 325. I. 1 and II. 2 were described fully by Dinkelacker himself (S. 6), and II. 3, born after the publication of his (Dinkelacker) paper, was described by Valentin. (See Bibl. No. 24, S. 150.) It is also probable that I. 1 and II. 1 are the father and son referred to in Quincke. (See Bibl. 1, S. 129.) Our account of I. 1 and II. 2 is taken from Dinkelacker, and II. 3 from Valentin. I. 1, seen by Quincke and Jeanneret in La Chaux-de-Fonds, in 1873, when he was 22 years old—the head of a big watchmaking business; he came of a family in which nervous diseases, migraine and similar maladies were unknown; had scarlet fever at age of nine months, and this was soon succeeded by acute circumscribed oedema, recurring every week, usually on Friday, Saturday or Sunday; at first he was affected with severe colic, vomiting, thirst and constipation, this lasting from his first to seventh year; from then till he was 14 he was free of all troubles, but after this had in addition to his gastric crises circumscribed oedema, especially in region of joints (hands, elbows, fingers, knees, ankles, toes) and occasionally in face; for some years he has also been subject to great swelling of laryngeal mucous membrane, producing profound dyspnoea and cyanosis and necessitating scarification; attacks recurred with great regularity every eight days; he was seen in these by Quincke in 1873, 1878, and by Valentin at a later period. II. 1, referred to by Dinkelacker and said to be unaffected. II. 2. (In Dinkelacker's thesis.) ♂, one year old, healthy and lively but affected with his father's malady, since he was three months old; swellings on extremity and face (once in neck), preceded by anorexia, vomiting and thirst; seen by Quincke and Valentin. II. 3, Valentin's patient—no details as to age but stated to have been affected from the first week after he was born; Valentin saw him in attacks. II. 4, ♀, a baby, unaffected (in Valentin). (See Bibl. Nos. 1, 2, 6 and 24.)

Fig. 100. *Bramwell's Case.* Angioneurotic oedema occurring in four ♂ and three ♀ in three generations. I. 2, ♀, aged 83, alive, twice married; attacks of swelling commenced when a girl at school; up to age of 18 face only affected; since then the throat has sometimes been affected, also limbs, feet; abdominal crises with vomiting. II. 2, ♂; swellings began in infancy in face and throat and especially scrotum; limbs escaped; never had gastric crises; died of swelling of throat, aged 21. II. 6, ♀; swelling began in infancy, face, throat and limbs being affected; abdominal crises present; died from swelling of throat at age of 47. II. 10, ♂, aged 41; a coal trimmer; began to be affected at the age of ten, the swellings appearing in hands, scrotum, feet and legs; eyes, face, throat and mouth not affected; abdominal crises present; swellings occur at irregular intervals, usually every three or four weeks, but sometimes oftener; they occur without apparent cause. III. 10, ♂, aged 14; disease manifested itself between two and three years with swellings of limbs and abdominal crises. III. 23, ♂, aged 17; disease began at seven years with swelling of hands and limbs, and abdominal crises, throat not affected, III. 26, ♀, aged eight; began at six with abdominal crises only; so far there have been no swellings. (See Bibl. No. 23, p. 374.)

Fig. 101. *Ross's Case.* I. 1, 2, 3, 4, healthy. II. 1, healthy. I. 2, aged 46, had occasional local oedematous swellings of hands, feet, face, eyelids; no periodicity; some gastro-intestinal disturbance but no vomiting or colic. III. 1, healthy. II. 2, aged six, had several attacks of acute relapsing oedema of eyelids. (See Bibl. No. 38, p. 512.)

Fig. 102. *Fritz's Case.* Eight cases of angioneurotic oedema in three generations. I have not personally seen Fritz's paper. The account given is from data kindly extracted by Prof. John Mackenzie of Toronto. He adds that no further trace of the family could be got and that the author is dead. I. 2, died of oedema of glottis. I. 3 and 4, not mentioned. II. 4, was insane six months before death, which

was due to pulmonary abscess. II. 5, alive and well. II. 1, an aunt of III. 4,—whether paternal or maternal is not stated—she suffered from oedema, which appeared and disappeared at various times. III. 4, Dr Fritz's patient, Mr P., aged 23, single, a printer by occupation, born in United States; admitted into Buffalo general hospital, May 18, 1893; the day previously he fell and hurt his right temple; eyelid began to swell and eye was completely closed up, the swelling extending to cheek, lip, and cellular tissue of neck; when admitted he had dyspnoea and almost complete aphonia; uvula very swollen, likewise left tonsil, L. pillar of fauces and left side of glottis; symptoms rapidly subsided; he had always enjoyed good health, but had observed that drinking on an empty stomach, over eating or slight injuries would bring on attacks of swelling on extremities and eyelids; he had been affected in this way since he was four years old; the day after admission swelling of face and neck subsided, but swelling appeared on R. foot and ankle. III. 5, comparatively healthy—no other information. III. 6, troubled with oedema of glottis—no details of attacks. III. 1 and 2, both died of oedema glottidis. III. 7, 8, two sons died of oedema glottidis. (See Bibl. No. 20, p. 286.)

Fig. 102 bis. *Morris's Case.* Of the paternal grandparents, I. 1 and 2, nothing known; assumed to be normal. I. 3, the maternal grandfather, died from unknown cause. I. 4, maternal grandmother, living and in good health, aged 83; no history of importance found in relatives, other than those recorded. II. 2, alive and well (1903). II. 3, living, has swellings of her hands, similar to those of the patient, III. 1; she has also stomach trouble; after death of III. 1, she has shown more numerous evidences of angioneurotic oedema; the swellings, however, have been chiefly on limbs and face; she has had no throat symptoms, although neck has lately been inclined to swell; of her children, III. 2 and 3 are living and well, III. 4 was still-born, III. 6 died of "epilepsy" at the age of 18 months, III. 7, another child of the mother's, it is said was "choked to death" suddenly at the age of two weeks; the other two siblings are oedematous. III. 5 is living, "any blow on her face causes it to swell." III. 1, stenographer, died, aged 23; he was first affected at age of 12 in the hands, later in feet, knees, elbows, scrotum and penis; at 17 he had oedema of glottis, for which he was tracheotomised; another attack had to be relieved by introduction of trocar into trachea; had abdominal crises, nausea, vomiting, pains in epigastrium; since the autumn of 1903 the patient has had two attacks of oedema of the larynx, the second proving fatal; in the first attack the patient was very dyspnoetic and somewhat cyanotic; he inspired with great difficulty; he gradually became relieved in three hours with $\frac{1}{4}$ gr. of morphia; the swellings in his body following slight blows became more severe and numerous after this attack; the fatal attack occurred in 1905, the medical man finding the patient dead on his arrival; partial autopsy revealed mouth and pharynx filled with blood, no special change in lungs, but larynx presented a most extreme grade of oedema, which involved epiglottis, aryteno-epiglottidean folds, the false and true vocal cords, the ventricle being obliterated. (See Bibl. No. 37, 1904, p. 812 and 1906, p. 382.)

Fig. 103. *Ensor's Case.* Ensor has provided the most complete history yet published of family angioneurotic oedema. The original account, which comprised 80 members, 33 of which were known to be affected, was published in 1904. Dr Ensor has made a further extensive investigation of the family for the *Treasury*, and our pedigree is now constructed from the new data supplied by him. The family at present includes 141 members in seven generations; some of the members live in Ansty in Wiltshire (England). Of these 141 people a definite account exists of 49 who have been affected, viz. 28 males and 21 females. Of those affected no fewer than 12 died from suffocation due to oedema of the glottis. With the exception of IV. 50, who was first attacked at the age of eight, the disease usually commenced about the age of puberty. Of those that succumbed directly to oedema of the throat, one was under 20, three were from 20—30, two were between 30—40, and five were between 63—70. The exact age of the twelfth fatal case is not definitely known, but she was over middle age. In this family the disease was of classical Quincke type with marked abdominal symptoms in many of the cases. In one instance, V. 20, the disease was inherited through a normal unaffected mother, IV. 19, and in two, VI. 19 and 20, through an unaffected father, V. 44. I. 1, aged 70, was the first member of the family known to be affected; he was liable to swellings in various parts of his body, and was found lying dead in his house in 1843. I. 2, his wife; no history given but presumably healthy. II. 1, son of I. 1; was frequently the subject of oedematous attacks, but did not die of the disease; his nephew, III. 20, says he had attacks of intestinal colic and vomiting every 10 days and lasting about 12 hours; they were independent of the attacks of oedema. II. 2, wife of II. 1; healthy; no blood relationship to her husband. II. 3, aged 61; died of acute abdominal disease, said by the doctor who attended him to be inflammation of the stomach; he was taken suddenly ill with pain in the stomach and vomiting and died in 24 hours; he suffered from attacks of colic every nine days, and is known to have had repeated attacks of oedema. II. 4, wife of II. 3; her son, III. 24, informed Ensor that she was healthy and not related to her husband. III. 1, aged 66; liable to sudden swellings of the throat and other parts of the body; on Oct. 16th, 1895, he was returning home from his work, and at 3.50 p.m. was seen by a shepherd, who spoke to him; he seemed to be in his usual health, but at 4.5 p.m. he was found dead at the roadside near the spot where he had spoken to the shepherd; Dr Ensor saw the body half an hour later; it was lying prone, the hands clenched, the face

and visible mucous membranes of a purplish colour; an autopsy was not permitted. III. 2, wife of III. 1; healthy. III. 4, sister of III. 1: beyond the fact that she was the subject of oedematous attacks but did not die of suffocation, details are wanting; she married and had two normal daughters. III. 5, was liable to colic and had often been affected with swelling of face and hands; his daughter, IV. 11, says his face was frequently so swollen that his eyes were closed up; he had an attack of oedema of the hands on the morning of May 24th, 1902, but was able to go to work; he returned home however about midday, as his throat was becoming affected; a doctor was sent for, but the patient had succumbed with suffocation at 1.45 p.m. before his arrival. III. 6, his wife; healthy; not related to husband. III. 7, affected; married III. 8, but had no children. III. 9, normal. III. 10, died at 64 years; affected with oedema but did not die of it; his daughter, IV. 22, says he had many attacks in the face, hands and throat, and also attacks of colic; on one occasion, in 1883, he was threatened with suffocation during a throat attack lasting three minutes; he ultimately became an imbecile, and is said to have died from cerebral softening. III. 11, wife of III. 10, normal; not related to husband. III. 12, affected; he married but had no children; was the subject of frequent attacks of epistaxis not always connected with the oedema; did not die of suffocation. III. 14, affected and died of suffocation; married and had three children, all of whom, IV. 25, 27, 29, were affected. III. 16, affected, aged 60; cause of death not known. III. 17, his wife; healthy; unrelated to husband. III. 18, known to have suffered from swellings in throat; he enlisted in the Grenadier Guards and died during an attack of suffocation on May 2nd, 1853, at the age of 22. III. 19, 20, 22, 26 and 27, were healthy and unaffected. III. 24, aged 68; used to suffer severely, but for the last three or four years has been comparatively free; he now suffers oedema chiefly in hands and feet, whereas earlier in life he had repeated swellings in his mouth and throat which on one occasion threatened to prove fatal; he has been very subject to colic and abdominal pain but is now more free. (Further note taken March 21st, 1909.) Since the previous note he has had many manifestations of his disease, and is now chiefly troubled with severe colic and vomiting which appears to have returned with increased frequency; during a recent attack of colic, the whole of one arm was much swollen and became blistered after he had bathed it with hot water: *vide* V. 27. IV. 1, aged 50; suffered frequently from swelling of the throat or about the body; he was attended by Dr Eusor for severe colic in 1897, although he had no external visible oedema at that time; on three occasions the throat swellings have threatened suffocation. (Note made March 21st, 1909.) Attacks continue with equal frequency, mostly take the form of severe colic and circumscribed oedema of arms; patient during the past few years has had several attacks of epistaxis: *vide* III. 12 and IV. 23 and V. 23. IV. 2, wife of IV. 1; healthy; not related to husband. IV. 4, and oldest and youngest of IV. 5, not affected. IV. 5, middle brother, is aged 40; stated on Dr Ensor's chart to suffer with urticaria. (Note.) The patient's description of the urticarial attacks is typical of the common lesion and does not warrant his inclusion as an affected member of the family. IV. 6, both normal. IV. 7, aged 26; died of oedema of larynx, Oct. 16, 1883; he had oedema of hands several times but only the one throat attack. IV. 8, normal; has had nine children, V. 6—11, all normal. IV. 10, aged 60; affected and died at Hereford (Jan. 22nd, 1901) of oedema of larynx; his sister, IV. 23, received a letter stating that he was taken suddenly ill and died of suffocation with swelling of throat; his sister, IV. 21, says that she believes tracheotomy was performed when the patient was moribund. IV. 11, unaffected; died of phthisis. IV. 12, subject to oedema; in March 1885 he was found sitting by the roadside gasping for breath; he was able to say that his throat was swelling and died a few minutes after. IV. 13, his wife, healthy. IV. 14, affected; enquiries regarding details were negative except that he is known to have been affected. IV. 15, normal. IV. 17, died, aged 41; liable to attacks of oedema from girlhood; on the afternoon of June 29th, 1891, while engaged in housework, her face became swollen; she was however able to go to tea at her sister's house, but a little later the swelling rapidly increased and her eyes became closed; breathing became laboured, and within a few minutes she died of suffocation; she was seen by Dr Ensor. IV. 19, normal; this is one of the two instances in this family where a normal person transmitted the disease; she is not mentioned in Dr Ensor's text; only one (V. 20) of her eight children is liable to the disease. IV. 21, normal. IV. 23, aged 55; has suffered from oedema as long as she can remember; has had 15 or 20 attacks in the throat; has had four children all affected, one of whom, V. 29, died of suffocation. (Note on March 17, 1909.) Attacks during the last two years have become more frequent; the tongue and throat have swollen twice during this period; six weeks ago she was threatened with suffocation, and during the attack there was profuse epistaxis. IV. 25, affected, severely; had many attacks of intestinal colic and swelling of hands and arms. IV. 27, affected, died, aged 65, but not of oedema; intestinal colic was a marked feature, but she had many attacks of oedema of face, hands and feet. IV. 29, affected, chiefly colic; accidentally killed; his son, V. 44, tells Dr Ensor that the attacks would sometimes be so severe as to incapacitate his father from work for two or three days. IV. 31, 32, 33, normal. IV. 34, normal, married second cousin, V. 22, also normal; they have three children, VI. 7, 8, 9; this is the only instance of intermarriage in the family I can discover; their ages are seven, six and four years; it will be interesting to note their further history; at present they are probably too young to show any sign of the disease. IV. 35, affected, not married; in letter of April 2, 1909, he says he still suffers, especially in his stomach, and his hands and arms swell. IV. 36, affected. IV. 38, 40, 42, 44, 45, 48 and 49, all normal. IV. 50, died, aged 16;

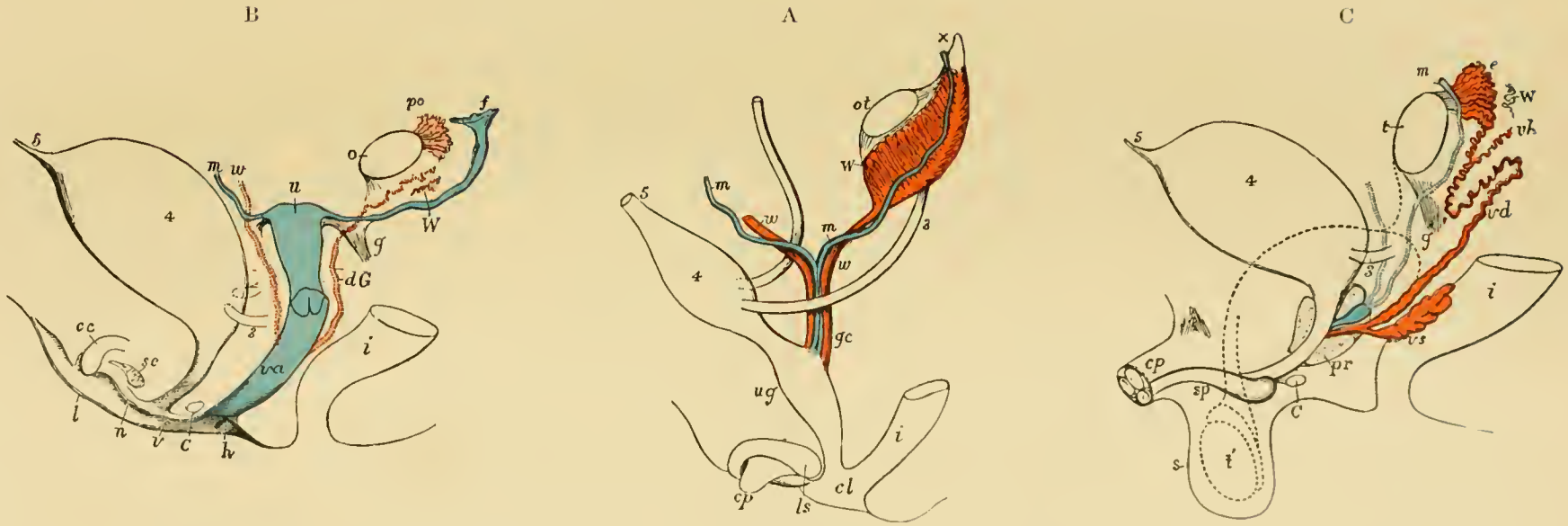
was first attacked at the age of eight; on April 15th, 1883, one of her hands swelled; she went to bed and slept, but in the early morning awoke with sensation of choking from which she died in 1½ hours. V. 1, aged 14, affected; has had no laryngeal attacks; hands and arms usually affected; a slight blow will sometimes cause an attack. V. 2, died of scarlet fever at 12 months. V. 3, affected, aged 13; died in twelve hours from some acute lung trouble; she was seen by Ensor, who certified the case as acute capillary bronchitis, but he thinks that possibly it may have been acute oedema. V. 4, normal. V. 6—11, normal. V. 13, affected; has two normal children; very liable to colic and swelling of hands. V. 14, affected; an only daughter is free. V. 16, normal. V. 18, 21, both normal. V. 19, aged 18; affected; had first attack in 1899, when she was 15 years old, and three subsequent ones, two of face and one of the hands. (Note on April 1st, 1909.) Her aunt, IV. 23, tells Dr Ensor that she has had many attacks since and is subject to colic. V. 20, aged 29; affected; mother, IV. 19, normal. (Note, March 25th, 1909.) Has had only one attack, six or seven years ago; in this instance it is possible that the determining influence may have been due to slight trauma or nervous shock; the attack supervened two or three hours after a choking fit, caused by some food or liquid getting into the larynx. V. 21, normal; three children all normal; this woman married her cousin, IV. 34. V. 23—24, all normal, ages 27, 25, 23, 21, 18, 15. V. 28, liable to epistaxis, no apparent cause. V. 25, normal; married, no children. V. 27, aged 29; affected; during an attack of oedema of the face, larynx, and buccal membrane, the face was bathed with very hot water which blistered her; a day or two later she developed facial erysipelas from which she died: *vide* III. 24. V. 29, died, aged 24; affected; has had several attacks of oedema; on October 16th, 1895, he was much agitated at hearing the cause of death of his grand-uncle, III. 1, and two days later Dr Ensor was called to see him, but on arrival found him dead; he had been suddenly attacked with dyspnoea and succumbed in a few minutes; his mother stated that he had several times been threatened with suffocation and was afraid to go to sleep when his face was oedematous. V. 30, aged 25; affected in hands, but has had no attack which threatened suffocation. V. 31, aged 24; affected; suffers from swelling of hands and has had three attacks in the throat. (Note, March 17, 1909.) His mother, IV. 23, informs me that the attacks continue and he has several times been threatened with suffocation; he is married and has two sons, VI. 10 and 11, both free at present. V. 33, both normal. V. 34, aged 50; affected; has severe colic and swelling of face and arms; married and has one daughter, VI. 12, who is normal. V. 36, aged 53; normal. V. 37, normal. V. 38, aged 44; affected; gets severe colic frequently, and less often her hands and face swell; is married and has one daughter, VI. 13, who is affected. V. 40, affected; intestinal colic and oedema of hands and face; has frequent attacks; married, and has one daughter. V. 42, normal. V. 44, normal, but has transmitted the disease to two of his children. V. 46, aged 39; affected; is chiefly troubled with colic and swelling of hands, but has never had oedema of face or larynx; during the last few years he has been less liable but previously like his father, IV. 29, he was sometimes unable to work owing to the severity of the abdominal pain. V. 48, aged 37; affected; in same way as his brother, V. 46. V. 50, aged 34; affected; has four normal children. V. 52, aged 33; normal. V. 53; affected; aged 30; colic and oedema of hands and arms; has had swelling of face. V. 55, affected; aged 22; liable to oedema of hands and face and intestinal colic; his brother, V. 46, says he does not think he has ever had a suffocative attack. V. 56 and 57, all normal. V. 58—64, all normal. VI. 1, female, normal. VI. 2—12, normal. VI. 13, aged 25; affected, hands and face. VI. 15, 17, 18, all normal. VI. 19, aged 22; affected, and VI. 20, aged 21; affected; disease transmitted through normal father, V. 44. VI. 21, normal. VI. 22, aged 21; affected; she says that her attacks are chiefly intestinal, but that sometimes her hands and arms swell. VI. 23, aged 12; has been subject to attacks of swelling of hands and arms for the last 12 or 18 months. VI. 24—28, normal. VII. 1, aged three years; probably too young to have shown any symptoms. (See Bibl. No. 14, 1904. This has been supplemented by additional information specially provided for this *Treasury*.)

Fig. 104. *Wagner's Case.* The original was not consulted personally; our account was kindly abstracted by Prof. J. Morgenroth in Berlin. I. 1, father of Wagner's patient; suffered occasionally from sudden swellings, especially of hand. I. 2, no information. II. 1, "Handelsmann," aged 50; acute swelling of left axilla, disappearing in 24 hours; six weeks later swellings on both soles; six months later swelling of L. hand and arm, lasting 48 hours; fourteen days later swelling of L. knee; again in six weeks enormous swelling of prepuce and a little later swelling of left upper arm. (See Bibl. No. 30, S. 471.)

SECTION X α. HEREDITARY MALFORMATION OF THE GENITAL ORGANS, HERMAPHRODITISM.

By W. Bulloch, M.D.

The possibility of hermaphroditism in man arises from the fact that at a certain stage in embryonic life the rudiments of the future sexual organs are in a bisexual, undifferentiated state. At a later period development proceeds normally in one or other of two directions, viz. male, or female, certain parts becoming the permanent sexual organs whereas other parts undergo arrest of development or atrophy and are represented in post-embryonic life as mere rudiments. In certain invertebrates the simultaneous development of male and female organs of generation in one and the same individual is the rule, the result being a true hermaphrodite, *i.e.* an individual capable of impregnating another and at the same time capable of being impregnated by another individual or even by itself. The occurrence of male and female sexual glands (testicles and ovaries) has been frequently asserted to occur in man, but even the cases apparently most authentic have not withstood the test of careful microscopic examination (cf. Meixner on the cases reported by Heppner, Obolonsky, Schmorl and Blacker and Lawrence). Theoretically a testis and an ovary might coexist in one and the same individual, but up to the present such cases have not been the objects of actual observation. Within the last few years, however, some five cases have been examined in which both testicular and ovarian tissue—so-called ovotestis—has been found in one and the same sexual gland. In no case, however, have both tissues been functionally perfect. The five admitted cases of ovotestis are those recorded by von Salen, Garré, L. Pick (two cases), and Schickele. Apart from them all other reported cases of hermaphroditism come really in the category of pseudohermaphroditism, the characters of which are that the appearance of the external genitals does not conform to the type of sexual gland present but are heterosexual. In other words, with sexual organs apparently female externally and internally or both, the sexual glands are testes, or with sexual organs apparently male externally the sexual glands are ovaries. In other cases both male and female sexual ducts may be developed in greater or less degree internally, whereas the external genitals may be of one type only, male or female, according to the character of the sexual gland (testicle or ovary) present. In other cases again the whole genital system may remain so rudimentary and undeveloped that even with the most careful microscopic examination it is impossible to pronounce the character of the sex at all, such individuals being, strictly speaking, sexless—*homines neutrius generis* (Virchow). Pseudohermaphroditism in its essence, as Neugebauer has said, is a paradoxical incongruity between the genital *glands* on the one hand and the genital *ducts* and external genitals on the other. As far as concerns the latter the male type of the external genitals in pseudohermaphroditism is to be interpreted as a formation *per excessum* of the female external genitals, whereas the female type of pseudohermaphrodites is to be regarded as an arrest of development of the male external organs. This is clear



Diagrams to show the development of male and female generative organs from a common type. (Allen Thomson.)

B.—Diagram of the female type of sexual organs.

o, the left ovary; *po*, parovarium (epoophoron of Waldeyer); W, scattered remains of Wolffian tubes near it (paroophoron of Waldeyer); *dG*, remains of the left Wolffian duct, such as give rise to the duct of Gartner, represented by dotted lines; that of the right side is marked *w*; *f*, the abdominal opening of the left Fallopian tube; *u*, uterus; the Fallopian tube of the right side is marked *m*; *g*, round ligament, corresponding to gubernaculum; *i*, lower part of the intestine; *va*, vagina; *h*, situation of the hymen; *C*, gland of Bartholin (Cowper's gland), and immediately above it the urethra; *cc*, corpus cavernosum clitoridis; *sc*, vascular bulb or corpus spongiosum; *n*, nympha; *l*, labium; *v*, vulva.

A.—Diagram of the primitive urogenital organs in the embryo previous to sexual distinction.

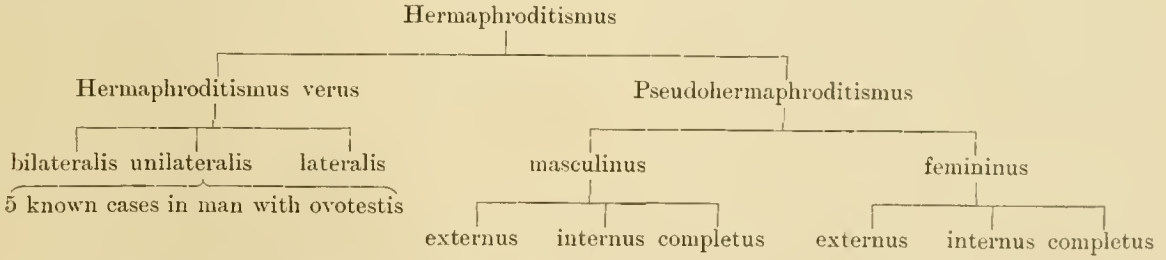
3, ureter; 4, urinary bladder; 5, urachus; *ot*, the genital ridge from which either the ovary or testicle is formed; W, left Wolffian body; *w*, *w*, right and left Wolffian ducts; *m*, *m*, right and left Müllerian ducts uniting together and running with the Wolffian ducts in *gc*, the genital cord; *ug*, sinus urogenitalis; *i*, lower part of the intestine; *cl*, cloaca; *cp*, elevation which becomes clitoris or penis; *ls*, fold of integument from which the labia majora or scrotum are formed.

C.—Diagram of the male type of sexual organs.

t, testicle in the place of its original formation; *e*, caput epididymis; *vd*, vas deferens; W, scattered remains of the Wolffian body, constituting the organ of Giraldès, or the paradidymis of Waldeyer; *vh*, vas aberrans; *m*, Müllerian duct, the upper part of which remains as the hydatid of Morgagni, the lower part, represented by a dotted line descending to the prostatic vesicle, constitutes the occasionally existing cornu and tube of the uterus masculinus; *g*, the gubernaculum; *vs*, the vesicula seminalis; *pr*, the prostate gland; *C*, Cowper's gland of one side; *cp*, corpora cavernosa penis cut short; *sp*, corpus spongiosum urethrae; *s*, scrotum; *t'*, together with the dotted lines above, indicates the direction in which the testicle and epididymis descend from the abdomen into the scrotum.

when one considers the development of the whole urogenital system. In the indifferent bisexual stage it is possible to distinguish the Wolffian body with its duct from the Müllerian duct and the germ epithelium which constitutes the rudiment of the future genital gland, the nature of which determines the sex (Plate F, Fig. I.). As development progresses, the Wolffian body (mesonephros) and the germinal epithelium combine together to form the future testicle of the *male*, whereas the Müllerian duct even in the seventh or eighth week undergoes involution, being ultimately represented as the utriculus masculinus and the sessile hydatid of the epididymis (Fig. II.). In the female, on the other hand, the Müllerian ducts attain cardinal importance whereas the Wolffian ducts sink into obscurity, in so far that they are represented by the parovarium and the duct of Gartner, the Müllerian ducts constituting the Fallopian tubes, uterus and vagina (Fig. III.). The development of the external genitals is equally indifferent in the early embryo. Both Wolffian and Müllerian ducts open at first into the sinus urogenitalis, which above is connected with the bladder and below with the cloaca, into which latter also opens the rectum. By degrees a septum is developed between the rectum and the urogenital system, but before this has taken place the genital eminence from which the penis or clitoris is formed makes its appearance in front of and within the orifice of the cloaca. By the development of the septum two apertures are thus produced, a dorsal or anal and an anterior or urogenital, the latter a diverticulum as it were of the cloaca, carried outwards by the growth of the genital eminence or rudiment of the penis. This genital eminence is surrounded by a fold of integument which is the basis of the *mons veneris* and labia majora in the female and when united by median fusion of the scrotum and skin of the penis of the male. The lips of the urogenital furrow, which in the female become the nymphae, unite in the male to form the integument below the penis. Before closure takes place the external organs of generation appear alike in both sexes. If closure is interfered with the condition is called hypospadias, and this may exist in all degrees from complete cleavage of the scrotum and penis up to the slight forms in which the urethra opens on the under surface of the glans instead of on the end. In the female the clitoris remains small, the groove on its under surface becomes less marked owing to its opening out and continuance as the nymphae.

For the purposes of classification it is customary to subdivide cases of hermaphroditism as follows:



Pseudohermaphroditismus masculinus. Most of the recorded cases are to be placed in this group. In its complete form there are testes, vasa deferentia and prostate gland, but also development in varying degrees of completeness of Müller's ducts in the form

of Fallopian tubes, uterus and vagina. The external genitals are apparently of female character. In pseudohermaphroditismus masculinus externus—the commonest form of genital malformation—the external parts appear like those of a female whereas the internal organs are of male character. The external genitals, apparently female in type, owe their origin to hypospadias peniscrotalis with more or less incomplete development of the hypospadic penis. In cases where the penis is quite rudimentary and clitoris-like and the testicles are undescended the resemblance to a female may be striking. Complete development of the penis with a cleft state of the scrotum are rare, but all degrees of cleavage exist up to the slight deformity where the urethra opens on the under surface of the glans instead of at its apex.

In the internal form the external genitals may appear to be perfectly male, but in the pelvis alongside normal or rudimentary Wolffian ducts and their derivatives there may also be Müllerian products (uterus, tubes, vagina). In such cases the vagina opens on the caput gallinaginis. The secondary sexual characters of male (androgynoid) hermaphrodites are very varied. In some cases the general habitus, breast development, voice, beard, etc. may be absolutely of male type, at other times these individuals resemble females or may be neuter[1].

Pseudohermaphroditismus femininus (gyanandroid formation, *Pseudoarrhenie*). In its complete form, which is very rare, the external genitals are of male type while internally there are ovaries and more or less completely developed Müllerian and Wolffian ducts. In the "internal" form the external genitals are female in type, while internally, in addition to uterus and appendages, there may be Wolffian derivatives or the latter may alone exist. In the "external" type the resemblance to the male is due to the hypertrophy of the clitoris which may indeed reach a size comparable to a penis. Adhesion of the labia may complete the simulation to a male scrotum, especially if combined with ectopic ovaries or a rupture.

The occurrence of hermaphroditism has at all times excited interest among the profession and the laity, and fabulous tales have been handed down with respect to the procreative powers of those malformed in this way. With the revival of learning and the rise of anatomy, however, the condition was thoroughly studied and explained, the most important monographs on the subject being those of Caspar Bauhin, Arnaud, Steglehner, I. Geoffroy Saint-Hilaire, Guenther, Taruffi and von Neugebauer, the last a monumental work of over seven hundred pages. The frequence of hermaphroditism and abnormalities in the urogenital development is much greater than is usually supposed. With regard to the minor degrees of hypospadias Rennes found 10 cases among 3000 French conscripts, *i.e.* 1 : 300. That the condition may also occur in several members of a family or even in several generations has been known from the time of Bauhin (1614). Unfortunately the pedigrees of the affected families are almost all more or less incomplete, and to add to the difficulty of ascertaining the original data the references to the cases, especially the older ones, are, even for medical literature, extremely inaccurate and misleading.

[1] The extraordinary difficulty which in some cases may attend any attempt to determine sex is well illustrated by v. Neugebauer's plate (S. 110) of de Beurmann and Roubinowitch's case of *Pseudohermaphroditismus masculinus*. The external genitals give no clue to the sex, the figure and general habitus are essentially feminine; there was no uterus, but testes existed with seminal fluid, which however contained no spermatozoa.

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PLATE XVI. Fig. 105. *Pozzi's Case.* I. 1, normal, no consanguinity with his wife, I. 2. II. 2, ♀, married and has four children; it is not stated whether these are affected. II. 3, believed to be a female, but probably male; the mother believes its genitals were malformed; died at age of 19 months. II. 4, 5, 6, normal females, died young. II. 7, aged 29, has a female illegitimate child, III. 2. II. 9, normal male, married. II. 11, declared to be female at birth but a male at age of 17; lives with a woman. II. 12, aged 18, christened a female but is really a hypospadic male. (See Bibl. No. 27, p. 109.)

Fig. 106. *de Beurmann and Roubinowitch's Case.* III. 1, 20 years old, looks like female; long hair on head, no beard, well-developed breasts; penis hypospadiaeus 2—3 cm. long; scrotum cleft; on each side a testicle, no uterus; at school III. 1 was known as "Mademoiselle-monsieur," as her instincts were boyish; at 15 "she" fell in love with a girl; she also cohabited with men; her brother, III. 2, committed suicide; an "uncle," II. 3, and an "aunt," II. 4, were also hermaphrodites; the uncle committed suicide; the "aunt," II. 4, a prostitute in London, cohabited with men and women; she was a male with penis-scrotal hypospadias; II. 1 up till 26 had no hair on his face, II. 2 was mentally weak. (See Bibl. No. 28, p. 47.)

Fig. 107. *Born's Case.* Man, I. 1, with foreskin so narrow that he could expose the glans only with great difficulty; he also showed a slight degree of hypospadias, the urethra opening behind the frenum; his son, II. 1, was affected in like manner, but had four sons all normal. (See Bibl. No. 29, p. 113.)

Fig. 108. *Ritter's Case.* Boy with imperforate anus, died on 15th day after birth; he had two normal sisters; his father and grandfather were hypospadics; no details of other members of family given. (See Bibl. No. 30, p. 350.)

Fig. 109. *Heuermann's Case.* Wrongly cited by every author from Meckel onwards. Our pedigree is obtained from a quotation extracted by Prof. C. J. Salomonsen, who found Heuermann's work in an uncut state in the University Library, Copenhagen. The case is that of a family with two male hypospadics of the peniscrotal type so that at birth it was uncertain to what sex they really belonged. The mother informed Heuermann that malformations like her children occurred in her family from time immemorial and were propagated from one generation to another. She herself was married to a normal man of a different family. Her brother was also a hermaphrodite and had married a woman although he had no children by her. The woman's parents also alleged that the malformation of the genitals was known to be in the family, in every generation of which some of the males were affected but never the females. The latter, however, even on marrying normal men of a different family, were capable of transmitting the malformation to their male offspring. (See Bibl. No. 31, p. 234.)

Fig. 110. *Lindsay's Case.* Three hermaphrodites in a family of nine. I. 1 and I. 2 say they are normal, but I. 1 wears a truss for a double inguinal hernia; no abnormality known among the relations. II. 1 and II. 2, sex not stated, died of scarlet fever. II. 3, a miscarriage. II. 4 and 5, stated to be normal but no sex stated. II. 6, seen by a doctor at birth, and on his advice reared as a male. II. 7 and 8, who followed in immediate succession, were not seen by a doctor but parents of their own notion and in accordance with opinion of midwife reared them as girls (Jessie, aged six, and Lizzie, aged five); they were hypospadic males. II. 9, normal ♂. (See Bibl. No. 32, p. 161.)

Fig. 111. *von Neugebauer's Case.* Two male hypospadics, brought up as girls; mother had 14 of a family of which the majority died in early childhood of infective diseases; it is not stated or known whether any of those dead were malformed. II. 2, "Katharina," brought to Neugebauer in 1895 for pain and swelling in groin; on examination "she" proved to be a male hypospadic with undescended testicles; her general habitus and voice were male; andromastie; erectile split penis 5½ cm. long; below, a vaginal opening with a hymen; vagina 2—3 cm. long, a cul-de-sac; five years later she was again seen, and it was noted that she showed male characteristics more pronounced than formerly; abundant hair on face, breast and extremities, nates and pubis; erections of penis; testicles palpable; in 1904 her sex was altered by law. II. 3, "Martha," "sister of above," brought to Neugebauer; similar to II. 2 and undoubtedly a male. II. 4, normal girl, five months old. (See Bibl. Nos. 11, S. 419, and 33, S. 673.)

Fig. 112. *Fenoglio's Case.* Three children, I. 1—3 (out of five), with same malformation probably; all male hypospadics, witb cryptorchismus. (See Bibl. No. 34, not accessible. Reference in Neugebauer, Bibl. No. 11, S. 183.)

Fig. 113. *Matthei's' Case.* Four daughters of a peasant in Rome. II. 1, married and had children, the other three were really males, with hypospadias peniscrotalis. (See Bibl. No. 35, not accessible. Reference in Taruffi, Bibl. No. 10, p. 312, and Neugebauer, Bibl. No. 11, p. 322.)

Fig. 114. *Matthes' Case.* Woman after a series of children had two, II. 2 and 3, which were the subject of extensive malformations, viz. hernia cerebri, meningocele occipitalis, labium leporinum, palatum fissum, scrotum fissum, situs partim inversus viscerum; the urethra had no external opening, but there was no retention of urine as the urachus was open; testicles were undescended; polydactyly of all four extremities; no reference is made to other members of the family. (See Bibl. Nos. 36 and 38, p. 761.)

Fig. 115. *Van der Hoeven's Case.* Two sisters, who were in reality male hypospadics. II. 2, aged 23, of feminine appearance; penis cleft, two inches long, testicles in each side of the split scrotum; no pubic hair, no uterus, prostate, seminal vesicles or ovaries. II. 1, 28 years, exactly same as II. 2. (See Bibl. No. 39, cited here from Neugebauer, No. 11, p. 255.)

Fig. 116. *Levy's Case.* Levy says that he himself was born "regelrecht geschnitten," so that there was nothing to circumcise, his father fulfilling the law by making a small incision with a needle till blood came. Not only Levy but his four brothers, all dead as children, showed the same peculiarity. (See Bibl. No. 40, p. 539.)

Fig. 117. *Parlier's Case.* On p. 492 Bouisson refers to a personal communication made by M. Parlier with reference to hypospadias in three young children of a family, the result of a consanguineous marriage. No other details or relationship of parents. (See Bibl. No. 41, p. 492.)

Fig. 118. *Fancourt Barnes' Case.* Family of 11 children, two of which were hermaphrodites. II. 1, aged 19, brought up as a girl; at nine years of age hair began to grow on pubis and spread up towards umbilicus; about 16 moustache and beard began to grow; examination showed a sessile penis like a clitoris; no uterus, ovaries or testicles could be found; the breasts were of the male type. II. 3, youngest child examined by Barnes and found exactly to resemble II. 1; no family history given except that when mother, I. 2, was two months pregnant with II. 1 she had a fright. (See Bibl. No. 42, p. 203.)

Fig. 119. *Martin's Case.* Two hermaphrodites in a family. I. 1, insane at time of conception. II. 1, aged 22, has never menstruated, no pubic hair, vagina a short cul-de-sac, no sign of uterus. II. 2, aged 20, brought up as a girl and earned her living as a nurse; has never menstruated; at 19 was operated upon by a surgeon for right inguinal hernia; at the operation a solid body, supposed to be an ovary, presented in the wound, but was replaced in the abdomen; when seen by Martin 12 months later she was typically feminine but with poorly-developed breasts; no hair on pubis; external genitals like a normal nulliparous female; clitoris not enlarged, vagina a cul-de-sac $3\frac{1}{4}$ inches deep, no cervix or uterus, no evidence of prostate gland; swelling in left inguinal canal was operated upon and proved by microscopic examination to be a testicle with imperfect spermatozoa; no other details as to family history. (See Bibl. No. 43, p. 35.)

Fig. 120. *Mundé's Case.* No history or details except that two "sisters" (*sic!*), who failed to menstruate up to the 20th and 22nd year respectively proved on examination to be hypospadic males. (See Bibl. No. 44, p. 334.)

Fig. 121. *Chiarleoni's Case.* Case of two hypospadic brothers brought up as girls; no details as to pedigree. (See Bibl. No. 45, p. 126.)

Fig. 122. *du Basty's Case.* Two "sisters," 42 and 35 years of age respectively; they consulted Mabaret du Basty to know whether the younger one was fit for marriage. On examination she showed a male physique, with andromastie and deep voice, face shaved, clitoris 4 cm. long; below urethra is a 5 cm. long canal out of which blood is said to flow monthly; the elder sister with similar malformation. Parents stated to be normal. (See Bibl. No. 46, p. 502.)

Fig. 123. *Shorthouse's Case.* Case of a poor woman who suffered from an intrauterine malformation of the hands; she married a man whose urethra opened under the penis behind the glans; by him she had four sons all hypospadic like the father, and two daughters with hand deformities like herself; no other details given. (See Bibl. No. 47, p. 512.)

Fig. 124. *Bryant's Case* (ii). Patient, I. 1, aged 25, with urethral orifice below glans penis; he was father of three sons, II. 1—3, all affected in like manner; no other details. (See Bibl. No. 48, p. 419.)

Fig. 125. *Bryant's Case* (iii). Man, I. 1, had four children, three of whom, III. 1—3, were males; all three, like their father, had hypospadias, the opening of the urethra corresponding to the frenum; no other details. (See Bibl. No. 48, p. 419.)

Fig. 126. *Liersch's Case.* Two individuals in one family, both hermaphroditic. II. 1, aged 21, hair on face, andromastie, hypospadias, peniscrotalis or clitoris hypertrophy with labial union. II. 2, about 15; post mortem examination showed andromastie, penis hypospadiaeus, scrotum cleft only in upper part, uterus 4 cm. long, with tubes, ovaries well developed but no follicles. (See Bibl. No. 49, S. 519. Cited from Neugebauer, Bibl. No. 11, S. 297.)

Fig. 127. *Traxel's Case.* Hypospadias in father and son. The child, II. 1, was born to a peasant girl and was brought to Traxel to have its sex determined; it proved to be a hypospadic male, with testicles; the mother declared that she had never been with a man but admitted "geschlechtliche Umgang" with a girl, Johanna K. The latter, I. 1, 37 years old, was made like a man; face shaved,

breast hairy, voice a deep bass; on medical examination "she" proved to have hypospadias peniscrotalis with complete cleavage; urethra opened into perineum; a testicle was present on each side of the cleft scrotum. (See Bibl. No. 50, S. 289.)

Fig. 128. *Simeons' Case* (i). A hypospadic man, father of six children, two sons being hypospadic; no other details. (See Bibl. No. 51, p. 228.)

Fig. 129. *Simeons' Case* (ii). Hypospadias in two brothers; no details. (See Bibl. No. 51, p. 228.)

Fig. 130. *Legueu's Case.* Two "sisters," 16 and 14 years respectively; both were hypospadic males with testicles; no other details. (See Bibl. No. 52, p. 140.)

Fig. 131. *Belloc's Case.* I. 1, had the urethra opening at the base of the frenum glandis, *i.e.* a slight degree of hypospadias; he had four children, two of whom were similarly affected; order of birth not stated; sex of unaffected not stated. (See Bibl. No. 53, p. 596.)

Fig. 132. *Burgess' Case.* Two boys, V. 1 and V. 2, 8 and $2\frac{1}{2}$ years respectively, the subjects of hypospadias; parents not related; mother's father's uncle and cousin also affected. "The mother's father's aunt states that on the maternal side the great-grandfather's brother suffered, also the great-grandfather's nephew." (See Bibl. No. 54, p. 11.)

Fig. 133. *Haim's Case.* Hypospadias in two individuals supposed to be girls; no other cases known in family. II. 2 and II. 3, brothers. V. 1, "Barbara," aged 20, hypospadic male with blind vaginal sac and testes in the cleft scrotum. V. 2, ♀, aged 13, similarly affected. (See Bibl. 55, S. 335.)

Fig. 134. *Rigaud's Case.* An individual, I. 2, was father of six children, of which three were hypospadians, III. 1—3; the father and two uncles of this individual were also hypospadics. (See Bibl. 56, p. 586.)

Fig. 135. *Nonne's Case.* II. 1, aged 21, admitted to Klinik at age of 16 for inguinal rupture; male type of body, hair on face, penis the length of a finger, cleft underneath; vagina $2\frac{1}{2}$ cm. long, a cul-de-sac; scrotum cleft, testicle on each side, no prostate or uterus; at age of 21 "she" was of very masculine type. II. 2, aged 18, affected with syphilis by a labourer; "she" showed a penis 5 cm. long with peniscrotal hypospadias with testicles; male-like in appearance generally. (See Bibl. No. 57. Cited from Neugebauer, Bibl. No. 11, S. 440.)

Fig. 136. *Van Mons' Case.* Case of twins, IV. 1, 2, affected with hypospadias peniscrotalis and cryptorchismus; the younger died in four months of cyanosis and asphyxia, the elder surviving four days longer; mother a primipara, aged 22; her maternal grandfather had twins also, sex not stated. (See Bibl. No. 58, p. 417.)

Fig. 137. *Sir Everard Home's Case.* A family of three, living near Modbury in Devonshire (1779); the oldest and youngest were "hermaphrodites," the middle one a normal girl. I. 1, a day labourer, presumably normal. II. 1, aged 13, very fat and lazy, mons veneris loaded with fat, no penis, imperfect scrotum, testicles present (hypospadias peniscrotalis). II. 2, normal. II. 3, sexual organs like II. 1; uncommonly fat, idiotic, had a supernumerary finger on each hand and an extra toe on each foot. (See Bibl. No. 59, p. 321.)

Fig. 138. *Sulima's Case.* Russian peasant, II. 1, brought his newly-married "wife," II. 2, to be examined because he maintained she was a man; the "wife," Xenia by name, was 20 years old, tall in stature, masculine face but feminine head of hair; voice, larynx, breast, pelvis, mons veneris of male type; complete hypospadias peniscrotalis with testicle and epididymis on each side; sinus urogenitalis 3 cm. deep, vagina a cul-de-sac; she had desires for neither men nor women; her two "sisters," 9 and $5\frac{1}{2}$ years of age respectively, were similarly affected; three months after marriage the husband applied for divorce, but, according to Russian law, had to wait *three years* after marriage before this could be obtained! (See Bibl. No. 60, p. 111. Cited after Neugebauer, Bibl. No. 11, S. 537.)

Fig. 139. *Fulgoso's Case.* Fulgoso has reported under the reign of Ferdinand I., King of Naples, the case of the children of Louis Guarna, of Salerno. There were five daughters, of whom the two eldest were called Francisca and Carola respectively. When they were fifteen years old the genitals of each took on a male appearance so that it became necessary for them to change their dress, and subsequently they were named Franciscus and Carolus. (See Bibl. No. 61, edition 1604, p. 52.)

Fig. 140. *Curling's Case.* Twins, II. 1 and 2, examined by Curling in 1847; at first he was inclined to regard them as males but afterwards suggested names applicable to either sex; the parents, however, gave them male names; one died in seven weeks, the other in nine weeks; after death it was proved that there were no testicles, but ovaries, uterus and a vagina, the latter opening into the urethra close to the neck of the bladder; two years later the mother had another child, II. 3, similarly malformed

as far as could be ascertained (no post mortem); in a third pregnancy the mother bore a well-formed boy, II. 1; no reference is made to any of the other members of the family. (See Bibl. No. 62, p. 84.)

Fig. 141. *Naegele's Case.* Twins, born 1794, regarded as girls and baptized Katharina and Anna Maria respectively; later their sex was a matter of doubt, and at 17 years of age they took the part of males and were re-baptised Karl and Michael; medical examination showed them to be males with severe degree of hypospadias penisrotalis; father dead, mother alive; no details stated with reference to any other members of the family. (See Bibl. No. 63, S. 136.)

Fig. 142. *Lehmann's Case.* According to the reference in Schmidt's *Jahrbücher* the twins were affected with a slight degree of hypospadias; they also both suffered from encephalocele occipitalis; mother healthy; three normal children preceded the malformed twins. (See Bibl. No. 64, S. 161.)

Fig. 143. *Katzky's Case.* Twins born of healthy parents; the first a living girl, II. 1, normal except that "she was clearly marked with the signs of hermaphroditism"; she died soon after birth. The second, II. 2, also a girl, was "very deformed and monstrous," no head or neck; the genitals were "in places female but with male scrotum depending"; bicornuate uterus; kidneys huge. (See Bibl. No. 65, p. 61.)

Fig. 144. *Bryant's Case* (i). Twins born with hypospadias; father affected in like manner; no other details. (See Bibl. No. 48, p. 419.)

Fig. 145. *Paré's Case.* Twins (joined back to back) represented as hermaphrodites. Paré's account runs: Anno Domini 1486 ad Heidelbergam in agri Palatini pago, Robarchio gemelli duo nati sunt dorsibus cohaerentes et hermaphroditi ambo. (See Bibl. No. 66, p. 743.)

Fig. 146. *Saviard's Case.* Under the title "Upon the delivery of a woman of hermaphrodite twins," Saviard refers to a woman delivered at term of two "hermaphrodites"; one lived eight days, the other six weeks; post mortem examination showed them to be males; penis well formed but urethra absent; scrotum cleft in middle line, like a vulva, at the bottom of which was an opening which resembled a vagina; no details as to parents or pedigree. (See Bibl. No. 67, p. 184.)

Fig. 147. *Lesser's Case.* Family in which hypospadias was hereditary; only two of the 11 cases, viz. IV. 10 and IV. 15, seen personally by Lesser; information with regard to the others stated to be trustworthy, as the family was an educated one and was aware of the hereditary character of their malformation and took an interest in it; most of the information was supplied by III. 3. IV. 10, aged 35, prepuce short, on the lower surface of glans a wedge-shaped depression with a minute opening ending blindly; about 1 cm. behind this is the opening of the urethra; between the depression and the opening of the urethra is another opening from which some drops of urine come. IV. 15, aged 25, condition almost identical with that in IV. 10, except that the minute opening between the depression and the urethra was placed a little further back; no other details are given of the affected individuals in the family; the third and fifth generations were free from the deformity; it is noticeable that II. 3 passed the malformation on to the fourth generation, the third escaping. (See Bibl. No. 68, S. 537.)

Fig. 148. *Strong's Case.* The case seen by Strong was that of a soldier, aged 21, unmarried, who had contracted gonorrhoea; he was found on examination to be a hypospadic, the meatus urinarius halfway between the end of the penis and the corona glandis; penis normal in shape and at the end was an indenture where the normal opening should be; he gave the following family history: His great-grandfather, grandfather, father and three brothers were all affected in the same way; he had never seen his great-grandfather's nor his grandfather's penis, but had been told the above facts; he had seen his father's and three brothers' organs, and one of them he said was "worse" than his own; he had one brother unaffected, and one of his brothers (which?) was married and had hypospadias, had a child "who had the same thing"; no other data given. (See Bibl. No. 69, p. 125.)

Fig. 149. *Lingard's Case.* Cases of hypospadias in six generations; details very meagre. "At the beginning of this (19th) century a hypospadian, whose father and grandfather were also hypospadians, contracted a marriage with a lady not related to him, who bore him three sons, hypospadians; the eldest of these sons married and in his turn became the father of four other hypospadians; of these four sons two married, the first procreating two fresh hypospadians, the second becoming the father of one; the other brothers did not marry;.....the third of the six hypospadians just referred to died a few years after the birth of his three sons; his widow within 18 months after his death contracted a second marriage, the husband in this instance not being a hypospadian and having no history of any such deformity in his family; by this marriage she had four sons, all hypospadians; two of these hypospadic sons had hypospadians in their turn, but one of these sons had three boys without any deformity, although the eldest boy was a hypospadian; the acquired predisposition of the mother thus gradually wore itself out; unfortunately it has been impossible to trace the history through the females of these six generations." In a private communication from Dr Lingard, dated 30. xi. 08, Dr Lingard states that he had great difficulties getting his data as the family objected strongly even to discuss the case. IV. 2 is hypospadic according to the above, but marked normal in Lingard's pedigree. (See Bibl. No. 70, p. 703.)

Fig. 150. *Corrado's Case.* Two children, the seventh and eighth of family, the other members of which were four girls and two boys, all normal. II. 7, Rafaelina, six years old in 1892, but clitoris hypertrophic; no testes to be felt; in 1899 clitoris 32 mm. long. II. 8, Angiolina, born 1890; in 1892 hypertrophic clitoris 2½ cm. long, no testicles to be felt. II. 10, boy, with delayed descent of testis. Corrado regarded II. 7. and II. 8 as ♂ with cryptorchismus. (See Bibl. No. 71, p. 53. Cited from Neugebauer, No. 11, S. 151.)

Fig. 151. *Kellock's Case.* III. 9 and III. 10 showed small ill-developed penis, much curved downwards; groove on under surface of penis lined with mucous membrane, scrotum completely divided, perineum well formed. In III. 9, aged six, two testicles present; in III. 10, aged 2½, testicles have not descended. III. 11, four miscarriages; the order of birth of the miscarriages not stated; father and mother had both 11 brothers and sisters, but neither among them nor their children, which are numerous, have there been deformities of any kind. (See Bibl. No. 72, p. 242.)

Fig. 152. *Corby's Case.* I. 1 and I. 2 were second cousins. I. 1, dead, but was healthy. I. 2, of somewhat large build, with coarse features and marked hirsute development on her face; she is said to have died from cancer, although alive at the date of publication of Corby's paper; she had 12 children, all born at term except II. 10 and II. 11, who were born alive at seven and six months respectively, but died; only three are now alive, one having paralysis of foot from diphtheria; all the other children (except II. 8), born at term, had died of consumption. II. 4 was a "hermaphrodite" (like II. 8), died in eight months of dyspepsia. II. 8, "Willie," aged 15, admitted into Cork South Infirmary with tumour of abdomen; external genitals like a female except that there appeared to be a penis projecting from the vulva; his features were those of a boy and he had an excessive deposit of adipose tissue all over his body; one leg was shorter than the other; he had six toes on each foot, and a supernumerary digit on one hand had previously been removed when he was a baby; his intellectual development was poor; an operation for the removal of the tumour showed that it had started in the left *ovary*; it was a fibroid; a uterus was also felt; he was placed in female wards and was known as "Willie Mary." (See Bibl. No. 73, p. 710.)

Fig. 153. *Stonham's Case.* I. 1 and 2, presumably normal. II. 1 and II. 2, presumably normal. II. 5 and II. 6, husband and wife, parents of III. 15. II. 2, aged 42 years, married 22 years, has had eight miscarriages and 14 children, seven of which are dead from various causes; in none was there a question of malformation. III. 4, normal genitals but has bifid nipple; pregnant in ninth month (IV. 1). III. 5, normal ♂. III. 6, normal ♀. III. 7, a cripple—genital organs normal. III. 8, 12 years old, penis and scrotum well formed, but no testicles. III. 9, six years old, like III. 8. III. 10, operated on for right oblique inguinal hernia; testicles undescended; death after operation for radical cure of rupture; P.M. organs looked of female type, but what looked like ovaries were testicles; uterus dragged over to right side and terminating below in a vagina ending in a prostate gland; lumen from vagina passed through prostate into meatus urinarius; external organs of male type; scrotum deeply puckered with well-marked raphé; penis normal in appearauce but showing a slight degree of hypospadias. III. 12, niece of II. 2, said by II. 2 and II. 3 to have had a child taken from her side; had always been considered a hermaphrodite; she resembled a man; on one side of her genitals is a "lump," but on the other side she is like a woman; she is said to have passed urine through the umbilicus; another niece of II. 2, viz. III. 15, was born with united labia, but these were separated. (See Bibl. No. 74, p. 219.)

Fig. 154. *Hengge's Case.* Two "sisters," hermaphrodites, in a family of six, the oldest and youngest respectively. I. 1 and 2, parents of II. 1 to II. 3; nothing stated about them. II. 1, of small stature and "peculiar" mentally. II. 3, died of tetanus; the nephews and nieces (number, etc. not given) of II. 3 and II. 4 stated to be normal III. 2, aged 32, "married" to II. 1 for nine years, no children; she presented a female appearance and is stated to have cohabited with her husband, although in all probability "she" is a male; vagina closed, no internal sexual organs to be felt, but in labium majus on right side a testicle and epididymis palpable, on left side similar body in inguinal ring. III. 10, aged 19, unmarried; tall, well-developed figure, of feminine build; has never menstruated; mammae well developed, clitoris not enlarged, vulva appears closed, swelling on each side of labium; by operation these swellings proved to be imperfectly-developed testicles. (See Bibl. No. 75, S. 24.)

Fig. 155. *Lepechin's Case.* Three male hermaphrodites in one family. I. 1 and I. 2, stated to be normal. II. 5, aged 20, a soldier, handsome, tall and well built; while being examined for the army it was discovered that he had female breasts with a pink areola and protuberant nipples; his voice was feminine and his face beardless; his penis was short (two thumbs and three lines long) and his scrotum cleft; there was no urethra, but in its place a groove running along the under surface of the penis; testes were present; from the description this individual was a male with peniscrotal hypospadias. II. 3, aged 22, married for five years but had no children on account of his wife's ill-health; his genitals were exactly like those of II. 5, except that they were more flaccid. II. 1, aged 30, married for 10 years and the father of four normal children, two of which are dead; he likewise was formed like his brothers. II. 6, other children, of both sexes, normal, number not stated. (See Bibl. No. 76, p. 525.)

Fig. 156. *Phillips' Case.* Four cases in a family of nine. II. 3, well-formed but short (5 ft. 7 in.); right inguinal region bulges, especially on coughing, and the abdominal wall is evidently weak; the urethral meatus is prolonged downwards to join the fraenum. II. 5, died of cardiac disease. II. 4, mother of hermaphrodites, aged 32, has no peculiarity. III. 3, a miscarriage at $3\frac{1}{2}$ months, brought on by prolonged constipation. III. 4, ♀, born at full time, but died in $12\frac{1}{2}$ hours with convulsions. III. 5, ♂, aged 10, "normal in every way." III. 6, a "hermaphrodite," christened as a girl; died in 24 hours from wasting; genital organs had the appearance of those of III. 11; the mother was badly frightened by a runaway horse in her third month of pregnancy. III. 7, ♂, aged seven, had the right testicle in the inguinal canal, but on relaxation of the cremasteric muscle it descended into the scrotum. III. 8, a "hermaphrodite," similar to III. 6, died of wasting in 59 days; during the whole of the pregnancy mother had been in great fear that the child would have some deformity of the genitals. III. 9, ♀, alive and well, aged three. III. 10, a "hermaphrodite," christened as a boy, died in 40 days; the scrotum was more closed up than in III. 6 and 8, and the clitoris was much larger. III. 11, a "hermaphrodite," lived 19 days; during the pregnancy the mother was greatly concerned about having children so often with this deformity; medical examination revealed genitals apparently of a female but with a very large clitoris, with a glans half covered by a mobile prepuce; the glans is grooved by a depression in its lower zone which is apparently perforated and corresponds to the meatus urinarius; the depression is continuous with the urogenital sinus; post mortem the organs were found to be entirely female. III. 17, 18, twins, born dead. III. 19, still-born. (See Bibl. No. 77, p. 158.)

Fig. 157. *Brière's Case.* Guyon refers on p. 128 of his thesis to a personal communication of M. Brière with reference to a man aged 34, who was affected with balanic hypospadias; his son, $3\frac{1}{4}$ years old, was affected in like manner; no other details. (See Bibl. No. 78, p. 128.)

Fig. 158. *Wood's Case.* Two hermaphrodites in a family of 12, of which five were male and five female; parents presumably normal. II. 11, aged 13, a "girl"; no appearance of masculinity; examination showed that there was no vagina but testicles on each side; clitoris well developed. II. 12, brought up as a boy, four years old; legs, arms and thighs soft and round; genital organs have the appearance of those of a female—male hypospadian. (See Bibl. No. 79, p. 52.)

Fig. 159. *Kauw-Boerhaave's Case.* This refers to four Siberians, sons of one parent, who had exactly the same deformity of genitals; the description of these individuals was sent by Gmelin to the Imperial Academy, which thought it worth while to send for them. The rest of Boerhaave's paper is taken up with the description of a hermaphrodite sheep. (See Bibl. No. 80, p. 61.)

Fig. 160. *Dixon-Jones' Case.* Three hermaphrodites in a family of eight, all of whom were daughters, or considered to be so. II. 5, aged 21, large in stature and like a handsome woman; mammae and nipples fairly well developed, voice feminine; two swellings in labia proved to be testicles; two other "sisters," II. 6 and 7, were stated to be affected likewise. (See Bibl. No. 81, p. 724.)

SECTION XI α. INSANITY.

BY A. R. URQUHART, M.D., F.R.C.P.E.

The heredity of insanity may be investigated in relation to the incidence of mental disorder or defect in family histories taken at random from the records of the general population; or in relation to families in which at least one case of mental failure is known to have occurred. In the first class the statistics will prove of a wider and more exact interest, in the second class they appeal more exclusively to the psychiatrist. Records of special hospitals or specialised experience naturally vitiate general results, unless they are kept in due proportion to the records of the country at large. Notwithstanding, these separate investigations have a definite value in any scientific discussion of the genesis of insanity, for I have shown elsewhere that persons admitted to James Murray's Royal Asylum, Perth, show 48 per cent. of insane heredity and 81 per cent. of neuropathic heredity. That those percentages are underestimated cannot be doubted, for deliberately false or accidentally false information is

often corrected by patient investigation, while invincible or wilful ignorance baffles all efforts to arrive at the exact truth. Our labours in this field are but partially successful, and our results must be regarded as more or less trustworthy. Generally, the more intelligent the family, the more correct are our observations—assuming that the recorder is willing to spend time and pains in the pursuit of tiresome details.

We have urged that medical science would be greatly advanced by the collection of a thousand family records which might be subjected to a mathematical scrutiny in order to elucidate probabilities in genetic development. That is evidently a long-delayed duty for the British Medical Association. Professor Karl Pearson has shown that the inheritance of physical characteristics can be discerned in a fairly constant ratio, and suggests that mental characteristics will be subject to a similar rule. Montaigne's general observation, "that not only bodily marks, but resemblance of humours of the soul descend from fathers to their children," can be stated in the formulae of arithmetic.

No doubt *all* the vital, anthropological, medical facts should be recorded in these scientific family histories. They should be perfected by observations relative to character and attainment, etc., as well as by the incidents of disease. Such an investigation presents insuperable difficulties. It is almost impossible to find agreement among the children of one generation concerning the colours of eyes among the individuals of the previous generation. Thus, the exact facts are soon lost to memory, and the fond recollections of the living confuse the recorder of their statements. Still, recognising that the perfect family history is hardly to be constructed for more than three or four generations, allowing for family pride or family affection, for the family bias, for the personal equation, many relevant and important facts can be recorded to the advancement of eugenics and that medical science of which it is a new-found and fruitful branch.

It is to be recognized that Genius or Idiocy is comparatively rare. The vast majority of men and women are represented round about the average standard of ability. Bodily characters are similarly grouped—in height the giants or the dwarfs are rare, their numbers are negligible in relation to the great population of the world.

On these general principles I plead for the fullest information about the individuals of families—no matter how common-place. Even if nothing could be learned other than the dates of birth and death there would be a distinct value in the return, while the addition of causes of death would obviously increase that value.

What then is Insanity? Relative to the observations which are desiderated, Insanity may be regarded broadly—as a disease or disorder of the brain affecting the integrity of the mind. It is a rare chance to discriminate between the varieties of mental affection, unless records of special hospitals are available, and it does not appear possible to go far back in point of time in order to indicate these varieties as described to-day. A few points may be cleared up—the mental disease may be congenital or acquired. We might expect some slight indication as to the occurrence of gross pathological significance or obscure causation. We might even record the

symptoms of melancholia, mania or dementia, *i.e.* state whether the individual was depressed, excited, or weak-minded. Melancholia is commonly an initiatory stage, which may pass into mania, which again may pass into dementia. The mental aberration may be cut short at any stage, or it may be of varied and recurrent characteristics[1].

Classification. Acquired	1.	Melancholia	To each of these terms a descriptive word or words might be added:—Paralysis, General Paralysis, Alcoholism, Syphilis, Tubercle, Epilepsy, etc., or signs such as have been devised.
	2.	Mania	
	3.	Dementia	
Congenital	1.	Idiocy	
	2.	Imbecility	

But it would be a narrow view of insanity which would cause the observer to restrict his records to cases of declared failure of the integrity of mind. It is now recognized that the graver neuroses (hysteria, somnambulism and the like), that eccentricity, that a want of mental balance frequently appear among the progenitors of the insane. There is a transformation of neuroses in one generation into obvious insanity in the next. Similarly alcoholism in one generation may issue in insanity in the next, and on the other hand the most inveterate drunkards are often the immediate descendants of insane persons. It is not so much to prove such statements as these that investigations are required, as to place them in well-defined relationship with all the medical circumstances ascertained. It is obvious that records of this wider interest include the incidence of tuberculosis, cancer and all the degenerative diseases which are of importance in eugenics. Formerly, there was an impression that the recognition of insane individuals in a family history was sufficient for scientific purposes; but it has been shown that no sound conclusion can be drawn from that incomplete method. The normal persons are quite as important as the abnormal for a true statistic. For instance: in 28 families observed, fathers and mothers were both insane. Children—alive and sane, 33·10 per cent.; insane, 44·14 per cent.; dead, 22·76 per cent. The evidence of regeneration in this worst possible stock is remarkable.

It has been objected that paralysis, the result of disease of the brain, is negligible in considering the causal factors of mental disorders. That, too, is a partial and misleading opinion. Paralysis is a frequent occurrence in the families of the insane. It results from degenerative lesions which are often of a hereditary nature, but its true incidence among sane or insane stocks can only be determined by research and computation. All insanity is weakness, whether the weakness is the result of obscure toxic conditions, of ordinary chemical poisoning, of gross pathological changes or traumatic injuries of the nervous elements. From the racial point of view, the fine distinctions of specialists are of little weight, whether the mental incapacity is fatuous or furious is a detail of slight importance in sociology, our endeavours are rather devoted to

[1] The Register of First Admissions to James Murray's Royal Asylum is designed to show *inter alia*:—Personal number; name; sex; date; nativity; urban or rural life; education; religion; character; scale of ability; temperament; temper; success; occupation; hygienic conditions; civil state; heredity of character, of bodily disorders, of mental disorders, of neuroses, of alcoholism; pre-existing bodily disorders or injuries; bodily condition at present or injuries; height and weight; congenital; acquired; mental stress or errors; physical stress or errors; period of life; existing personal mental disorder; prognosis; attack—congenital—no. of attack; age—on and duration since first attack—existing attack—admission; onset of attack shortly; seasonal inception, by months; previous certification; age on leaving; disorders on leaving; date on leaving; duration since first attack, of last attack, of residence; destination; cause of death; notes after discharge; final results.

a determination and a prevention of the causes of a failure in bodily health which involves unsoundness of mind. That is a question for practical medicine in the widest sense, but the mere sectary has no grasp of what lies beyond his immediate reach. What is wanted, in brief, are the medical details concerning each individual belonging to families to the number of one thousand at least; certainly not the exclusive records of specialized interest to this or that particular cult engrossed in its own narrow affairs.

Statistical enquiries in lunacy have always been vitiated by failure to attach to the name of every insane person coming under official cognizance one number and one only. Readmission to the national register, transfers in placement, inevitably confuse the returns and prevent the registration of recurring insanity, and consequently any strict computation of the yearly incidence. This is a matter which demands public attention.

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HEREDITARY CASES.

PLATE XVIII. Fig. 161. *Urquhart's Case* (i). Cancer, phthisis, rheumatism, and alcoholism; no consanguinity. I. 1, died in middle life of cholera. I. 2, died of old age. I. 3, died, aged 30, cause unknown. I. 4, died of cancer. II. 2, died of paralysis. II. 3, died of phthisis. II. 5, rheumatic; died, aged 63, of second attack of paralysis, hemiplegia of right side; he was twin with II. 6, and they were so much alike that their children failed to identify them sometimes; he had seven sons and three daughters. II. 6, twin with II. 5, had seven daughters and three sons. II. 8, died of phthisis, alcoholic. II. 10, 12, 14, dead. II. 16, died in infancy. II. 21, successful in Australia. II. 22, had gall stones and appendicitis; had acute rheumatism in 1899 and nine subsequent attacks; died of appendicitis in 1905, aged 67. III. 4, diabetic, kept in check by diet; IV. 1, 2 and 3 are well and parents sober and well-doing. III. 10, goitrous. III. 13, died a month after childbirth, from clot of blood. III. 15, unmarried. III. 16, alcoholic, unmarried; was a soldier. III. 17, patient, aged 31, alcoholic three years; had alcoholic mania to slight dementia and acne rosacea; he was a tobacconist, made an unfortunate marriage, and fell under evil influences; he had repeated delirium tremens. IV. 4 and 5, were twins, they with IV. 6, 7, and 8 died in infancy. IV. 10, eleven children, of whom one died in infancy. IV. 11 and 12, twins. IV. 13—16, one of these died in infancy. IV. 17, dead. IV. 19, died in infancy.

IV. 23, unmarried. IV. 26, died in infancy. IV. 29, one died in infancy. IV. 30, two died in infancy. IV. 31, seven died in infancy. IV. 33, one died in infancy. IV. 35, 36, 37, a pair of twins in this family. IV. 38, 39, 40, a pair of twins in this family. This is pedigree of a Lowland Scots family.

Fig. 162. *Urquhart's Case* (ii). Epilepsy, paralysis, cancer, phthisis, hernia, melancholia; no consanguinity. I. 1, epileptic, fell in a well during a fit and was drowned. I. 2, died aged. I. 3, died, aged 81. I. 4, died of paralysis. II. 1, died of phthisis. II. 4, was operated upon three times for cancer of the lips; died, aged 84, in 1900, very suddenly on getting out of bed in the night, cause unknown; had a hernia, otherwise was a healthy man. II. 5, 6 and 7, no information. II. 8, died of paralysis. II. 9, died insane. II. 12, dull, melancholia at times, a "soft" woman in feeble health, died suddenly, aged 60, in 1879, of cardiac disease. III. 1, a phthisical family. III. 3, born 1836, died 1870, probably of cancer of uterus. III. 5, normal, twice married. III. 7, died in infancy. III. 9, died in childbed, aged 36. III. 10, melancholia; died of an accident to his spine, aged 55; he failed in farming. III. 11, very ill at present, internal tumour. III. 13, normal. III. 14, now convalescent from melancholia; first attack at adolescence, second attack at the climacteric, third at 58. III. 15, normal. III. 16, apparently normal. IV. 1, all abroad. IV. 2 and 3, families reported normal. IV. 4, 5, 6, 7, 9, 10, so far successful. IV. 10, born during mother's second attack. IV. 8, died of hydrocephalus, had a squint. Family Lowland Scottish on paternal side and Highland on maternal.

Fig. 163. *Urquhart's Case* (iii). Phthisis, paralysis and rheumatism; no consanguinity. I. 1, died young. I. 2, died, aged 80. I. 3, died young. I. 4, did not reach old age. I. 5, died, aged 72. II. 1, an active, clear-headed man, died of cardiac disease, aged 83. II. 3, died aged. II. 6, died of paralysis at 84. II. 7, died, aged 92. II. 9, died aged, family scattered. II. 11, had rheumatic arthritis, unmarried. II. 12, eleven in number, mostly emigrated. III. 1, alive. III. 2 to 10, all dead. III. 13, died of phthisis. III. 14, paralytic dementia, unmarried, aged 62. III. 15, single. III. 16, died of phthisis, aged 42; had one normal child. III. 18, married, no descendants. III. 20, died, aged 28, of diphtheria. III. 21, died of throat trouble in childhood. III. 23, scattered. Lowland Scots, lower middle class.

Fig. 164. *Urquhart's Case* (iv). Alcoholism, cancer, rheumatism and insanity; no consanguinity. I. 1, died, aged 93. I. 2, died, aged 73. I. 3, died aged. I. 4, insane, died at home. II. 1, died at 76, in 1903. II. 2, died of abscess in his side, aged 44. II. 3, alive, aged 70. II. 4, rheumatic, died in 1882, aged 54. II. 5, 6, died of liver troubles, aged 60. III. 1, alcoholic in periods for long, aged 51 now. III. 2, formerly alcoholic, apparently reformed[1]. III. 6, twin with III. 7, died of cancer of the bowels in 1907. III. 7, twin with III. 6, has had long-continued chronic mania since 29 years of age, now aged 44. IV. 2, 3, six children, one died of tubercular meningitis. Lowland Scots.

Fig. 165. *Urquhart's Case* (v). Phthisis, rheumatism, alcoholism, neurosis and insanity; no consanguinity. I. 1, had rheumatism and angina pectoris, died, aged 74. I. 2, died, aged 60, in 1881, married, aged 18, in 1840. I. 3, died aged. I. 4, in good health, aged 90. II. 1, died alcoholic, unmarried. II. 2, a literary man of slight ability, died insane; melancholic insanity. II. 3, normal. II. 4, married an artist, has a large family, all well. II. 7, neurotic, not successful. II. 8, not robust. II. 9 and 10, died of phthisis. II. 11, died of fever. III. 2, in Canada, had two children, one dead, one normal. III. 4, infantile paralysis, left leg. III. 5, died in convulsions, aged 15 months. III. 7, patient, stuporose after adolescent mania, is recovering, aged 18. III. 8, died of tuberculosis in infancy. III. 9, died of bronchitis in infancy. III. 10, overdoes her strength, neurotic. Lowland Scots.

Fig. 166. *Urquhart's Case* (vi). Cancer, rheumatism, paralysis, alcoholism; no consanguinity. I. 1, died aged. I. 2, died aged. I. 3, died early, of blood-poisoning. I. 4, died very aged. II. 1, all reported normal. II. 2, died of alcoholism and paralysis, aged 81. II. 3, died of cardiac disease and weak lungs, aged 81. II. 4, reported normal, but rheumatic. III. 2, rheumatic. III. 3, rheumatic. III. 10, died of pneumonia. III. 13, 14 and 15, died in infancy. III. 18, certified insane, on first attack at 41, not recovered. IV. 1, delicate. IV. 6, died in childbirth. IV. 8—11, delicate. V. 1, died at birth. V. 2, alcoholic. Lowland Scots.

PLATE XIX. Fig. 167. *Urquhart's Case* (vii). Phthisis, rheumatism, alcoholism, epilepsy, paralysis and insanity; no consanguinity. I. 1, died of phthisis, aged 64. I. 2, died, aged 84, a very strong woman. I. 3, was alcoholic, died of sudden apoplexy. I. 4, died of cardiac disease, aged 67. II. 1, 2 and 3, alive and well. II. 4, died of phthisis, was never strong. II. 5, has had pleurisy and liver trouble; suffers from cardiac weakness and rheumatic hipjoint; is now in better condition than lately; he and his wife lived a very strenuous life dairy farming and he overworked his family also; they now farm arable land. II. 6, died of phthisis. II. 7. died in infancy. II. 8, died of fright and mania. II. 11, suffered from paralysis and mania, but partially recovered. II. 13 and 14, twins. III. 1, died of tubercular meningitis. III. 2, aged 27, operated on for gumboils. III. 3, phthisical neurotic. III. 4, died of appendicitis, influenza and heart failure; 5 ft. 11 in. in height; was always nervous, died at 19. III. 5,

[1] By error of engraver marked "deformed" instead of "reformed" on plate.

patient, suffering from adolescent mania of sudden development, a finely-made man and very strong, he recovered soon, but his future is doubtful, aged 22. III. 6, suffers from liver trouble and catarrh of stomach. III. 7, died in convulsions in infancy. III. 8, died in infancy. Lowland Scots, west country farming class. All heredity of insanity was at first denied, although it is shown on both sides of the tree.

Fig. 168. *Urquhart's Case* (viii). Insanity, cancer and tubercle; no consanguinity. I. 2, died of cancer of pyloris, aged 68; she was insane three times; her granddaughter states she had 22 children, who mostly died in infancy. II. 1, an alcoholic dement, died, aged 50. II. 3, dead; her daughter, III. 1, is insane. II. 5, was deformed. II. 7, was of great ability, but insane. II. 8, sixteen children, mostly died as infants. II. 9, died of cancer. II. 10 and 11, ten children, four dead. III. 1, insane. III. 2, still-born. III. 3, hydrocephalic, died insane. III. 4, imbecile. III. 5, epileptic. III. 6, died of phthisis, aged 20. III. 7, neurotic somnambulist. III. 8, died of diphtheria, aged 11 months. III. 9, neurotic but of exceptional ability as scientist and teacher. III. 10, was certified insane—aged 15 on first attack of mania, which was followed by moral and impulsive insanity—she recovered and relapsed and remained insane for years. III. 11, died of pulmonary congestion. III. 12, neurotic somnambulist. An English family.

Fig. 169. *Urquhart's Case* (ix). Tropical life and insanity; no consanguinity. I. 1, died of tropical disease in middle life, neurotic. I. 2, died of tropical disease in middle life. I. 3, died in tropics, aged 50. I. 4, died of paralysis in America, aged 70. II. 1, similarly affected to patient III. 1, she was in a London asylum, was discharged and disappeared. II. 2, died early of spinal disease. II. 3, had spinal weakness, was an invalid. II. 4, a sailor and wanderer, was rheumatic, has disappeared. II. 5, aged 52; suffers from sciatica and recurrent melancholia of a mild type. II. 6, died of liver disease. II. 7, widowed, no family. II. 9, aged 43. III. 1, patient, always peculiar, now undecided and difficult, aged 22. III. 2, suffers from overstrain and weak heart, was delirious with influenza, 1891—92, now aged 21, neurotic. III. 3, has had acute rheumatism and jaundice, now aged 18. III. 4, died as an infant of peritonitis. III. 5 and 6, twins, aged 12. III. 7, aged $7\frac{1}{2}$. English on both sides.

Fig. 170. *Urquhart's Case* (x). Eccentricity, rheumatism, exsanguinity. I. 1, able and successful, died aged. I. 3, probably British, died aged. I. 4, probably Hindoo, died young. II. 1, died of malaria, aged 60, eccentric. II. 2, eccentric, a failure, attempted suicide and died in middle life. II. 3, attempted suicide. II. 4, Eurasian, aged 65, rheumatic and neurotic. III. 1, certified insane after first attack, aged 27. III. 2, twin, died at birth. III. 3, twin, insane from adolescence. III. 4, insane from adolescence. III. 5, all young.

Fig. 171. *Urquhart's Case* (xi). Cancer, rheumatism, asthma and insanity; no consanguinity. I. 2, died of asthma, neurotic. I. 3, died aged. I. 4, died very aged. II. 1 to 8, seven sons and a daughter, all sober and long-lived. II. 1, was asthmatic and neurotic; he died of pneumonia, aged 72, in 1877. II. 9, died of cancer, aged 50, in 1866. II. 10 to 13, two lived to 80. III. 2, lives in America, is now aged 75 and well except for rheumatism; she was in Scotland three years ago; a cousin had rheumatic arthritis. III. 3, patient, she suffers from paralytic dementia, cardiac and kidney disease, aged 72. III. 5, died of rheumatic cardiac disease, aged 62. IV. 1, fourteen children, some of them dead, in America. IV. 3, rheumatic fibroid tumour of uterus, had one miscarriage, no family. IV. 4, died of hydrocephalus in infancy. IV. 5, died in teething troubles. IV. 10, died of typhoid in 1906. IV. 12, died of kidney disease after typhoid in 1904. IV. 14, died of fever in infancy. IV. 17, phthisical. IV. 19, died of pertussis as an infant. V. 1, a miscarriage. V. 2, nine of these children, all alive and well. V. 3 to 8, all alive and well. V. 9, died of diphtheria. V. 14, married 10 years, and has no family. Lowland Scots.

Fig. 172. *Hart's Case.* I. 1, mill hand, rheumatic, died, aged 88. I. 2, died, aged 88. II. 1, dead. II. 2, died, aged 70. II. 3, insane, then recovered and afterwards committed suicide. II. 4, died, aged 61. II. 5, insane, died, aged 71. II. 6, a gardener, rheumatic, died of old age. II. 7, died, aged 80. II. 8, married agricultural labourer, suffered from bronchitis, "winter cough" and asthma, died, aged 84. II. 9, died of blood-poisoning following an injury. II. 10, married a gardener, died, aged 82. II. 11, died insane, aged about 75, after a fit occurring at 74, wife of a labourer. II. 12, died, aged 16. II. 13, died between 40 and 50, of bronchitis. II. 14, died between 40 and 50 of blood-poisoning, had varicose veins. II. 15, died, aged 64, of heart disease, was rheumatic. II. 16, died 86, healthy. III. 1, a labourer, died, aged 70; had chronic rheumatism. III. 2, wife of a labourer, healthy. III. 3, died, aged three, of measles. III. 4, labourer, insane, senile melancholia; had congenital dislocation of the hip and double inguinal hernia, aged 57. II. 5, labourer, died, aged 53. III. 6, labourer, suffers from "winter cough." III. 7, dead, coachman. III. 8, wife of painter, died, aged 53, of jaundice; suffered from chronic rheumatism. III. 9, gardener, about 50, had rheumatic fever at 24, followed by complete baldness. III. 10, three born dead or died in infancy, all males, place in family uncertain. III. 11, aged 67, healthy, wife of a coachman. III. 12, aged 61, labourer, insane, has had several attacks of recurrent mania; was alcoholic and suffered from indigestion. III. 13, domestic servant, insane, dead. III. 14, coalminer,

alive, healthy. III. 15, died, aged two, in convulsions. III. 16, wife of boiler smith, aged 56; is obese, has bad heart, and had an attack of nervousness and syncope. III. 17, engine-driver, aged 52, has lumbago. III. 18, gardener, aged 50, blinks his eyes, probably a tic. III. 19, wife of labourer, aged 49. III. 20, wife of wheel-tapper, aged 46. III. 21, died aged 11 months, of bronchitis[1]. III. 22, wife of plasterer, aged 41. III. 23, labourer, aged 35, healthy[2]. III. 24, three premature births, place in family uncertain. III. 25, draper, had bronchitis, died, aged 44, of pulmonary tuberculosis. III. 26, carman, died of pulmonary tuberculosis, aged 38. III. 27, died, aged nine months, of croup. III. 28, wife of stableman, aged 60; obese, suffers from chronic bronchitis. III. 29, wife of platelayer, aged 51, has sciatica and rheumatism. III. 30, epileptic, imbecile. (From Dr Hart's MS. in Laboratory.)

Fig. 173. *Bennett's Case* (i). I. 1 and 2, nothing said. II. 1 and 2, normal. II. 3, insane. III. 1, at special school. III. 2, had fits. III. 3, died from convulsions. III. 4, still-born. III. 5 to 8, strong. (Bibl. No. 75, p. 166.)

Fig. 174. *Bennett's Case* (ii). I. 1, blind. I. 2, alcoholic. I. 3 and 4, nothing said. II. 1, temporarily insane. II. 3, insane. II. 4, defective, temporarily insane. II. 5, almost imbecile. II. 6, feeble-minded. III. 1, tuberculous. III. 2, in reformatory. III. 3, in special school. III. 4 to 9, all more or less mentally and morally defective. III. 10, in special school. (Bibl. No. 75, p. 167.)

Fig. 175. *Bennett's Case* (iii). I. 1 and 2, nothing said. II. 1, tuberculous. II. 2, alcoholic. II. 3, normal. III. 1, epileptic. III. 2, in special school. III. 3, 4 and 5, normal. III. 6 to 9, died in convulsions. III. 10 to 13, dead. (Bibl. No. 75, p. 167.)

Fig. 176. *Macdonald's Case* (i). I. 1 and 2, nothing said. II. 1, imbecile. II. 2, insane and has three insane children, III. 1, 2 and 3. II. 3, 4 and 5, normal. III. 4 to 11, either idiots, imbeciles or insane. III. 12, imbecile, now in asylum. (Bibl. No. 75, p. 493.)

Fig. 177. *Bennett's Case* (iv). I. 1 and 2, nothing said. I. 3, normal. I. 4, insane. II. 1, insane. II. 2, normal. II. 3, dull and alcoholic. II. 4, insane. II. 5, epileptic. III. 1 and 2, mentally feeble. III. 3 to 6, two of these are now in a special school and the other two dull. III. 7 to 10, dead. III. 11, was in special school. III. 12, tuberculous. III. 13, tuberculous. III. 14, imbecile and alcoholic. IV. 1, in special school. IV. 2, tuberculous, dead. IV. 3, cripple. IV. 4 to 7, died young. (Bibl. No. 75, p. 167.)

Fig. 178. *Macdonald's Case* (ii). I. 1, normal. I. 2, insane. II. 1, normal. II. 2, insane. II. 3 to 6, normal. III. 2, insane and epileptic. III. 3, idiot. III. 4, premature dement, had illegitimate child. (Bibl. No. 75, p. 494.)

SECTION VI β (*see* Vol. I. p. 27). HEREDITARY DEAF-MUTISM, *continued.*

(EUGENICS LABORATORY.)

PLATES XX., XXI. Fig. 179. *Graham Bell's Case* (i). Lovejoy family. This family appear to be all descendants of I. 1, who was a farmer in Andover, Massachusetts, before 1640; he had 12 children, seven sons and five daughters. Longevity seems to have been a characteristic of the earlier members of the family, several living to be over 90 years of age. There was a great mortality among the young children of the third generation from "throat distemper," several families being almost wholly swept away. The descendants spread from Andover as a centre and settled in the neighbouring towns and states. They occupy all sorts of positions in society. Some are found among the wealthy and cultivated classes, others occupy very humble stations in life. There are three branches given in the pedigree, the Sebec, Sidney and Concord branches. The members of the Sebec branch seem all to be respectable people. The descendants of the Sidney deaf-mute, on the other hand, have fallen very much in social rank, and the standard of morality among them seems to be low. There are brilliant exceptions, however, to this rule. III. 7, appears to have been alcoholic; church records show that he was charged with being "disguised with drink," "speaking falsely," etc., and reference is made to an earlier public confession of his before the church, in which he pleaded guilty to the "very same sins in form and kind of which he is now accused," and promised reformation; on the strength of that promise, he had been admitted to church membership, now, however, he was formally expelled; he appears to have been an habitual drunkard, shiftless and improvident, and to have died in the most abject poverty. III. 8, seems to have been a most estimable woman, she was blind at the time of her death in her 102nd year. IV. 3, 4, 5, 6 and 14, died young. IV. 7, the ancestor of the Sebec branch, had marked ability, lived to advanced years, and does not appear to have developed any special defect, but cases of insanity have appeared among his descendants, taking a suicidal form. IV. 9, the ancestor of the Sidney branch, a man of energy and marked mental ability, became wealthy; he was a prominent man in his section of the country, a "Judge of Probate" and "Representative in the Provincial Assembly"; convivial in early life, the habit of drinking grew upon

[1] Erroneously marked on pedigree as "prematurely born," instead of "died in infancy."
[2] Should have male instead of female symbol in pedigree.

him; he became blind in middle life and temporarily insane. IV. 10, was one of 25 children by two marriages of her father. IV. 11, had marked ability and developed no defect himself; among his descendants appeared men of great mental calibre, even men of genius; two talented brothers, who have made their mark in the history of their country, were his grandchildren; their father was insane in his latter years. IV. 13, was married. but little is known of her, she may have had a blind son, but this is uncertain. IV. 15, probably died young, but nothing is known of him beyond the fact of his birth. Sidney Branch. V. 14, became blind in middle life, several of his brothers and sisters had failing eyesight as they advanced in years; none of his children were blind, but a grandson, III. 27, lost his sight in mid-life from cataract; this grandson's son, VIII. 31, also became blind from the same cause, and a daughter of this son, IX. 26, has failing eyesight (cause unknown); thus out of seven successive generations of the family, blindness (or a tendency to blindness) has appeared in six, there being a gap in the succession at the fourth generation. V. 19, a deaf-mute, married a hearing woman, not a relative, and had seven children, of whom the fifth, VI. 34, was a deaf-mute; all the deaf-mutes in this branch were born deaf. VI. 21—30, nothing said about them except that several of the family had failing eyesight. VI. 25, had two deaf-mute children, both illegitimate, one a male, VII. 30, living 1888, unmarried; the other a female, VII. 31, who is dead. VI. 34, was a deaf-mute, who married twice; his first wife, a hearing woman, was probably a distant relative of his mother, being of the same name, but this does not certainly appear; by her he had three sons, of whom the eldest was a deaf-mute; by his second wife, a hearing woman and not a relative, he had seven children, three of them deaf-mutes; one of the hearing children, VII. 77, was born deaf in one ear, but heard well with the other. VII. 28, was illegitimate. VII. 31, the illegitimate deaf-mute daughter of VI. 25, had eyes of different colours, one eye dark and the other blue; one of her illegitimate children, VIII. 36, was born blind or had undeveloped eyes; some state that there were no eyeballs in the sockets, and that it was deaf-mute, but it died in infancy, too young to have the deaf-mutism ascertained. VII. 59, a deaf-mute, married a hearing unrelated woman and had eight children, of whom three, VIII. 53, 58 and 61, were deaf-mutes; so far there had been no inter-marriages with deaf-mutes or with near relatives; the deafness had persisted in the family for four successive generations, in spite of the introduction of fresh blood at each marriage. VII. 63, was a deaf-mute. VII. 75, a deaf-mute, married VII. 86, a congenital deaf-mute, who had a brother, VII. 87, and several other relatives deaf and dumb; there were also several consanguineous marriages and cases of idiocy in his family; they have had eight children, three of them, VIII. 73, 76 and 77, deaf-mutes; in 1888 these children were still very young. VII. 75, had a child, VIII. 72, before her marriage with VII. 86, but no information is given either about father or legitimacy of this child. VII. 77, who was born deaf in one ear, married a congenital deaf-mute, VII. 89, who had two brothers and a sister, VII. 90, 91 and 92, all deaf-mutes; they have had three children, one, VIII. 81, a deaf-mute; the youngest was a baby-in-arms in 1888. VIII. 53, a deaf-mute, married a congenital deaf-mute, VIII. 89, who had a deaf-mute brother and sister, VIII. 90 and 91, and the sister, VIII. 91, has a deaf-mute son, IX. 42; of the five children of this marriage, two, IX. 32 and 33, are deaf-mutes; the children were very young in 1888, the youngest a baby-in-arms. VIII. 58, a deaf-mute, married a hearing unrelated man, VIII. 59, and had three hearing children, very young in 1888. We now turn to the Sebec Branch. No trace of deafness could be found in this branch till the marriage of VI. 11. VI. 11, married VI. 10, who traced her descent, by two lines of ancestors, from persons who came from Chilmark, Martha's Vineyard, a township in Massachusetts, remarkable for the number of deaf-mutes who have been born in it; in 1880, one person in every 25 of the population was a deaf-mute, and, according to the Hon. F. B. Sanborn, all these deaf-mutes were descended from the deaf-mute son of a missionary who settled there in 1720 (vide *Lancet*, p. 221, 1877); the deaf tendency in VI. 10's family is well-marked; she had a sister, VI. 6, who had five deaf-mute children and another, VI. 8, who had deaf grandchildren; she had one cousin, VI. 1, who had seven deaf-mute children, another, VI. 2, who had two, and a third, VI. 3, who had three; VI. 10 had two deaf-mute children, VII. 14 and 17, one of whom, VII. 17, died in infancy. VII. 14, a deaf-mute, married VII. 15, a hearing woman, not a relative, who was slightly insane; they had four children, three of them deaf-mutes, VIII. 19, 20 and 23. VIII. 19 and 20, were unmarried in 1888. VIII. 21, normal, married and had one son, IX. 21; this son married and had one child, XI. 1, a baby-in-arms in 1888. VIII. 23, a deaf-mute, who died of tuberculosis, married a deaf-mute, VIII. 24, but had no children. Lastly we have the Concord Branch. Deaf-mutes first occur in Generation VII. VII. 114, 115, 120 and 121 were deaf-mutes, the ancestry on the mother's side is unknown. VII. 114 and 115, died young. VII. 120 and 121, left no descendants. VII. 116, the only member of his family who seems to have married was at one time insane; he married his first cousin and had four children, the eldest, VIII. 121, a deaf-mute, who married a deaf-mute, VIII. 120, but had no children and died of tuberculosis. (Bibl. *a*, p. 28, No. 9, p. 359.)

Fig. 180. *Graham Bell's Case* (ii). Brown family. I. 1, was one of the early pioneers of New Hampshire. II. 8, his deaf-mute son, married a hearing woman, II. 9, and had a deaf-mute son and daughter, III. 21 and 22. III. 21, was said to have had an aunt, II. 10, and two cousins, III. 24 and 25, deaf and dumb, but this branch of the family has not been certainly identified. III. 21, married a deaf-mute, III. 20, and had two children, IV. 16, a deaf-mute, and IV. 18, a hearing daughter, who died young.

IV. 16, married IV. 17, a hearing woman, and there is no information with regard to his descendants. III. 22, deaf-mute, married a hearing man, III. 23, and had three sons; the eldest, IV. 19, was born deaf-mute and married a deaf-mute, IV. 20, and his three children, V. 6, 7 and 8, were deaf-mutes. IV. 21, the second son, was born deaf in one ear and lost the hearing of the other in childhood from measles, the third son, IV. 22, could hear. IV. 21, married a deaf-mute, IV. 24, by whom he had five children, all of whom could hear at birth, but two of them, V. 10 and 11, lost their hearing very early in life. V. 12, retained her hearing, and V. 13 and 14 died young. V. 10, married a deaf-mute, V. 9; it will thus be seen that three families of deaf-mutes have sprung from II. 8; in two of these deafness descended to the fourth generation, in the other it has descended to the third generation, beyond which the family could not be traced. IV. 24, had a deaf-mute brother, IV. 25, who married a deaf-mute, IV. 26, and had a hearing child, V. 15. IV. 27, a deaf-mute, twin brother of IV. 26, married IV. 29, a deaf-mute, and had a deaf-mute son, V. 16. IV. 30, a deaf-mute sister of IV. 29, married IV. 32, the deaf-mute son of deaf-mute parents, III. 33 and 34, and had a deaf-mute son, V. 17; he also had a deaf-mute cousin, and his mother, III. 34, had a deaf-mute cousin; on the other side of the family III. 20 had a hearing brother, III. 18, who had two deaf-mute children, IV. 14 and 15. IV. 14, married a deaf-mute, IV. 12, and had a deaf-mute daughter, V. 4. III. 20, had also a deaf-mute sister, III. 17, who married a hearing man, III. 15, who had five deaf-mute brothers and sisters, III. 10 to 14. IV. 12, had a deaf-mute mother, III. 8, and two deaf-mute maternal uncles, III. 4 and 6, both of whom married deaf-mutes, III. 5 and 7; she had also three deaf-mute brothers, IV. 6, 8 and 10, and a deaf-mute sister, IV. 11. IV. 6, married a deaf-mute, IV. 7, and had a deaf-mute daughter, V. 2. IV. 8, married a hearing woman, IV. 9, but there is no information with regard to their descendants. IV. 11, married a deaf-mute, IV. 4, and V. 1, a deaf-mute, is probably their son. IV. 2, a deaf-mute brother of IV. 4, married a deaf-mute, IV. 3, but it is not said whether they had descendants or not. (Bibl. *a*, p. 28, No. 8, pp. 28 and 29.)

Fig. 181. *Williams' Case* (i). II. 1 and 2, were first cousins and had eleven children, nine hearing and two deaf-mutes, III. 1 and 2. III. 1, was a non-congenital deaf-mute, deafness occurring at three years of age. III. 2, was a congenital deaf-mute. II. 3, also married a first cousin, II. 4, and had two deaf-mute children, III. 4 and 5. (Bibl. *a*, p. 28, No. 9, p. 308.)

Fig. 182. *Kerr Love's Case.* The Ayrshire Family. IV. 2, married IV. 1 of the same name about 1800; identity of name suggests consanguinity, but no evidence of a more positive kind has been got; seven deaf-mutes are known to have been born into this branch of the family, and in the present generation the tendency continues to express itself; several cases of intermarriage between cousins have taken place amongst the members of this branch of the family, but without any increase in the tendency to deafness; Dr Kerr Love states that the details of this part of the family were collected from old friends and acquaintances of the family, and have been corroborated from various sources. IV. 2, is second cousin to IV. 5, 6 and 9, who all occupied farms within a radius of 15 miles of the town of Galston, near which III. 2 lived; the facts about the descendants of these three farmers have been collected from the oldest representatives of the family still living in the county; there are fifteen deaf-mutes in this branch. IV. 13, a deaf-mute, was born in New York; his father had left Galston before his birth, and IV. 13 returned to Scotland in early life; absolute proof that IV. 13 was related to IV. 2, 5, 6 and 9 has been got, but the exact relationship has not been made out; there are 19 deaf mutes in this branch, the details of which have been collected by Mr Henderson, of the Glasgow Deaf and Dumb Mission from the family Bibles—the old Scotch register—of surviving members, and by Miss Cunningham and Mr Large, of Donaldson's Hospital; the facts about this branch have been corroborated by IV. 13's daughter, aged 84; every family sent colonists to Australia and Canada, and two at least of the five progenitors, IV. 9 and IV. 13, have deaf-mute descendants in our colonies; the descendants of IV. 9 are represented by three families who have deaf-mute members in Australia; into one of these three families two deaf-mutes have been born; into the other two, deaf-mutes have also been born but the number is unknown. IV. 13's family is similarly scattered. (Bibl. *a*, p. 28, No. 14, pp. 87 and 88.)

Fig. 183. *Connor's Case* (i). II. 5, one of five congenital deaf-mutes, married II. 7, one of three congenital deaf-mutes, and had six hearing children, III. 1—6; his sister, II. 6, first married II. 11, one of four congenital deaf-mutes, and had four hearing children, III. 7—10; she married, secondly, II. 16, one of four congenital deaf-mutes, and had two hearing children, III. 11 and 12. II. 17, brother of II. 16, married first II. 21, a deaf-mute, widow of II. 29, and had two hearing children, III. 13 and 14; he married, secondly, II. 31, one of two congenital deaf-mute sisters, who had also a deaf-mute cousin, and had one deaf-mute child, III. 23. II. 21 had by her first husband, II. 29, also a deaf-mute, two children, III. 21, a deaf-mute, and III. 22, hearing. II. 18, another deaf-mute, brother of II. 16 and 17, married II. 33, a deaf-mute, and had three deaf-mute children, III. 24, 25 and 26. III. 21, a congenital deaf-mute, married a congenital deaf-mute, III. 29, one of two deaf-mutes, and had two children, IV. 3, a deaf-mute, and IV. 4 hearing. III. 22, hearing, married III. 28, a non-congenital deaf-mute, and had two children, one deaf-mute, IV. 1, and one hearing, IV. 2. (Bibl. *a*, p. 28, No. 9, p. 312.)

Fig. 184. *Mattieson's Case.* I. 3 and 4, both hearing persons, not related in the slightest degree; no deaf-mutes have been known in either of their families, but they were parents of five deaf-mutes in a family of eight. II. 3, a deaf-mute, married II. 2; she and her sister had both lost their hearing at an early age by sickness; they had three children, III. 1, 2 and 3. III. 1 died in infancy, III. 2 at 13 months old, and III. 3, now $2\frac{1}{2}$ years of age, is apparently deaf and dumb. II. 4, a deaf-mute, married II. 5, also deaf, and had six hearing children, III. 4. II. 6, a deaf-mute, married II. 7, also deaf, and had five children, III. 5 and 6, four of them living; the children's faculties are unimpaired. II. 8, a deaf-mute, married II. 9, also deaf, and had one child III. 7, who can hear and speak. II. 10, hearing, married II. 11, hearing, and had two hearing children, III. 8 and 9. II. 12, hearing, married II. 13, a semi-mute, and had one hearing child, III. 10. II. 14 and II. 15, were single. (Bibl. *a*, p. 28, No. 9, p. 319.)

Fig. 185. *Royal Commission*, 1889. I. 1, deaf-mute. II. 2, normal, his five children, four sons and a daughter, all deaf and dumb, III. 2—6. (Bibl. *a*, p. 28, No. 9, p. 50.)

Fig. 186. *Royal Commission*, 1889. I. 1 and 2, were first cousins and had six deaf-mute daughters, II. 2—7. (Bibl. *a*, p. 28, No. 9, p. 50.)

Fig. 187. *Williams' Case* (ii). I. 1 and 2, were second cousins, and had nine deaf-mute children; one of these, II. 10, married a deaf-mute, II. 11, and had a deaf-mute daughter, III. 1, and three hearing children. (Bibl. *a*, p. 28, No. 9, p. 308.)

Fig. 188. *Connor's Case* (ii). III. 2, had a deaf-mute grandmother, I. 2, and two deaf-mute brothers, III. 3 and 4; he married III. 5, one of three deaf-mutes, and had seven children, of whom five were deaf-mutes, IV. 1 to 5, one, IV. 6, very hard of hearing, and another, IV. 7, who heard perfectly; his deaf-mute brother, III. 3, married III. 13, one of three congenital deaf-mutes, and had five children, IV. 16—20, who all heard perfectly. III. 4, another deaf-mute brother, married III. 17, a non-genital deaf-mute, and had one normal child, IV. 21. III. 6, a congenital deaf-mute, brother of III. 5, married III. 9, a congenital deaf-mute, whose parents, II. 6 and 7, were also congenital deaf-mutes; they had three children, IV. 8 and 9, deaf-mutes, and IV. 10, who was very hard of hearing. III. 7, a deaf-mute sister of III. 5 and 6, married III. 12, a deaf-mute, and had five hearing children, IV. 11—15; it is not stated whether III. 12 was a congenital deaf-mute or not. (Bibl. *a*, p. 28, No. 9, p. 312.)

Fig. 189. *Stephenson's Case.* III. 2, married her mother's younger brother, II. 3; five children were born, of whom IV. 1—3 are deaf-mutes, and one other, IV. 5, is still an infant whose condition is uncertain. (Bibl. *a*, p. 28, No. 14, p. 120.)

Fig. 190*a*. *Connor's Case* (iii). II. 2, a congenital deaf-mute, married II. 3, one of two congenital deaf-mutes, and had five children, III. 2—6, all deaf-mutes; his son, III. 2, married a hearing woman, III. 1, and had three children, IV. 1—3, all hearing perfectly; a danghter, III. 6, married III. 7, one of three congenital deaf-mutes, and had two children, IV. 4 a deaf-mute and IV. 5 hearing. (Bibl. *a*, p. 28, No. 9, p. 312.)

Fig. 190 *b*. *Sir A. Mitchell's Case.* I. 1, a deaf-mute, married I. 2, whose hearing was perfect, and had two children by her, II. 1, a deaf-mute son who died childless, and II. 2, a hearing daughter who married II. 3, a hearing man, and gave birth to two deaf-mute danghters, III. 2 and 3, and a hearing son, III. 4. III. 2, married a deaf-mute, III. 1, and had a hearing son, IV. 1. III. 4, married a hearing woman, III. 5, and had a deaf-mute son, IV. 2. (Bibl. *a*, p. 28, No. 5, p. 164. This pedigree of 1863 is identical with that given almost verbatim by Principal Charles Kerney of the Evans Deaf-mute School, Indiana, for a deaf-mute in Iowa in the *Report* of 1889: see Bibl. *a*, p. 28, No. 9, p. 317.)

SECTION VII β. ABILITY. (*See* Vol. I. p. 30.)

(EUGENICS LABORATORY.)

In this further contribution to the section on ability, the first pedigree (Fig. 192, A—G) must be looked upon as an attempt to illustrate in a pedigree-form the linked qualities which characterise the members of certain related stocks. It is a task not without difficulty from more than one standpoint, yet the result will probably be found to have some interest. The type of the family is peculiarly English, and in England, to our national profit, not so uncommon. It usually starts from a hardy north-country yeoman stock, accustomed for generations to outdoor life, healthy, full

of animal instincts, sound common sense, and a shrewdness which carries it rapidly forward in life. To a large extent without what from one side may be termed prejudice, from another side traditional culture, it forces its way forward and upward against existing social barriers and in doing so develops a sturdy, well-marked independence of thought and character. Contemporary opinion terms its members radical in politics and free-thinking in religion, or again liberal in religious and political thought according to its own attitude. The vigorous vitality of the original stock is persistent in a love of good living, wealthy surroundings, and sometimes of excessive pleasure. Occasional members of the older generations may have failed in life from this tendency. But this superabundant vitality, which may exhibit itself in such channels, has been largely the source of great success in commerce and politics. Those who know well the manufacturing districts of northern England recognise how much of the trade prosperity of the country and of its active political and religious life has been created by stocks of this kind. An apparently quiescent, but hardy yeoman stock, begins thrusting forth active members, who advance through great trade activity to responsible political and social position. This type of family forms such an essential and valuable factor of our national life, that an attempt has been made to trace in the different branches such a linked group. The characters, which have been estimated by one of the members of the group itself, to whose care and labour in this matter we are much indebted, are precisely those referred to above; namely: marked ability, chiefly but not solely shewn in building up and carrying on great commercial enterprises, occasionally exhibited in literary or scientific work; liberalism in political activity, and independence in religious thought; with frequent, but usually harmless manifestations of the older vigorous, pleasure-loving vitality. In judging the hereditary factor in the pedigree the ages of the younger generations must be taken into account, the age for achievement is not yet reached in many cases. Tuberculosis and some other anomalies have been indicated in the pedigree.

This account must be looked upon as tentative, but it may be not without suggestion to those wishing to deal with the descent of what they recognise as family characteristics, but find hard to analyse and depict.

The second pedigree, Fig. 193, represents a family far easier to deal with,—it shows in linked stocks a very large representation of marked ability in legal, executive or commercial spheres. The intermarriage and preservation of such stocks may be looked upon as essential to the due provision of an adequate supply of national leaders and administrators in these fields.

PLATES XXII.—XXIV. *Commercial Ability and Liberal Thought.* We start with branch D, Fig. 192. I. 1—6, the ancestors of these families had been settled for many generations in a wild hilly district in the north of England; they were yeomen or tenant farmers, served as "high constables," churchwardens, etc., and from their wills appear to have been men of moderate substance, more especially the families of I. 5 and 6. With the expansion of trade in the early 18th century, they established prosperous woollen mills and afterwards cotton mills along the streams of their native valleys, built houses and churches and became leaders of local activities; there were constant intermarriages between all the families residing in the district; the early part of the pedigree down to II. 10 and 11 is pieced together from wills and registers and does not claim to be exhaustive; the children given are taken from the wills of one or other parent or grandparent; a few have been verified from registers but the surnames are so few in number, and the Christian names also so limited, that it is very difficult to assign correct dates for births, deaths

and marriages; from II. 10 and 11 onwards, descriptions are taken from private and public records, portraits and personal knowledge. I. 3, survived marriage 36 years. I. 4, survived marriage 41 years. I. 5, a man of influence and character, established a prosperous business. II. 4, a man much in request as trustee and executor. II. 9, presumably a first wife, of same character as II. 11, had three children. II. 10, died aged 56, a very successful man. II. 11, died, aet. c. 90, remarkably capable woman, shared in all the business interests of her sons, well educated. II. 13, journey-man carpenter from another district, strong, short, fair, ruddy complexioned, handsome, and full of vitality, afterwards drank heavily; there is said to have been insanity in his family; one of the daughters of II. 4 (included in III. 10 and 12) ran away with him; order of children unknown. III. 1, 3, 5, 16, 17 were all in the same business, having branches in England, S. America, and Germany; they were extremely successful and pushing and may all have had great, they certainly had noteworthy capacity. III. 17, who seems to have been the leader in all foreign and colonial developments, certainly had marked ability. III. 3, died aet. 62. III. 5, died, aet. 76. III. 15, one daughter married and died aet. 80 of old age. III. 16, died, aet. 55. III. 17, a most active man, his early manhood was most adventurous, going abroad during the Napoleonic Wars to establish agencies, swam several miles from shipwreck to shore, had many adventures up the Amazon and in Brazil; a most energetic, shrewd, clever man of business, no scholar, sanguine, cheerful, benevolent, unconventional; M.P. for several years, follower and supporter of Cobden, enthusiastic teetotaller, very healthy, deaf in old age, strong and well-knit frame, died, aged 86, of chill caught at wood-cutting on a winter's day. III. 18, suffered many privations in childhood, often went barefoot and hungry, became nursery governess, great personal charm, no special ability or intelligence; died aet. 32 of consumption, which developed soon after birth of youngest child. III. 20, a carpenter, short, fair, stout, blue eyes, ingenious, peculiar, died in middle age. III. 21, a soldier, strong man, died in middle age. III. 23, a carpenter, rather weak-minded, melancholy, committed suicide. III. 25, wife of farmer, committed suicide, aet. c. 60. III. 25 to 27, hardly known, do not seem to have risen in social scale. IV. 2, a clergyman, lived to a considerable age, married first a cousin, second a young woman in later life. IV. 3 and 4, remained single. IV. 5, died unmarried, aet. c. 30. IV. 10, resembled her mother in person and her father in character; considerable energy which she always overtaxed; great intellectual ability, constant student of classics, mathematics and politics; highly nervous temperament, strong sense of duty but no very wide sympathies or artistic aptitudes; cataract set in about 50 years of age, but did not greatly develop; died, aet. c. 61, of pleuro-pneumonia following chill. IV. 11, man of moderate abilities, interested in money-making, shooting and farming; rather unsuccessful, fond of good living; as young man had pleurisy which destroyed one lung; died from diabetes and fatty degeneration of the heart, aet. 57. IV. 12, died, aet. c. 70. IV. 13, ill regulated life, average abilities, suffered from bronchitis, died, aet. c. 70. IV. 15, remarkable bodily strength and considerable mental powers, said to have been eccentric and uncertain; emigrated and was killed in driving accident, aet. 46. IV. 16, an energetic very capable and very good business woman. IV. 17, very delicate child, probably consumptive, died, aet. six, of inflammation of the lungs. IV. 18, adventurous and hardy, travelled incessantly, first-rate health and strength, in younger years clever original man, fond of all out-door sports and of pleasure, liable to attacks of inflammation of the lungs, died, aet. c. 70, of general decay. IV. 20 to 24, of mill hand status, do not seem to have risen in social scale. V. 2, VI. 1—5, and VII. 1, 2, well-to-do family. V. 3, mentally defective. V. 4, born paralysed, almost inarticulate, of considerable natural ability, a cripple. V. 5, 7, 9, 11, 13, 15, 17, 19, 21, 25, family of moderate ability and energy, all abounding in vitality. V. 23, eloped, then got divorced. V. 34 and 36, strong successful business men, any amount of go. V. 38, healthy, very capable woman. V. 40, 41, 42, all born hereditarily diseased, died as infants, aet. 14 months, two months, and a few days. V. 43, feeble-minded, aet. 38, alive. V. 44, invalid, died, aet. 15. V. 46, 47, 48, 50, 51, normal women, fond of outdoor life, good practical managers. V. 51 and 52, recently married. VI. 8 and 9, recently married. VI. 13, 17, steady hard-working young fellows, aet. c. 22. VI. 15, married when only 18. VI. 26, 27, 28, 29, healthy sturdy children, aet. 15 to 2 years. VI. 30 and 31, aet. 12 and 9.

PLATES XXII., XXIII. Fig. 192, B. I. 7, successful postmaster and innkeeper in small town. I. 8, a barber of a family of yeomen and small traders resident in his county for at least 150 years, probably longer; said to be of Flemish descent and immigrants during the Spanish occupation of the Netherlands. I. 9, daughter of a weaver. II. 15, successful postmaster and innkeeper, had children, some of his descendants are said to still live in the town. II. 16, a capable woman and excellent mother, helped in shop and farm; much respected by her children, died, aet. c. 65. II. 17, barber, chirurgeon, farmer, linen-draper, after being journeyman in London; large progressive farmer, made considerable fortune; a thoughtful radical, married, aet. 36, died of old age and heart failure, aet. 74. II. 18, other children, probably not many, certainly one son and probably a daughter, married to a London chemist, who had children. III. 28, in business, bankrupt, able but hard liver; enlisted, went to America, died of yellow fever at Havanah, aet. 30. III. 29, a commercial traveller, able active man, strong radical politician, took to drink, became a member of the Society of Friends, tried unsuccessfully to reform himself; founded a great business, died, aet. c. 50, death accelerated by intemperance. III. 31, died, aet. c. 12 months. III. 32,

a clever and strong-minded woman, died, aet. 77, of old age, married III. 33, a country gentleman farming small estate, competent and much-respected man. III. 34, capable woman, died of old age, aet. 73. III. 35, active and clever till middle age, scorbutic, became imbecile in old age, died, aet. 71. III. 36, of a manufacturing family, died two years after marriage. III. 37, a remarkably vigorous strong-minded man, who succeeded in everything he undertook; a man of strong passionate nature, keen radical and social reformer; with his brothers, III. 29 and 39, built up a very prosperous business, M.P., knighted, died, aet. 71, of old age. III. 38, came of mercantile family, placid and good-tempered. III. 39, worked on father's farm, became commercial clerk, then Manchester warehouseman; built up large business jointly with his brothers, III. 29 and 37; keenly interested in all political, social and religious reforms, philanthropic and public-spirited M.P.; became somewhat infirm in middle life, fond of conviviality, died, aet. 64, of tumour of the brain. III. 40, commercial traveller and small tradesman. III. 41, active, clever woman, died, aet. 61. IV. 25, merchant and active business man, great railway interests, chairman of two well-known railway companies, very strong physically, good manager of men, broad churchman, follower of Peel in politics, considerable originating powers, an adroit diplomatist in negotiations, no artistic aptitudes; went bald and gray at 30; fond of good living; gouty symptoms, at one time tending to glandular swellings; had successive paralytic strokes, starting after severe blow on head, almost unconscious for last 12 months of life, invalid for last six or seven; died, aet. c. 74, of paralysis of heart and breathing organs. IV. 26, troubled at one time with bronchitis, alive, aet. c. 90. IV. 27, civil servant, member of well-known literary and liberal family, died c. 70. IV. 28, clever woman, of many accomplishments and much energy, hard upbringing, always delicate, died, aet. 25, of pulmonary tuberculosis. IV. 30, mental powers small, always delicate and threatened with lungs, died, aet. 42, of pleuro-pneumonia, occasioned by chill while bathing. IV. 31, still-born or died in very early infancy. IV. 32, delicate, ailing child, died, aet. about two years. IV. 33, four healthy offspring, one lived to be over 80. IV. 34, lived to be over 88, enjoyed good health; went blind about 76. IV. 35, died, aet. 62, married, s.p. IV. 36, clever, married a brother of IV. 35's husband, s.p. IV. 37, resembled his father (III. 37), M.P., died, aet. c. 40, unmarried. IV. 38, fond of good living, radical politician, M.P., lived to about 80; married twice, second wife when nearly 72; he was a free trader, abolitionist; died of apoplexy, height 6 ft. 4 in., weighed 17 stone[1], very energetic, great traveller. IV. 39, daughter of a prosperous merchant, strong liberal and nonconformist traditions, died, aged 60. V. 53, went to India, died, aet. 27, of abscess on the liver. V. 54, liberal, journalist, unmarried, died, aet. c. 60. V. 55, energetic, good social qualities, well educated. V. 56, remarkably able energetic man, established and conducted extensive sociological investigations, F.R.S., P.C. V. 57, clever naval officer, took to drink, died in New York of delirium tremens, aet. c. 30. V. 60, some healthy children. V. 63, died, aet. 35, became a Roman Catholic, unmarried. V. 64, became a Roman Catholic, unmarried. V. 65, died, aet. 36, never fully recovered from severe attack of rheumatic fever. V. 65*a*, delicate puny child, grew up strong, but developed tuberculosis after two attacks of pleurisy, aet. c. 45, said to have been cured by inoculation; industrious, liberal in politics and religion, aet. 57. V. 66, industrious, good business aptitude; all the descendants of III. 37 are said to have suffered from their livers. VI. 45, 46, said to show signs of resembling their ancestors in this respect. VI. 32, 34, 36, 38, 40, 41, all have considerable social, literary and conversational ability, ages 35 downwards. VI. 37, very musical, aged c. 29. VI. 39, member of distinguished literary family. VI. 42, died in childhood. VI. 45, 46, under eight years of age. VII. 4—19, under 15 years.

PLATES XXII., XXIII. Fig. 192, A. II. 19, cousin to II. 20. II. 20, member of old Lancashire yeoman family, managing clerk and foreman to large cotton spinning mill. II. 21, housekeeper to II. 20; short, stout, of gipsy, possibly of Jewish, extraction, erratic. III. 44, a daughter, said to have been queer. III. 46, in agency business, died c. 60, of general decay; peculiar and violently passionate. III. 48, in agency business, died, aet. c. 60, peculiar. III. 49, believed to have died in childhood. III. 50, clerk, died, aet. c. 20, of consumption. III. 51, died, aet. c. 23, of consumption. III. 52, merchant in Lisbon, died, aet. c. 86, of old age. III. 54, clever and unconventional, musical, partly insane for some years after birth of youngest child, attributed to "tubercles of the brain," recovered in later life; died, aet. 82, of old age and degeneration of heart substance. III. 55, died as a child. IV. 47, alive, considerably over 80. IV. 58, twice married, no children. IV. 60 and 61, both married, large families. IV. 62, three unmarried daughters. IV. 63, five other children. V. 70 or 71, difficult as a young man. V. 72, eccentric; generations IV. and V. of this branch are largely in the church, or married to clergymen, some are engaged in mercantile pursuits, some in the colonies; the majority call for no special comment here.

PLATES XXII., XXIII. Fig. 192, C. V. 1, remarkably clever masculine woman, good scholar, a hard reader, active politician, a wonderful manager, unconventional, great physical strength, nervous, inclined to sleeplessness, became victim of drug habit for about 12 years before death; this accentuated excitability and hastened death; gouty and rheumatic tendency, fond of pleasures of table; died in 61st year, heart failure, complicated with clots, vein trouble, and general collapse. V. 2, hard-working, steady business man, without much initiative; successful party leader; did good service in Town Council, on bench, etc.;

[1] There is a family history of corpulence, which cannot be followed on this occasion.

health failed physically and mentally to a certain degree after about 65; suffered from hernia, stomachic weakness; died of senile decay, congestion of arteries of brain, and thickening of internal tissues and paralysis. V. 3, good abilities of a practical nature, good manager of men, keen politician, interested in all social reforms, married c. 36, aet. now c. 62. V. 4, political and economic authority, high wrangler, M.P. for many years, P.C., married, aet. c. 50, now aet. c. 76. V. 5, exceptional energy and organizing power, quick-witted, sanguine, restless, most active member of county education authority, and an authority on technical education, used to hunt, aet. c. 60. V. 6, English manufacturing squire, keen on hunting and country pursuits, active in county administration, aet. c. 65. V. 7 and 9; these two brothers belong to a large family of merchant princes of German origin, crossed with Portuguese blood, all excellent men of business, fond of music, collectors of pictures, books and bric-a-brac. V. 9, admirable business man. V. 10, good mental powers and considerable literary gifts, liberal in religious thought, nervous but self-controlled, good manager; hair and eyebrows went gray about 40, aet. c. 58. V. 11, mental powers very unequal, at times total eclipse of memory and subject to fits of great depression, melancholy religious mania; considerable artistic talent; great physical energy; greatly resembled her grandmother, III. 54, in features; committed suicide, aet. c. 55. V. 12, 14, members of a large middle-class professional family with legal traditions, father K.C.; there is a large family of brothers and sisters with very large families. V. 12, is a different type from other members of his family, an eminent surgeon and successful financier. V. 14, able and distinguished lawyer, good party politician on strict tory lines, for some time M.P. V. 13, stout, good-natured, of foreign origin. V. 15, imaginative talented woman, much nervous but not much physical energy, enthusiastic, sanguine, tendency to overwork; highly "sensitive" in the psychical sense, saw visions, skilled at automatic writing, unequal in spirits, most loyal friend, considerable love of drawing and music; in constitution greatly resembled her mother; at 16 severe inflammation of lungs and liver produced condition which threatened lung troubles; two attacks of puerperal fever; died suddenly, aet. 41; suffered from insomnia. V. 16, good practical abilities, excellent manager; considerable interest in political, social and religious topics, advanced thinker; physically strong and fond of exercise; requires much sleep; industrious, aet. 54. V. 17, member of family producing excellent civil servants and clergy; public school scholar, double first at Oxford; M.P. for about 20 years, P.C., chairman of a County Council; hardworking, conscientious; aet. 54. V. 18, considerable physical energy, excellent health; able original woman; great personal charm; great intellectual energy and nervous power; fond of society and hard work; aet. 52. V. 19, very able man, of middle-class family; excellent administrator and organiser. V. 20, still-born son, possibly should come between V. 16 and V. 18. V. 21, died of scarlet fever, aet. $2\frac{1}{4}$, a fine, healthy, intelligent child. V. 22, clever, but not very successful man, literary, musical; nervous; poor family history; after several years' illness, died of locomotor ataxy. V. 23, in bad health till recently. V. 24, organises games and sports, secretary to golf clubs; practical and competent. VI. 1, public school scholar, excellent man of business, liberal M.P., fond of society, aet. 40. VI. 3, competent, practical, keen intellectual interests, fond of out-of-door pursuits; considerable nervous energy, requires much sleep, good health, aet. 38. VI. 4, good intellectual abilities, F.R.S., fond of outdoor pursuits, shooting, riding, etc., aet. 41. VI. 5, good man of business, fond of outdoor and social life, had severe typhoid fever, aet. 36. VI. 7, good practical abilities, rather excitable nature, considerable physical and nervous energy, aet. 34. VI. 8, good business man, keen on outdoor life, fond of society, aet. 32. VI. 9, emigrated to New Zealand for the sake of an outdoor life and manages a big station with success; had typhoid, aet. c. 19, energetic in outdoor pursuits, backward at school, delicate like VI. 8 in childhood, aet. 30. VI. 12, good business aptitudes and considerable intellectual interests. VI. 15, keen naturalist and excellent artist, fond of travelling and camping out; died, aet. c. 23, of appendicitis. VI. 16, interested in social reform, somewhat delicate, considerable organising power, aet. c. 32. VI. 18, resembles VI. 15, in army, keen sportsman, often away on big game expeditions; seems to be showing considerable organising ability. VI. 19 and 20, are same as VI. 37 and 36 in B above. VI. 21, leads a life away from home in London and colonies, a great collector, aet. c. 28. VI. 24, showing considerable ability in many ways, aet. 24. VI. 25, like VI. 24 and 18, considerable ability and intellectual interest, showing considerable business aptitudes. Groups VI. 28 to 34 and VI. 35 to 39, although double first cousins, are more unlike than any cousins on either side, both in person and character; all the first group very dark and inclined to be melancholy and wanting the marked family energy, the second group cheerful and fond of company; aet. 22 to 32. VI. 39, hardworking, considerable scientific aptitudes, aet. 19. VI. 40, formerly public school scholar, unconventional and socialistic in tendencies, aet. c. 29. IV. 41, interested in social and political work, aet. 27. VI. 42, civil servant, industrious and conscientious, comes of family with considerable philanthropic and artistic gifts. VI. 45, died at two years of acute inflammation of the lungs. VI. 46 and 47, public school scholars, cheerful normal boys at present. VI. 48, delicate nervous lad, very conscientious, troubled childhood, aet. c. 19; had pneumonia. VI. 49 to 53, ages nine to two years, said to be healthy, normal, boisterous children. VII. 1—15, all under 11 years; for full description see E IX. 18 to 30.

PLATE XXIV. Fig. 192, F. This family were settled in and around a small upland manufacturing town at the close of the 17th century. They claimed descent from a well-known county family in the

neighbourhood. They rose with the rise of the town; as VI. 12, through whom the pedigree is traced, migrated and settled elsewhere at an early age, the ties of kinship have been considerably relaxed and little is known of the family as individuals. They remain worthy members of the middle class, careful, frugal, industrious. III. 1, born c. 1685. IV. 1, a whitesmith. IV. 3, died in the prime of manhood, act. c. 34, an able nonconformist. IV. 4, respectable and venerable, very active and upright, strongly religious, Calvinistic opinions, believer in supernatural occurrences, died aet. 83. V. 1, child or children unknown. V. 2, delicate, anxious minded, died of consumption four years after birth of last child, aet. 46. V. 3, woollen cloth manufacturer and dyer, able successful man, industrious, ingenious, generous, affectionate, dutiful, ordained first Baptist deacon of new brotherhood, very strict, died aet. 80. V. 4, kind, industrious woman, survived her husband some years, died aet. 78. V. 5, eight children of whom three died in infancy. V. 6, quiet, unobtrusive, snuff-taker, died aet. 57. VI. 1, died in infancy. VI. 2, manufacturer. VI. 3, died aet. 73. VI. 5, died aet. 30. VI. 6, died aet. 72. VI. 9, manufacturer, died aet. 73. VI. 10, died aet. 31. VI. 12, very successful in business, shewed much originality and power of adaptation; great interest in municipal reform, and in all philanthropic and educational movements; liberal in politics and religion, conscientious, hardworking, trustworthy; healthy till old age set in, died of general decay, aet. 70. VI. 13, manufacturer, died aet. 60. VI. 14, woman of old family, well connected. VI. 15, died aet. 30, unmarried. VI. 16, died aet. 31, unmarried. VI. 17, died an infant. VII. 1 and 2, believed to have emigrated. VII. 6, went to U.S.A., a son did very well and grandsons are now said to be prosperous men out west. VII. 9, in business, went to U.S.A., died of yellow fever, aet. 52. VII. 11 and 12, died unmarried. VII. 15, presumably drowned in U.S.A. VII. 16, rather eccentric old man, very parsimonious and frugal, married late in life, died aet. c. 80. VII. 18, eccentric, aet. c. 78; keeps up old family traditions and house. VII. 19, lives with VII. 18, partially paralysed by a stroke, aet. c. 73, reverted to life of working woman, keeps no servants, well off but spends nothing. VI. 20, child or children who died in infancy. VIII. 1, died unmarried. VIII. 4, died unmarried. VIII. 7, died young on eve of marriage. VIII. 8, alive, aet. c. 73. IX. 1, in charge of Mission Hospital in East, aet. c. 40. IX. 2, aet. c. 38. X. 1 and 2, aet. eight and three.

PLATE XXIV. Fig. 192, G. This family were small landowners of yeoman standing, a property of about 80 acres remained in their possession throughout the period of this pedigree. The junior members were husbandmen, brewers or went into trade in the neighbouring seaport town. The family seems to have been strongly puritan and intermarried with other puritan families. They appear to be off-shoots of a small family of gentry who owned land in the district from time immemorial. A member of the older branch founded a grammar school c. 1685. Tradition relates that the family were dark, good featured, rather taciturn and self-contained, frugal, industrious. The early part of the pedigree is pieced together from wills and registers. From Gen. V. onwards it becomes a matter of family history and personal knowledge. I. 2, a yeoman of good position. I. 3, died 1650, probably aet. c. 50. I. 4, daughter of a small squire of good position. II. 3, yeoman, died aet. c. 82, married aet. 26. II. 6, died aet. 73, husbandman, had a small property. III. 3, daughter and niece of prominent presbyterian yeomen, a woman of character and influence, died 50 years after marriage. III. 4, yeoman, died, aet. 60. III. 11, mariner, died s.p., probably unmarried. IV. 5 and 6, died in infancy. IV. 7, yeoman. IV. 9, prosperous yeoman and tenant farmer. IV. 11, died unmarried, aet. 79. IV. 12, yeoman, died c. 90. IV. 13, daughter of a neighbouring squire, sister of IV. 16; a nephew in the army is said to have distinguished himself at Waterloo. IV. 14, died in infancy. IV. 15, yeoman, died aet. c. 88. IV. 16, sister of IV. 13, died aet. 68. V. 12. a staymaker. V. 13, died in infancy. V. 14, land agent and yeoman, died of decline, aet. c. 40. V. 15, died in infancy. V. 16, clever managing woman, married late in life a man younger than herself, died aet. c. 80. V. 17, a militia man. V. 18, unmarried, died aet. 66. V. 19, stern, reserved, affectionate, at first wine merchant, then farmed family estate, was engaged for 17 years but would not marry till he had paid off debts contracted by V. 23, a simple-living hard-working man, greatly respected and esteemed, died aet. c. 80. V. 20, small, fair, delicate woman, considerable refinement and grace, probably had north Irish blood, father a sea captain sailing to China, etc. in late 18th century; died aet. 70. V. 21, believed to have married. V. 22, lost sight of in U.S.A. V. 23, rather wild and unsatisfactory, died aet. 63. V. 24, barmaid, probably older than husband. V. 25, died in infancy. V. 26, died aet. 31. VI. 19, successful wine merchant. VI. 21, died at birth. VI. 22, capable, intelligent woman, full of energy, clever, well educated and well read, much interested in all her husband's and sons' concerns, a good housekeeper, fond of society and travelling; good general health till old age; several strokes of paralysis and stomach weakness, also gangrene; died aet. 73; resembled her father. VI. 23, clever, intelligent, married rather late in life, died aet. c. 71. VI. 24, M.P., friend of Cobden, strong radical, very successful financier, lived frugally, left a large fortune, married late, died aet. c. 80; some members of his family appear to have been eccentric; able, intelligent man. VI. 25 to 29, no details. VI. 30, of yeoman class. VI. 31, wild in youth, settled down as good man of business, died aet. c. 73. VI. 32, village schoolmistress, aet. c. 60. VI. 33, believed to be married; VII. 36, successful merchant and brewer, took up astronomy as hobby, studied comets, invented telescopic improvements, F.R.S., Royal Medallist, died aet. 81. VII. 38, paralysed from birth, suffered from internal tumour, intelligent, philanthropic, became excitable

and difficult, committed suicide, aet. c. 58. VII. 39, intelligent, capable, industrious, aet. c. 65. VII. 40, one of a large and prosperous city family, baronet, M.P., talented. VII. 41, 42, 43, died, aet. c. 30, unmarried. VII. 44 and 45, believed to be settled in the Colonies. VIII. 48 and 49, alive, aet. c. 80, unmarried. VIII. 51, died, aet. two days.

PLATE XXIV. Fig. 192, E. VII. 21, rather delicate, sensitive woman, good intellectual and artistic abilities, religious minded, very fond of children, a born educator, took part in various philanthropic and educational movements as far as strength would allow, latterly troubled with delicate heart and lungs, died of bronchitis and heart failure, aet. 64. VII. 22, rather wild in early manhood, fond of easy living and mild outdoor pursuits, carried on family traditions in politics and religion, died of inflammation of the lungs, aet. 64. VII. 23, woman from well-to-do nonconformist family, suffered in her last years from diabetes, died of general failure, bad veins, gangrene of foot, etc., aet. 64. VII. 24, prosperous, successful shipowner, active, capable, hardworking in middle life, considerable intellectual and artistic interests, formed a really fine collection of pictures; generous supporter of local University, and of liberal, religious, and social objects; suffered from hernia in last years, died aet. 70, after short illness from blood poisoning due to abscess on liver. VII. 25, second daughter of large cultivated nonconformist family, persistent, capable woman, works hard at various philanthropic objects; delicate for many years after birth of first child; most generous, unassuming woman, in spite of great wealth; religious minded, artistic, considerable intellectual abilities, immense energy, suffered from diabetes, aet. c. 76. VII. 26, died, aet. 2½ years, of low fever. VII. 27 and 29, two first cousins with poor family history. VII. 27, one of two sisters, the other has lived under control with a doctor; they had one brother who was eccentric, VII. 27, died of puerperal fever in third confinement, aet. c. 30. VII. 28, an able original man, almost a genius in mechanical devices; very successful shipowner, full of plans of all sorts; active in mind and body, unconventional, hardworking, steady, conscientious, strongly puritan in sympathies, and by instinct, suffers from hernia; had one or two slight strokes but recovered very fairly completely, alive, aet. 80. VII. 29, a most kind, cheerful woman, in middle age developed epilepsy, melancholia and suicidal mania; for many years under control, now somewhat better but failing in general health, aet. c. 63; one of at least seven sisters, of whom one died of consumption, and one of melancholia, brain trouble after influenza. VII. 30, a quiet very able man, the confidant and adviser of half his acquaintances; reserved, well balanced, unbending, simple in habits and tastes, almost ascetic in food and dress, very well read, very broad and sympathetic in thought, entirely detached from any self-interest or personal self-seeking, extremely active in philanthropic, religious, and social reforms, without allowing his name to transpire; spare, active, energetic, aet. 78. VII. 31, a sister of B. V. 56 in Plates XXII—XXIII; another sister was active school manager and Poor Law Guardian (died c. 60 of inflammation of the lungs); the elder brother and his two sons are first rate business men and have considerable intellectual ability; she was a clever, persistent woman of much ability, took little exercise; in last years liable to violent attacks of nose bleeding, died of some sort of apoplexy and heart failure, aet. c. 64. VII. 32 and 33 are given under C. V. 2 and 1. VII. 34, clever bright child, died of low fever, aet. six. VIII. 12, stockbroker, aet. c. 60. VIII. 13, 15, 16, 18, 19, 21, 23, 25, family of medium ability, the women industrious, and good managers, all said to take conventional views in politics, religious and social matters. VIII. 13, capable woman, good tastes and fair abilities, married, aet. c. 54. VIII. 14, son of a sister of VII. 25, aet. c. 47. VIII. 15, killed in carriage accident shortly before third confinement, aet. c. 28. VIII. 16, deaf after scarlet fever; took to farming successfully; suffered from varicose veins, clots, etc., died of some complication, aet. c. 35. VIII. 23. aet. 37. VIII. 24, daughter of a sister of VIII. 22, delicate, father died of rapid consumption. VIII. 26, infant son, died aet. five days. VIII. 27, capable industrious woman, interested in education, art, philanthropy, aet. c. 47. VIII. 28, very intelligent, active and capable, F.R.S., devoted to his scientific work, aet. c. 50. VIII. 29, intelligent, industrious woman, Honours in Science Lond. Univ. when 21, aet. c. 42. VIII. 30, daughter of a younger sister of VII. 29, alive, aet. c. 32. VIII. 31, shipowner, engineer, well read intelligent man, great traveller, very shy and self-conscious, had typhoid severely aet. 25, now aet. c. 40. VIII. 32, a peculiar, excitable child, very large head, died of water on the brain, aet. six. VIII. 33, aet. 35. VIII. 34, stillborn or died at birth. VIII. 35, as a child exceedingly delicate, not thought possible to rear him till after ten years or so, then supposed to be tuberculous, suffered from glands, digestive troubles, etc.; about 15 gained strength, now fairly normal. VIII. 36 to 47, same as C. VI. 1 to 12 inclusive. IX. 6, brought up on a ranch, aet. c. 16. IX. 7 and 8, twins, one born club-footed, aet. c. four. IX. 9, aet. c. 16. IX. 10, aet. c. 14. IX. 11, developed tubercular disease of bowels, delicate, aet. four. IX. 12, aet. two. IX. 13, clever, excitable child, nervous, much intellectual ability, aet. 13. IX. 14, aet. 11. IX. 15, died at birth. IX. 16, aet. seven. IX. 17, normal child, aet. four. IX. 18, clever child, considerable intellectual aptitudes, nervous, aet. ten. IX. 19, backward but intelligent child, slight paralysis of tongue, aet. nine. IX. 21, stillborn. IX. 22, intelligent, capable, well developed child, aet. nine. IX. 23, aet. seven. IX. 24, clever, restless child, aet. five. IX. 25, baby, aet. 22 months. IX. 26 to 29, rather delicate, backward children. IX. 26, squints badly. IX. 29, born prematurely, IX. 26 to IX. 30 are aet. 9 to 1.

PLATE XXIV. *Commercial and Legal Ability.* Fig. 193. I. 1, died aet. 59, successful manufacturer; started a type-founding business (*D.N.B.*). II. 1, died aet. 92, F.R.S., pioneer in meteorology (*D.N.B.*). II. 3, died aet. 79, calligraphist and author (*D.N.B.*). II. 8, died aet. 81, M.D., devoted his life to type-founding business and to the philological studies connected with it (*D.N.B.*). II. 15, dead, interested in locomotion; received a medal from government for an essay on employment of the poor in Ireland. III. 3, died aet. 76, F.R.S. (*D.N.B.*). III. 7, died aet. 68, physician and author of medical works (*D.N.B.*). III. 9, died, aet. 75, barrister and leading member of the Society of Friends (*D.N.B.*). III. 22, died aet. 83; a great authority on bibliography (*D.N.B.*). IV. 3, living, 79, engineer; collector of literary curiosities. IV. 5, living, aet. 77, D.C.L., Litt.D., banker and historical writer. IX. 9, died aet. 75, distinguished architect. IV. 21, living, aet. 82, has maintained and extended a large manufacturing business and taken an active part in philanthropic work. IV. 22, living, aet. 81, D.C.L., F.R.S., member of the Permanent Court of International Arbitration at the Hague, formerly Lord Justice of Appeal. IV. 28, living, aet. 76, P.C., M.P. IV. 41, living, aet. 72, F.S.A., M.P., created baronet. V. 1, born 1855—67. V. 7, living, aet. 31; fellow and lecturer in modern history at an Oxford college. V. 9, born 1861—72. V. 10, born 1875—82. V. 11, born 1875—82. V. 13, living, aet. 48, barrister-at-law; distinguished university career, shut out from active life owing to ill health. V. 15, living, aet. 46, an active guardian of the poor and in other benevolent and social matters. V. 17, living, aet. 42, artist and writer on art. V. 43, eldest, born 1862. V. 44, eldest, born 1888. V. 50, living, aet. 40, barrister-at-law; distinguished university career. V. 53, living, aet. 36; distinguished university career. V. 56, eldest, born 1870. V. 57, eldest, born 1885. V. 58, eldest, born 1891. (*D.N.B.* = account of in *Dictionary of National Biography.*)

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TROPHOEDEMA.

FIG. 75 Sutherland's Case

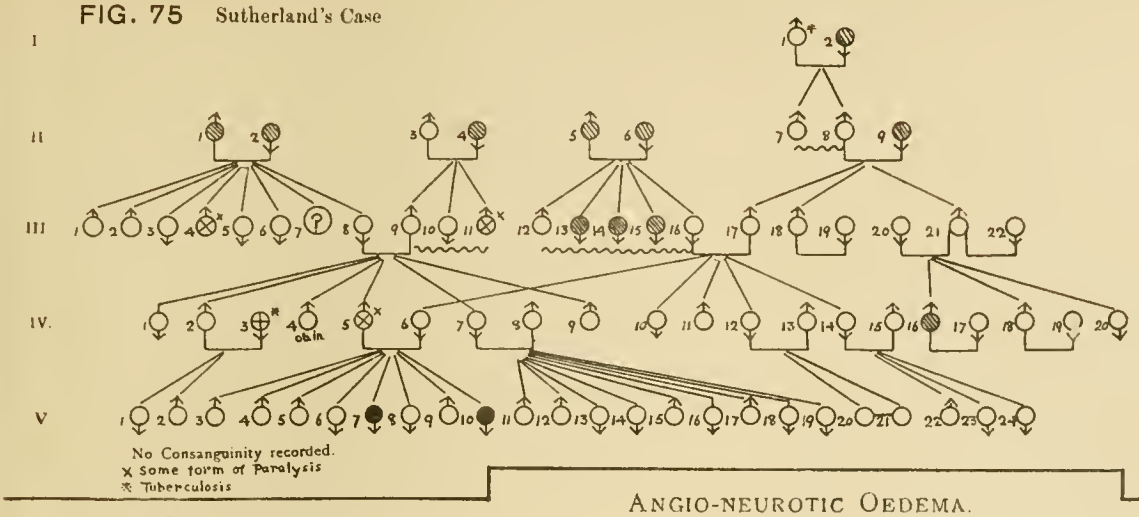
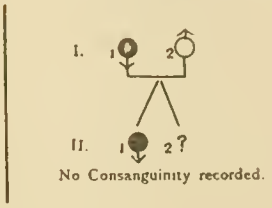


FIG. 76 Moyer's Case



ANGIO-NEUROTIC OEDEMA.

FIG. 77 Wardrop-Griffiths' Case

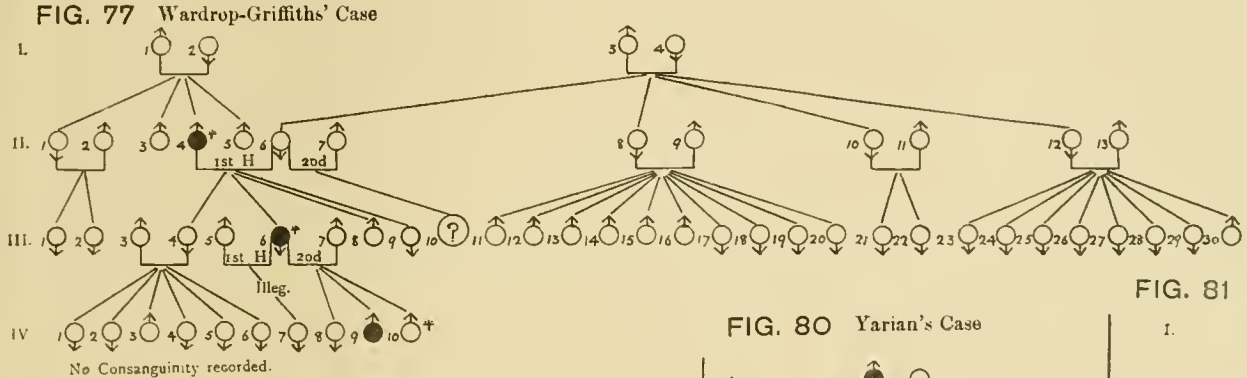


FIG. 78 Falcone's Case

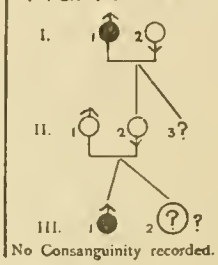


FIG. 79 Osler's Case

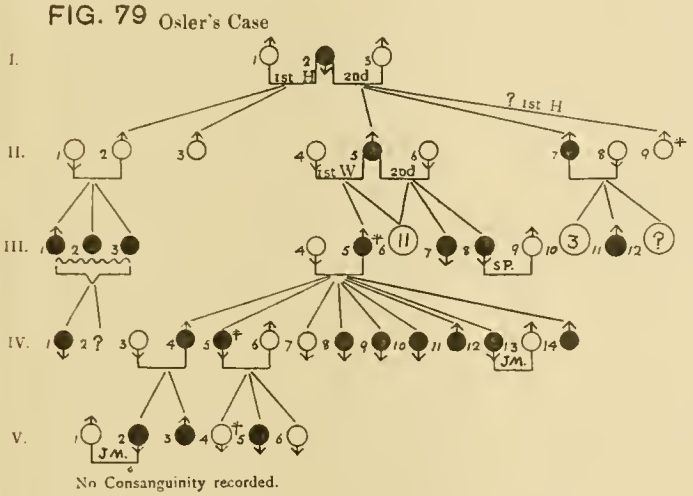


FIG. 80 Yarian's Case

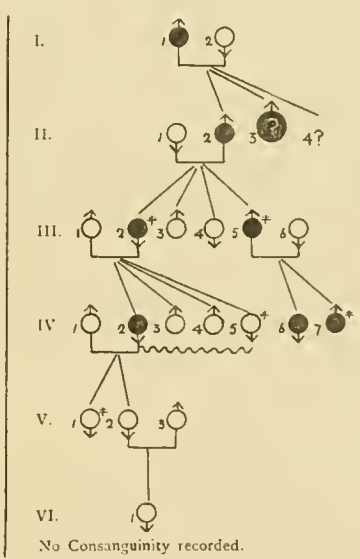


FIG. 81 Prior's Case

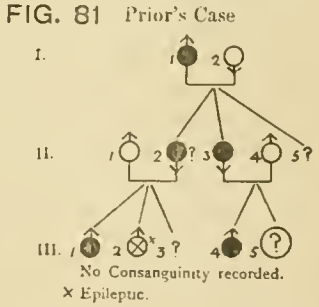


FIG. 82 Apert and Delille's Case

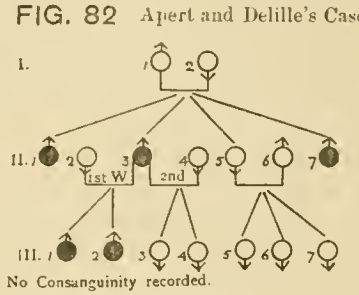


FIG. 83 Josephs' Case

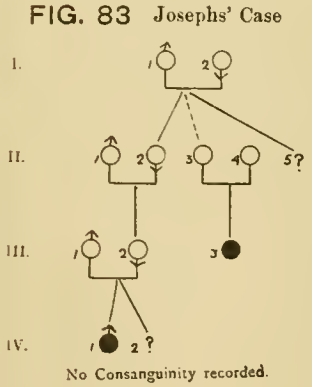


FIG. 84 Mendel's Case

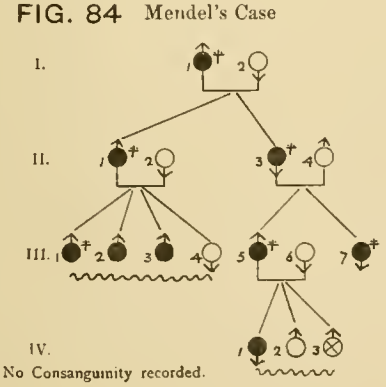


FIG. 85 Quincke-Dinkelacker's Case

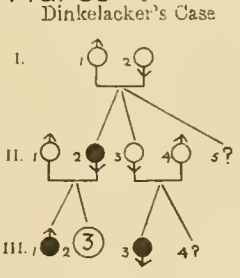


FIG. 86 Schlesinger's Case

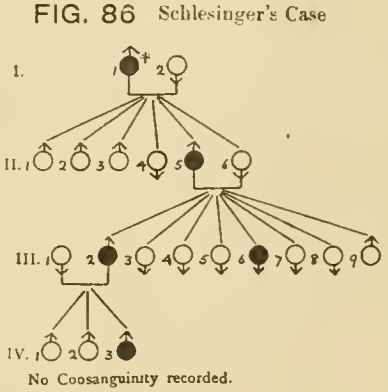
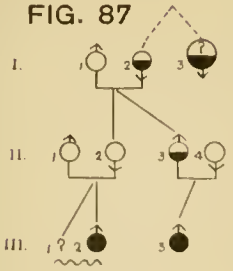
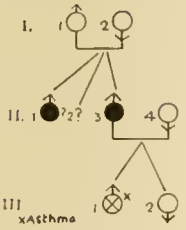


FIG. 87



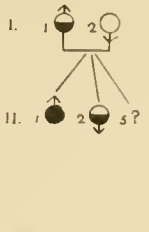
Halsted's Case (i)

FIG. 88



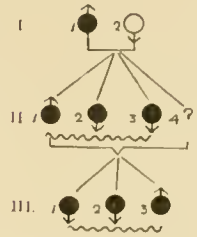
Halsted's Case (ii)

FIG. 89



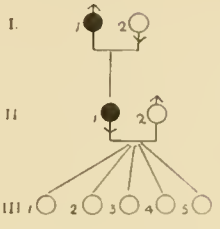
Halsted's Case (iii)

FIG. 90



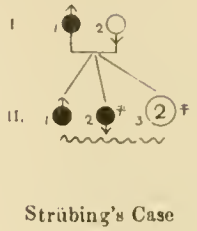
Ricochon's Case

FIG. 91



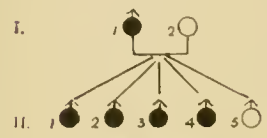
A. J. Smith's Case

FIG. 92



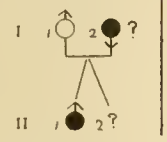
Strübing's Case

FIG. 93



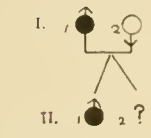
Courtade's Case

FIG. 94



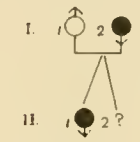
Cuntz's Case

FIG. 95



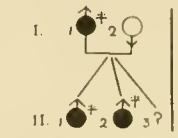
Quincke's Case

FIG. 96



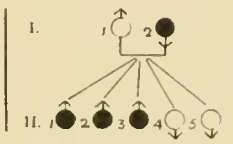
Roy's Case

FIG. 97



Sträussler's Case

FIG. 98

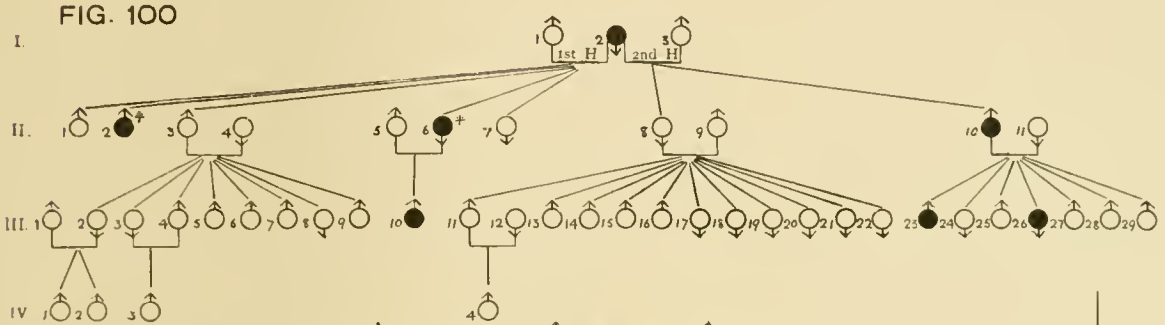


Mesler's Case

FIG. 99

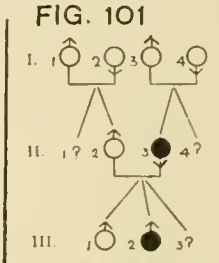
Quincke-Dinkelacker and Valentin's Case

FIG. 100



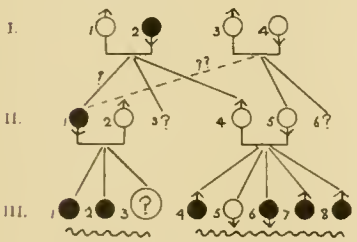
Byrom Bramwell's Case

FIG. 101



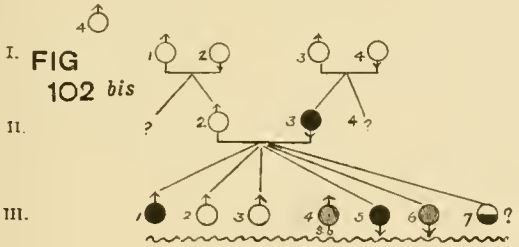
Ross' Case

FIG. 102



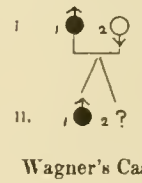
Fritz's Case

FIG 102 *bis*



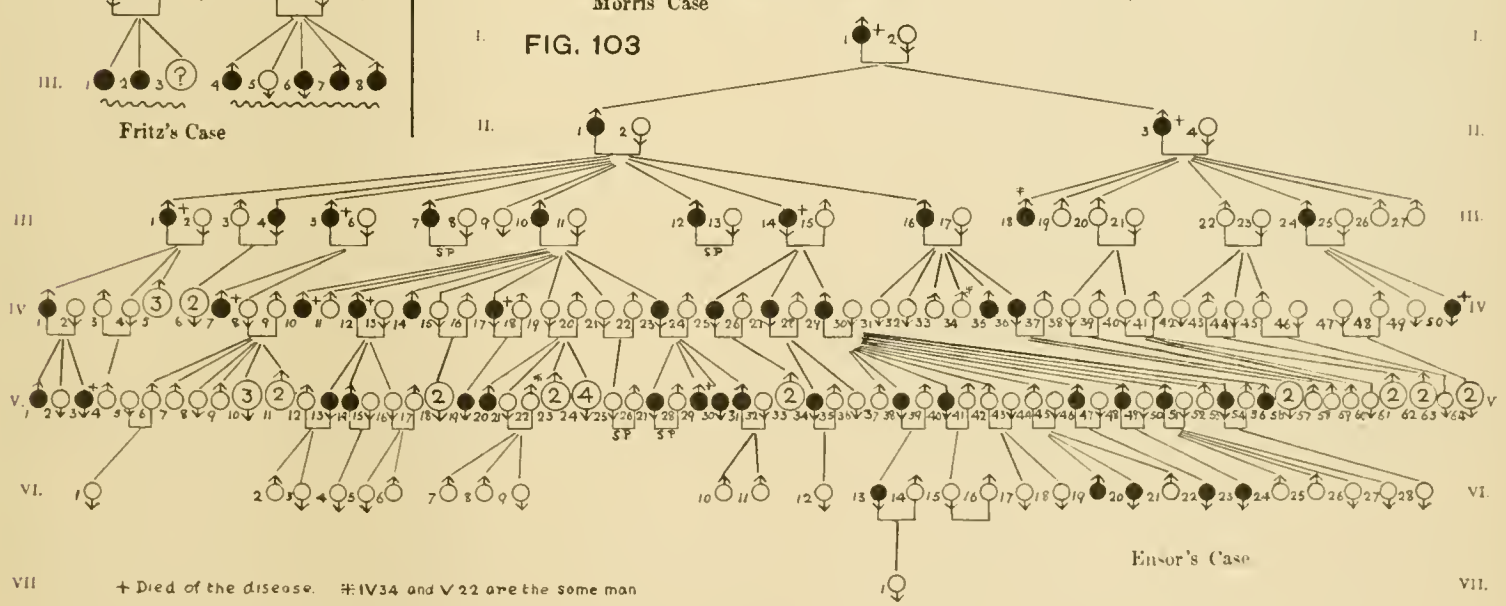
Morris' Case

FIG. 104



Wagner's Case

FIG. 103



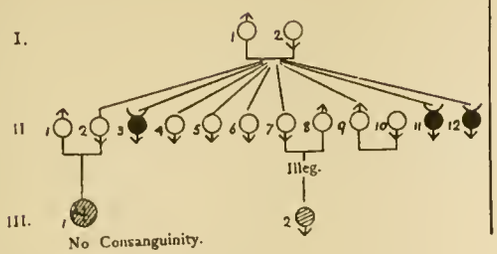
Ensor's Case

+ Died of the disease. #IV34 and V22 are the some man

Consanguinity in V 21's union

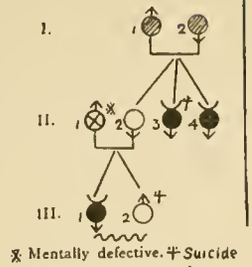
No Consanguinity recorded, in Figures 87-104

FIG. 105



Pozzi

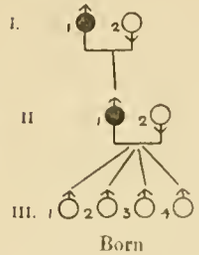
FIG. 106



⁂ Mentally defective. ✝ Suicide

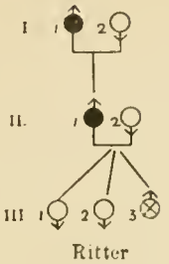
de Beurmann and Roubinowitch

FIG. 107



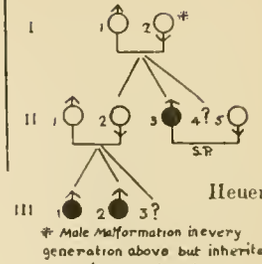
Born

FIG. 108



Ritter

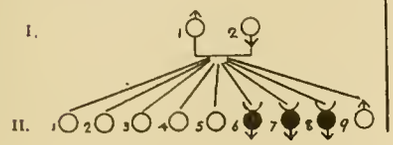
FIG. 109



Heuermann

* Male Malformation in every generation above but inherited through females

FIG. 110

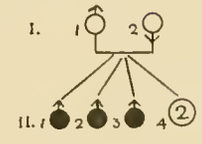


Lindsay

FIG. 111

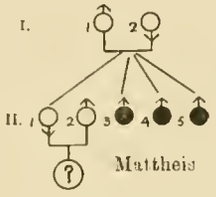
Neugebauer

FIG. 112



Fenoglio

FIG. 113



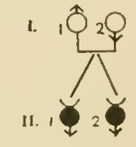
Mattheis

FIG. 114

✕ Hare Lip, cleft palate Polydactyly

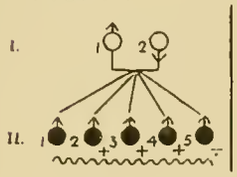
Matthes

FIG. 115



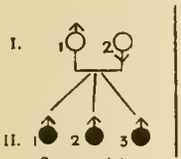
Van der Hoeven

FIG. 116



Levy

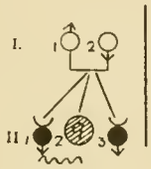
FIG. 117



Consanguinity. of I 1 and 2

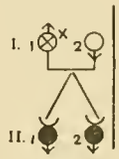
Parlier

FIG. 118



Barnes

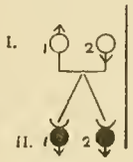
FIG. 119



✕ Insane

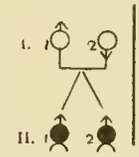
Martin

FIG. 120



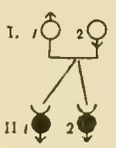
Mundé

FIG. 121



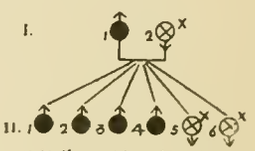
Chiarleoni

FIG. 122



du Basty

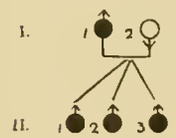
FIG. 123



✕ Malformation of hands

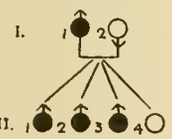
Shorthouse

FIG. 124



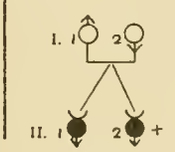
Bryant I.

FIG. 125



Bryant II.

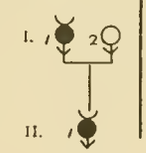
FIG. 126



+ Died at 15

Liersch

FIG. 127

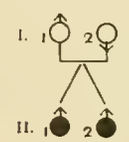


Traxel

FIG. 128

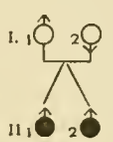
Simeons I.

FIG 129



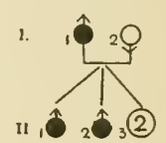
Simeons II.

FIG. 130



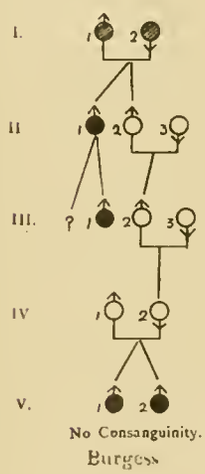
Legueu

FIG. 131



Bellec

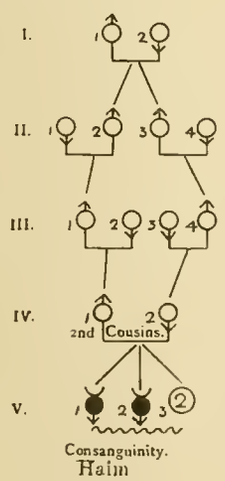
FIG. 132



No Consanguinity.

Burgess

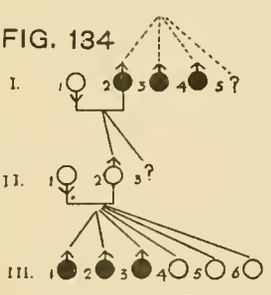
FIG. 133



Consanguinity.

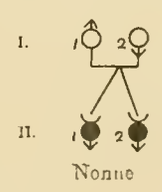
Haim

FIG. 134



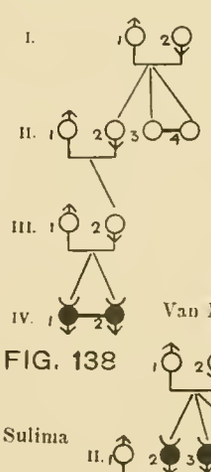
Rigaud

FIG. 135



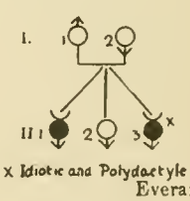
Nonne

FIG. 136



Van Mons

FIG. 137



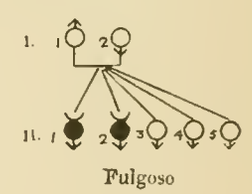
✕ Idiotic and Polydactyle

Everard Home

FIG. 138

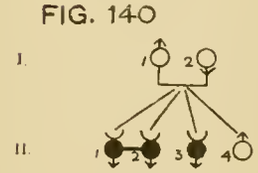
Sulima

FIG. 139



Fulgoso

FIG. 140



Curling

FIG. 141

Naegele

FIG. 142

✕ Encephalocele occipitalis

Lehmann

FIG. 143

✕ Monster

Katsky

FIG. 144

Bryant III.

FIG. 145

✕ Double Monster

Paré

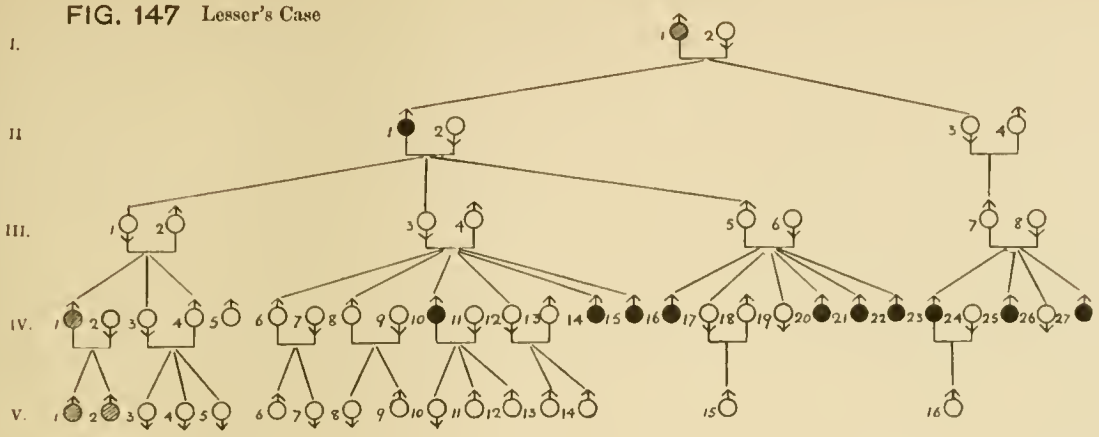
FIG. 146

Saviard

No Consanguinity recorded in this plate except where it is stated.

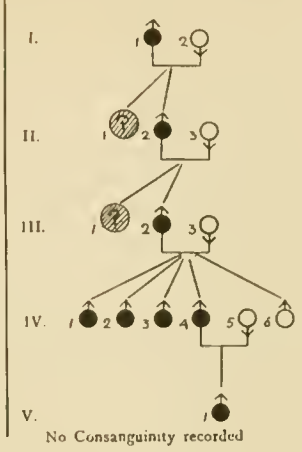
Denotes an individual whose sex has been at some period uncertain

FIG. 147 Lesser's Case



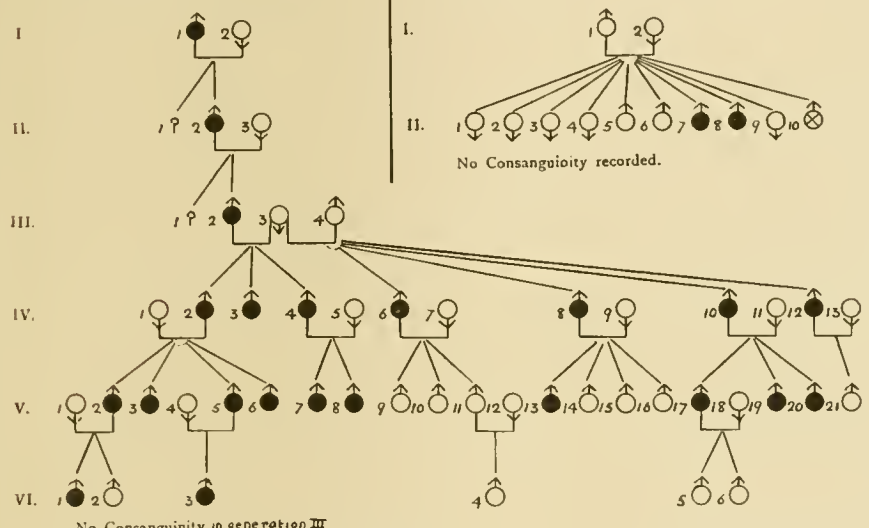
No Consanguinity recorded.

FIG. 148 Strong's Case



No Consanguinity recorded

FIG. 149 Lingard's Case

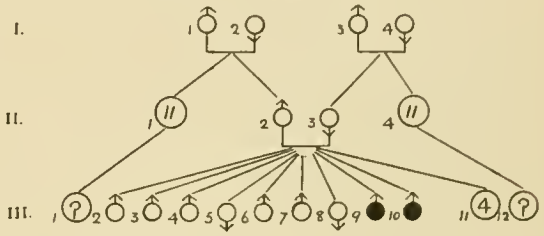


No Consanguinity in generation III
Female offspring not included in this pedigree

FIG. 150 Gaetano's Case

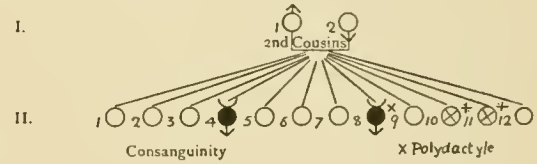
No Consanguioity recorded.

FIG. 151 Kellock's Case



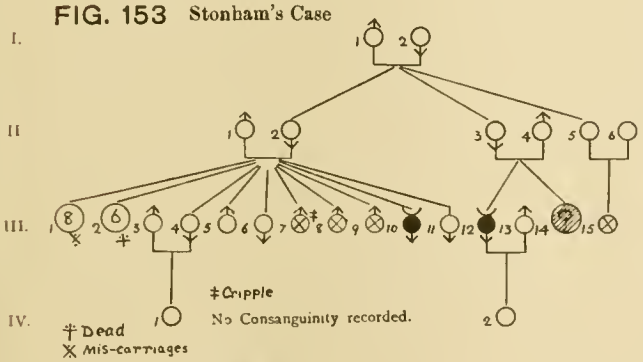
No Consanguinity recorded.

FIG. 152 Corby's Case



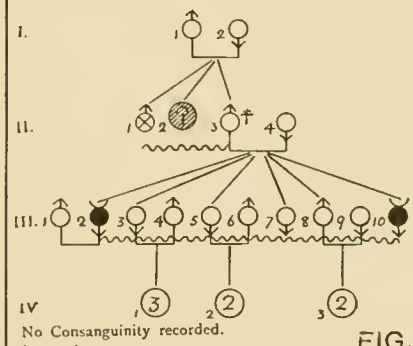
Consanguinity
x Polydactyle
II 1-9,12, all but three dead of Tuberculosis. + Premature birth & early death.

FIG. 153 Stonham's Case



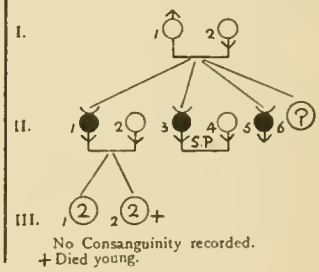
† Dead
✕ Mis-carriages
‡ Cripple
No Consanguinity recorded.

FIG. 154 Hengge's Case



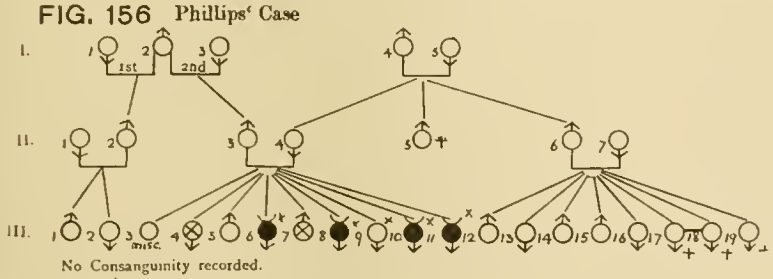
No Consanguinity recorded.
† Dead

FIG. 155 Lepechin's Case



No Consanguinity recorded.
+ Died young.

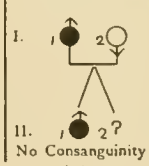
FIG. 156 Phillips' Case



No Consanguinity recorded.
† Dead
+ Died young.

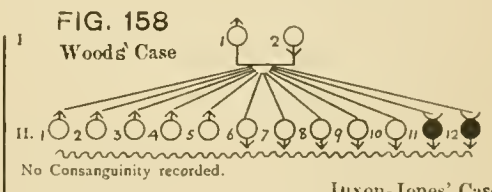
Denotes an individual whose sex has been at some period uncertain

FIG. 157 Brière's Case



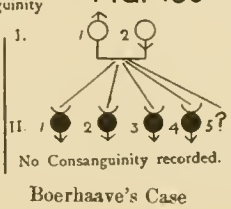
No Consanguinity recorded.

FIG. 158 Woods' Case



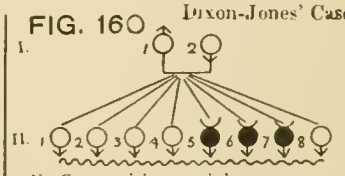
No Consanguinity recorded.

FIG. 159



No Consanguinity recorded.
Boerhaave's Case

FIG. 160 Dixon-Jones' Case



No Consanguinity recorded.

FIG. 161 Urquhart's Case

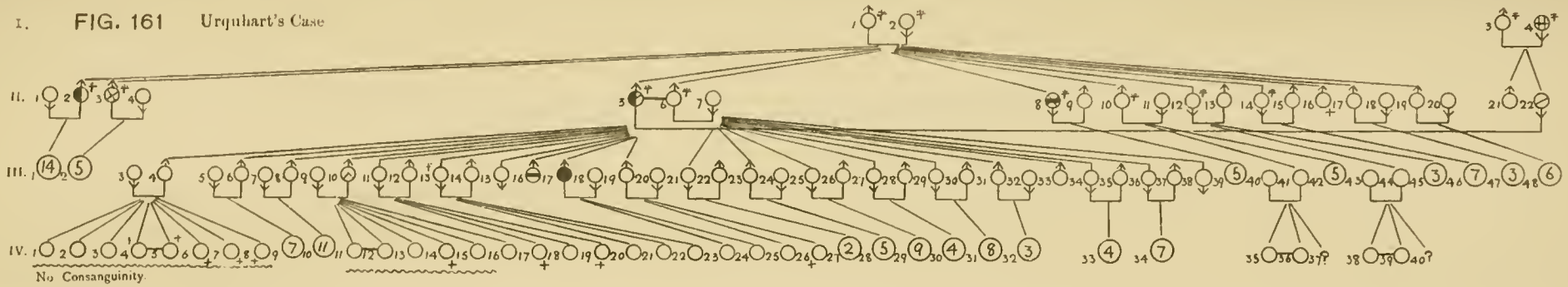


FIG. 162 Urquhart's Case

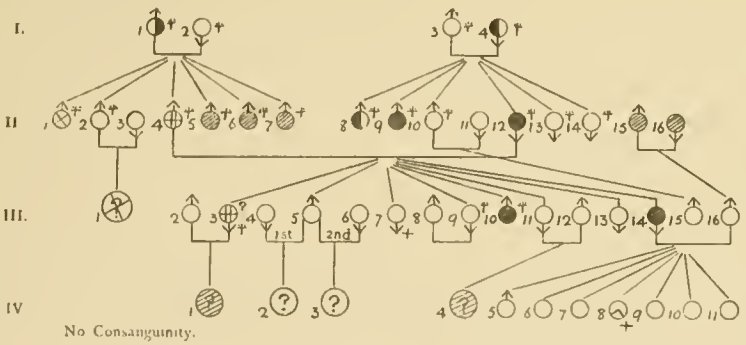


FIG. 163 Urquhart's Case

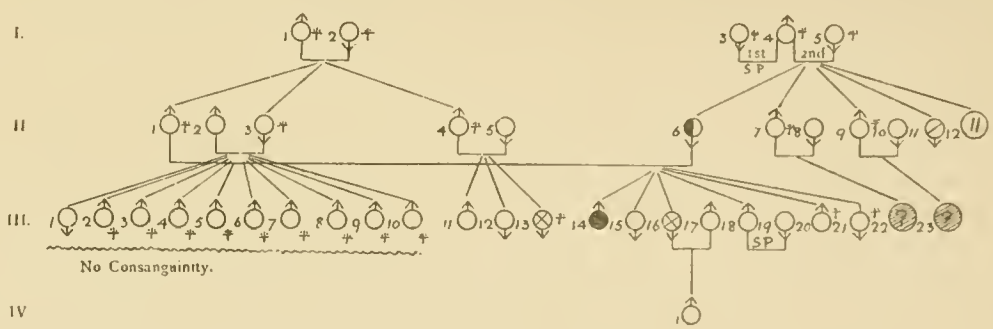


FIG. 164 Urquhart's Case

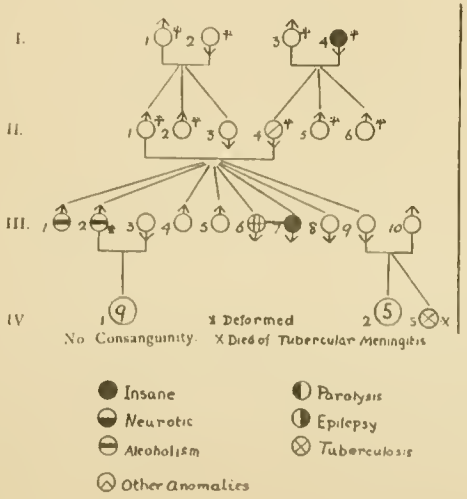


FIG. 165 Urquhart's Case

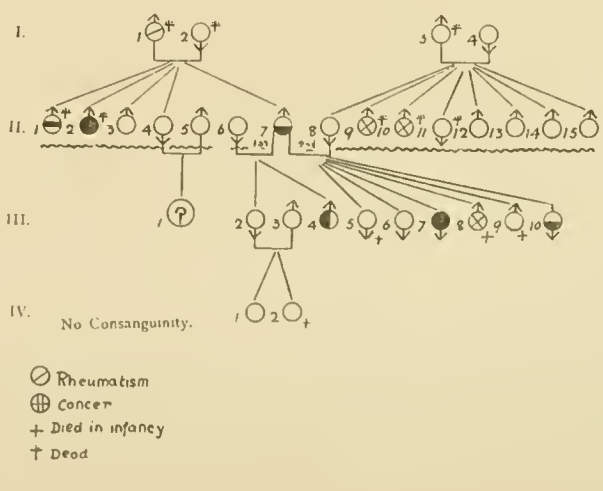


FIG. 166 Urquhart's Case

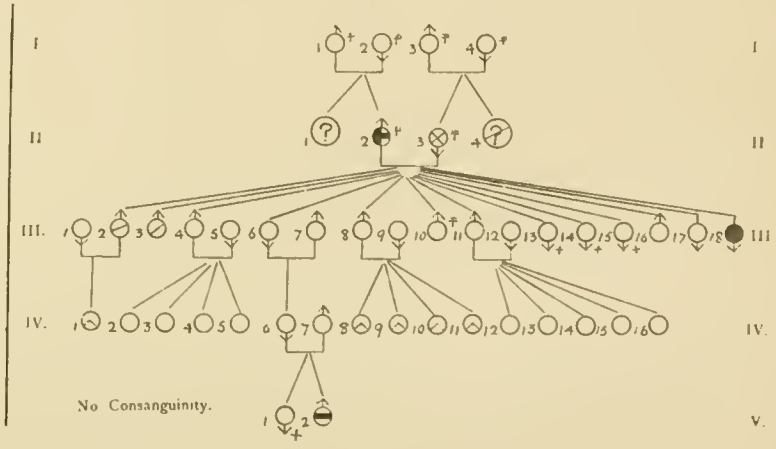
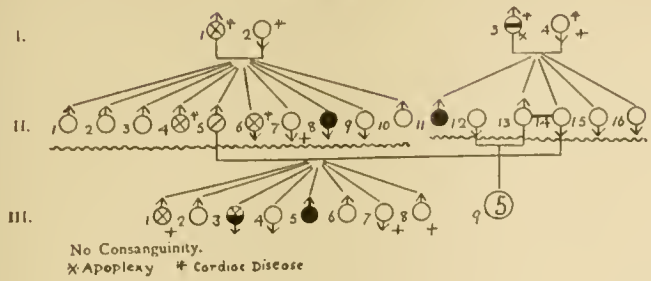
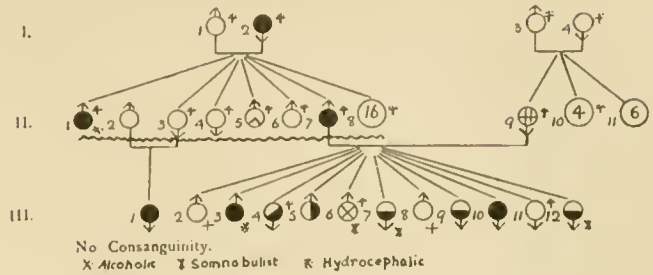


FIG. 167 Urquhart's Case



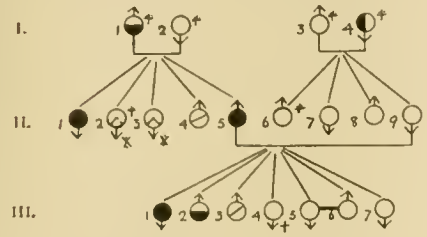
No Consanguinity.
X Apoplexy ✝ Cardiac Disease

FIG. 168 Urquhart's Case



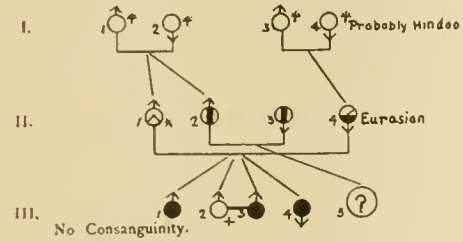
No Consanguinity.
X Alcoholic ‡ Somnambulist ✝ Hydrocephalic

FIG. 169 Urquhart's Case



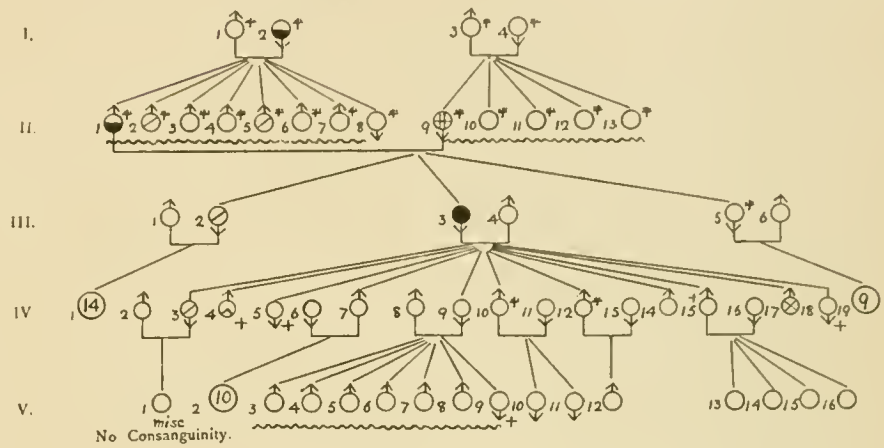
No Consanguinity.
‡ Spinal Disease

FIG. 170 Urquhart's Case



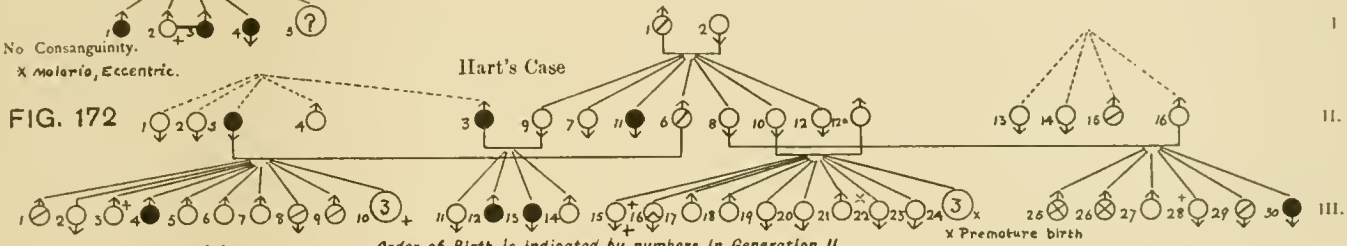
No Consanguinity.
X Malaria, Eccentric.

FIG. 171 Urquhart's Case



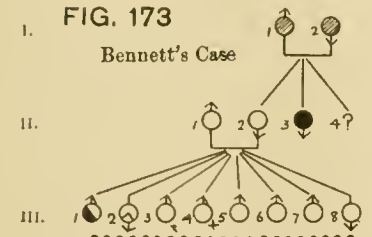
No Consanguinity.

FIG. 172 Hart's Case



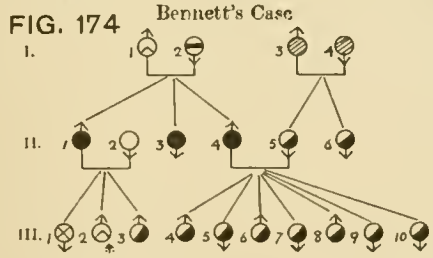
No Consanguinity recorded.
Order of Birth is indicated by numbers in Generation II.
X Premature birth

FIG. 173 Bennett's Case



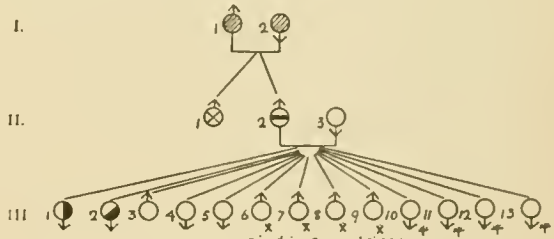
X Died in Convulsions
No Consanguinity recorded.

FIG. 174 Bennett's Case



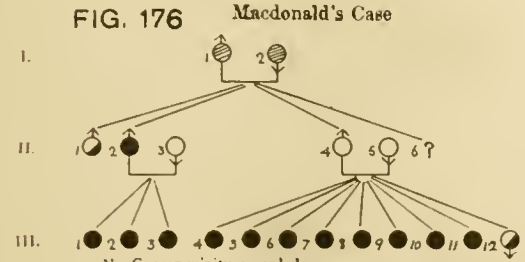
✝ Reformatory
No Consanguinity recorded.

FIG. 175 Bennett's Case



No Consanguinity recorded.
X Died in Convulsions

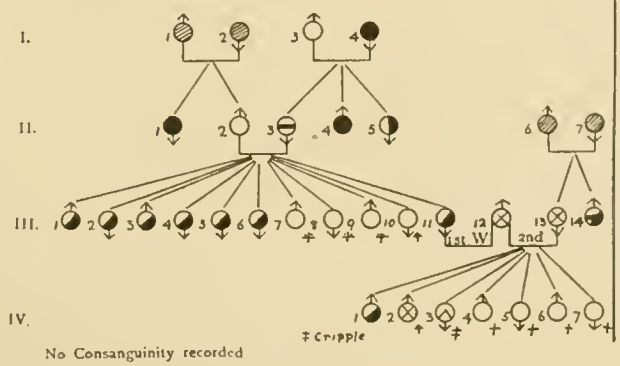
FIG. 176 Macdonald's Case



No Consanguinity recorded.

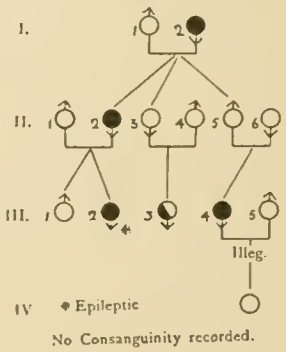
- Insane
- Neurotic
- Epilepsy
- Idiot
- Imbecile and Mentally defective
- Paralysis
- Rheumatism
- Tuberculosis
- Alcoholism
- Cancer
- Suicide
- Other Anomalies
- + Died in infancy
- ✝ Dead

FIG. 177 Bennett's Case



‡ Cripple
No Consanguinity recorded

FIG. 178 Macdonald's Case



✝ Epileptic
No Consanguinity recorded.

FIG. 179 Graham Bell's Case

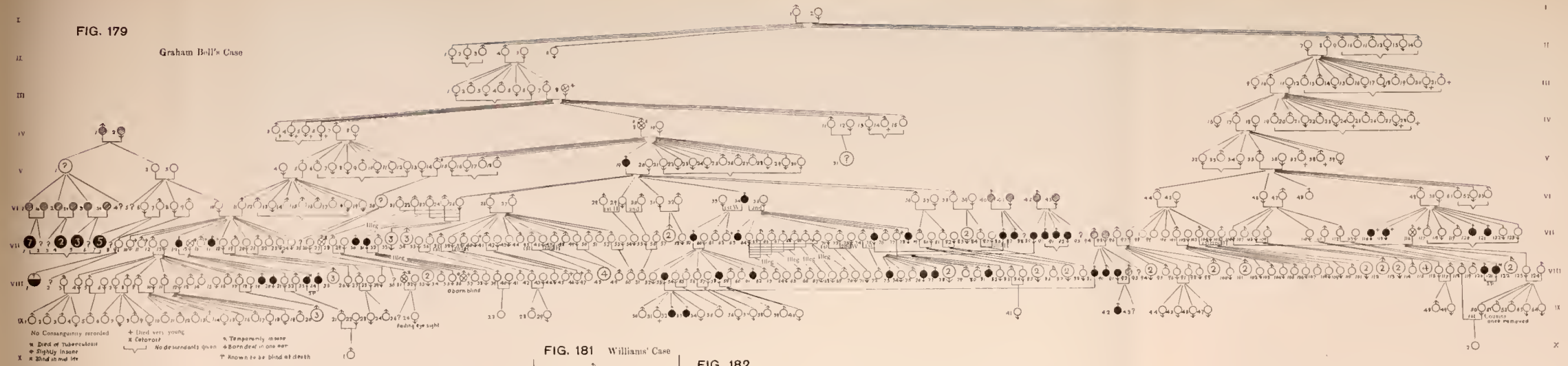


FIG. 180 Graham Bell's Case

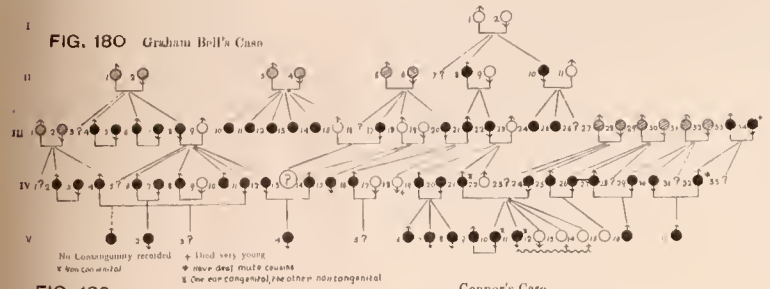


FIG. 181 Williams' Case

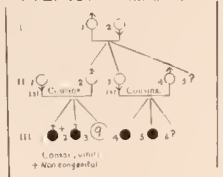


FIG. 182 Kerr Love's Case

FIG. 183 Connor's Case

FIG. 184 Mattieson's Case

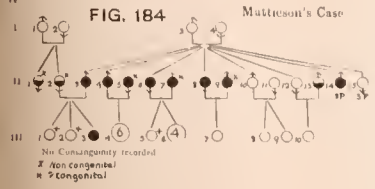


FIG. 185 *

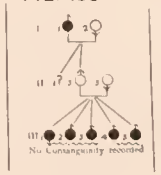


FIG. 186 *

FIG. 187 Williams' Case

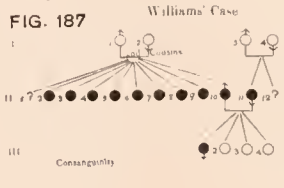


FIG. 188 Connor's Case

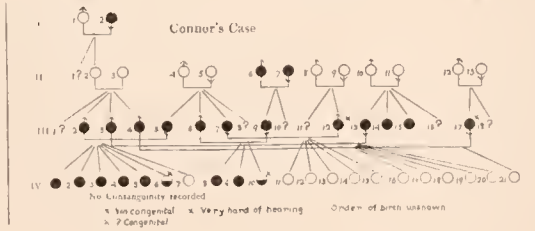


FIG. 189 Stephenson's Case

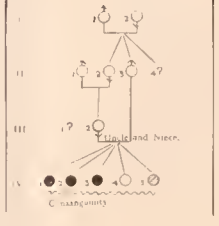


FIG. 190 Connor's Case

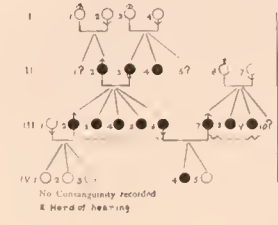
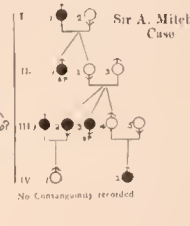
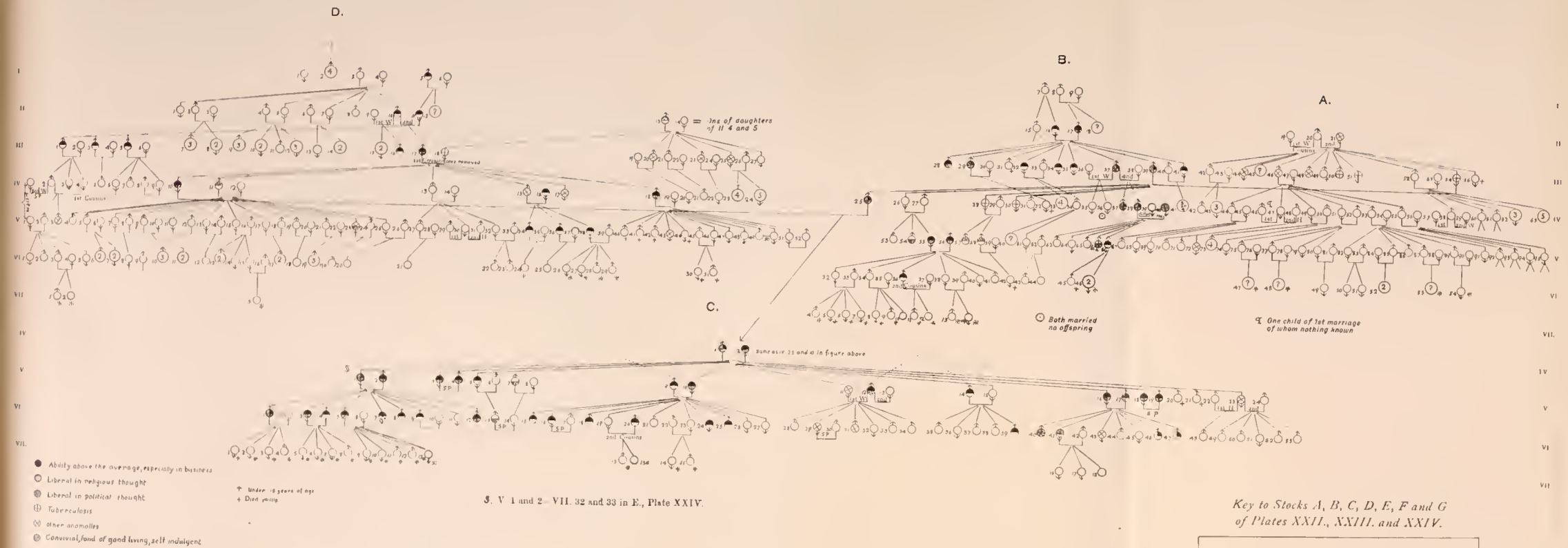


FIG. 190 *bis* Sir A. Mitchell's Case



× Royal Commission, 1889.

FIG. 192. A–D.



3, V 1 and 2 – VII. 32 and 33 in E., Plate XXIV.

Key to Stocks A, B, C, D, E, F and G of Plates XXII., XXIII. and XXIV.

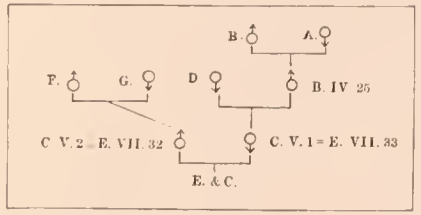
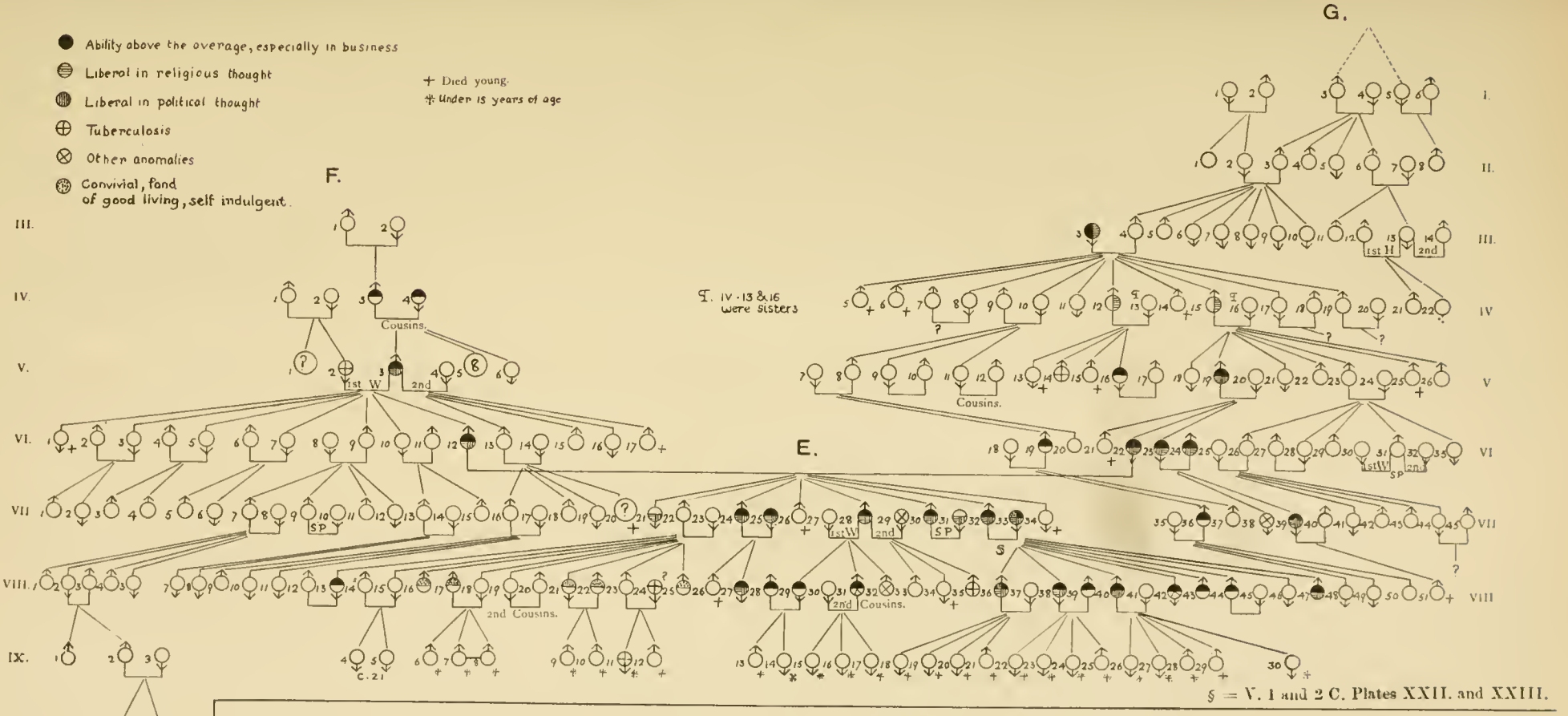


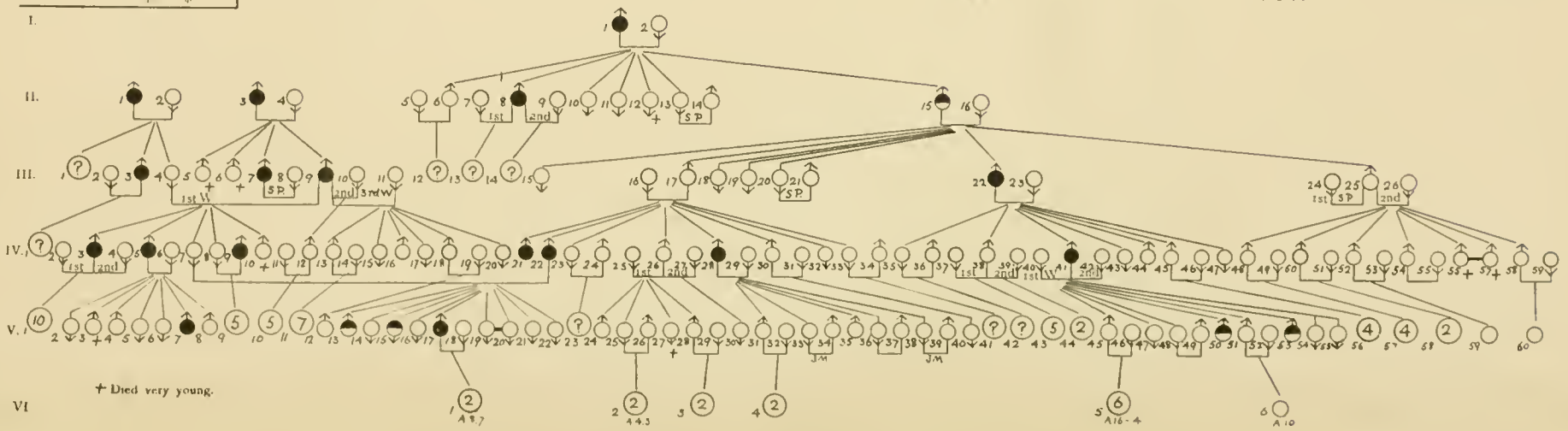
FIG. 192. E–G.



§ = V. 1 and 2 C. Plates XXII. and XXIII.

FIG. 193

COMMERCIAL AND LEGAL ABILITY.



UNIVERSITY OF LONDON

FRANCIS GALTON LABORATORY FOR NATIONAL EUGENICS

EUGENICS LABORATORY MEMOIRS. XI.

TREASURY OF HUMAN INHERITANCE

PART IV

SECTION XII *a*. HARE-LIP AND CLEFT PALATE

SECTION VI *γ*. DEAF-MUTISM

SECTION XIII *a*. CONGENITAL CATARACT

WITH 9 PLATES OF PEDIGREES AND 7 PLATES OF ILLUSTRATIONS

PLATES XXV—XXXIII PLATES G—M

PEDIGREES 193—372 *b*

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1910

CONTENTS OF PART IV.

(This will be replaced by a complete list of contents when the first volume is finished.)

ILLUSTRATIVE PLATES.

The next issue of the *Treasury of Human Inheritance* will be a double number (Parts V and VI) containing Pedigrees of Haemophilia, by W. BULLOCH, M.D.

PLATE XXIV. *Commercial and Legal Ability.* Fig. 193. I. 1, died aet. 59, successful manufacturer; started a type-founding business (*D.N.B.*). II. 1, died aet. 92, F.R.S., pioneer in meteorology (*D.N.B.*). II. 3, died aet. 79, calligraphist and author (*D.N.B.*). II. 8, died aet. 81, M.D., devoted his life to type-founding business and to the philological studies connected with it (*D.N.B.*). II. 15, dead, interested in locomotion; received a medal from government for an essay on employment of the poor in Ireland. III. 3, died aet. 76, F.R.S. (*D.N.B.*). III. 7, died aet. 68, physician and author of medical works (*D.N.B.*). III. 9, died aet. 75, barrister and leading member of the Society of Friends (*D.N.B.*). III. 22, died aet. 83; a great authority on bibliography (*D.N.B.*). IV. 3, living, 79, engineer; collector of literary curiosities. IV. 5, living, aet. 77, D.C.L., Litt.D., banker and historical writer. IX. 9, died aet. 75, distinguished architect. IV. 21, living, aet. 82, has maintained and extended a large manufacturing business and taken an active part in philanthropic work. IV. 22, living, aet. 81, D.C.L., F.R.S., member of the Permanent Court of International Arbitration at the Hague, formerly Lord Justice of Appeal. IV. 28, living, aet. 76, P.C., M.P. IV. 41, living, aet. 72, F.S.A., M.P., created baronet. V. 1, born 1855—67. V. 7, living, aet. 31; fellow and lecturer in modern history at an Oxford college. V. 9, born 1861—72. V. 10, born 1875—82. V. 11, born 1875—82. V. 13, living, aet. 48, barrister-at-law; distinguished university career, shut out from active life owing to ill health. V. 15, living, aet. 46, an active guardian of the poor and in other benevolent and social matters. V. 17, living, aet. 42, artist and writer on art. V. 43, eldest, born 1862. V. 44, eldest, born 1888. V. 50, living, aet. 40, barrister-at-law; distinguished university career. V. 53, living, aet. 36; distinguished university career. V. 56, eldest, born 1870. V. 57, eldest, born 1885. V. 58, eldest, born 1891. (*D.N.B.* = account of in *Dictionary of National Biography.*)

SECTION XII. HARE-LIP AND CLEFT PALATE.

BY H. RISCHBIETH, M.A., M.D., B.C. (Cantab.), F.R.C.S. Eng.

I. ANATOMICAL ACCOUNT.

Hare-lip, "Lippenspalte" (Cheiloschisis) or "Hasenscharte" (Labium leporinum) and "bec-de-lièvre" are synonyms. But no known form of hare-lip resembles closely the lip of the hare: see Plate H, Fig. 3[1].

Alveolar cleft, "Kieferspalte" (Gnathoschisis) may be associated with "Lippenspalte," frequently also with "Gaumenspalte" (Uranoschisis) or cleft palate.

"Wolfsrachen" (Rictus lupinus, Cheilo-gnatho-uranoschisis) signifies, in German, complete hare-lip and cleft palate. The origin of this term is not clear and its use is not quite satisfactory, for the wolf's palate and alveolus are not cleft; the name is, however, in general use in German medical language. Cleft of the soft palate alone is known as Staphyloschisis.

The two deformities, hare-lip and cleft palate are frequently associated, but either may be present alone. Hare-lip is a gap or cleft of congenital origin in the lip, as the result of want of proper union between its component parts. It is nearly always in the upper lip, very rarely in the lower. It generally occurs on the left side, but in some rare cases on the right, and it has been known to be median: see Plate J, Fig. 14, and Plate K, Fig. 18.

It may be unilateral or bilateral. Its extent varies. It may be, if the paradox be allowed, a mere linear scar running from the lip to the corresponding nostril

[1] References to the Plates throughout this section are intended to cover references to the Descriptions of the Plates, which it is essential that the reader should examine in conjunction with the photographs themselves.

(presumptive evidence of a hare-lip which has been healed *in utero*), a notch in the red part of the upper lip, or a broad cleft reaching to the nostril. The former conditions are called "incomplete hare-lip," the latter "complete hare-lip." In the case of complete hare-lip, the alveolar margin of the jaw is usually cleft as well; there may also be cleft of the palate. The nostril on the affected side is wider than on the other. The edge of the cleft on the lip is bound down to the bone, at least when the cleft is extensive, by a fold of mucous membrane. When the cleft of the lip is bilateral and the alveolus is divided, the division usually occurs along the line of normal junction of the praemaxilla and the maxilla on each side. Both the portion of the lip between the two clefts (the prolabium) and the praemaxillary bone are thus separated from their normal attachments, except above. They are, in such a case, together tilted forwards beneath the nose. The attachment of the praemaxilla above may be to the columna nasi alone, or it may be to the septum nasi: see Plates J and K.

In the normal individual the palate consists of two parts, the hard palate and the soft palate. The hard palate is a plate of bone of, roughly, semicircular form, placed in a horizontal plane. Of this semicircle the circumference corresponds to the alveolar margin of the upper jaw, and the chord placed posteriorly is free. It separates the cavity of the mouth from that of the nose. To the posterior border of this plate is attached, in the same plane, the soft palate, which is composed of a flat tympanum of fibrous tissue, like that of a drum, and the muscles which render it tense or lax (the other structures in it concerned with sensation etc. being here omitted). Its posterior border is free, and terminates in the middle line in a "queue"—the uvula. The soft palate can be elevated so as to close the posterior apertures of the nose completely. It is largely concerned in the function of articulation. The uvula in this respect is particularly concerned with the guttural articulations; such as that of "*r*" in many foreign languages. The simplest form of cleft palate involves the soft palate alone, which is divided in the middle line in a direction from behind forwards. The hard palate may also be divided in the same direction. This is usually to the left of the plane of the septum of the nose, but may be to the right of it; or there may be a cleft on both sides of that plane (bilateral cleft palate). Cleft palate may occur, in any of its varieties, with any of the varieties of hare-lip above enumerated, or it may occur alone: see Plates G and I.

An explanation of these conditions is to be found by reference to the development of the face and particularly of the nose and mouth. Into the earlier stages of that development it is unnecessary to enter, but the later stages may be briefly outlined[1]. In an embryo of about four weeks, the frontal tegmentum, or ectodermal covering of the fore-brain, has grown down in front of the latter. On each side of that frontal tegmentum the nasal depressions have appeared and the embryonic nasal areas can be recognised. Lower down, the facial part of that frontal tegmentum is raised into a transverse fold (the "muzzle fold") in the walls of which lie the embryonic nasal areas. The latter become formed into pits by the elevation of the fold about them,

[1] This account follows that given by His: Beobachtungen zur Geschichte der Nasen- und Gaumenbildung beim menschlichen Embryo. *Abhand. kgl. sächs. Gesell. d. Wissensch.* Bd. 27, 1901.

and thus there is a division into three—the mesial and two lateral frontal processes; above which is the frontal eminence. From the mesial and lateral frontal processes there are subsequently developed the mesial and two lateral nasal processes. Soon after this, another outgrowth from the frontal eminence occurs on each side below the others—the superior maxillary process. The mesial nasal process, or processus globularis, subsequently forms the septum of the nose, the praemaxillary bone, which bears the two central and two lateral incisor teeth and the prolabium. From the lateral nasal processes the lateral walls of the nose are developed.

From the maxillary processes are developed the superior maxillae, and, by a growth inwards from these, in a horizontal plane, the palate on each side. The primary palate is thus formed as a transverse bridge dividing the nasal cavity from the cleft of the mouth, and its closure begins as an adhesion between the approaching walls of the cleft. Subsequently this epithelial plate dissolves, and is replaced by a bridge of mesenchymal tissue. At about the fifth week the primary development of the palate is complete. It is by defect of growth and union on the part of these "processes" that the deformities hare-lip and cleft palate are produced. The lower jaw and lip is developed from Meckel's cartilage (the homologue of the first branchial arch of fishes) of either side and the tissues that cover this. By the junction of these in the middle line, in the manner of the above, the lower jaw and lip are produced. See Plate G, *j*, *k*, *l* and also *a* to *h*.

These clefts represent the persistence after birth of conditions normally present during intra-uterine life, but which have persisted owing to premature cessation of growth locally, i.e. owing to the 'arrest of development' of the early writers.

Concerning the precise manner in which this arrest of development is produced, it is occasionally held that in certain cases it is due to the influence of the amnion, that is, some cases of this deformity are supposed to be "amniogenic"[1]. In most, however, the precise manner of production of the defect is unknown. It can only be surmised to result from inherent tendency to defective growth or to some influence which is vaguely supposed to be exerted through the placenta[2].

The cases ascribed to amniogenic origin are accounted for in the following way[3]. The germinal layer raises itself as a longitudinal elevation from the vitelline sphere and extends horizontally under the developing rudiment of the encephalon and of the heart. The head end, on the lateral surfaces of which the branchial elements and first developmental elements of the face become visible between the 15th and 21st days, becomes free above in the longitudinal direction of the embryo, which is surrounded on all sides by the amniotic bladder. This, in which the embryo develops, is at one time tightly filled with fluid, at another time it envelops the embryo slackly, like a veil. At the beginning of the fourth week of pregnancy there is a strong ventral bowing of the head of the embryo—the cervical curvature. As a result, the

[1] For illustrative cases see Tables, Group B, p. 101 *et seq.*

[2] Any case in which there is resemblance between father and child in the defect seems excluded from this group. For possible illustrative cases see Tables, Group A, pp. 93–101.

[3] See His, *loc. cit.*

site of development of the face becomes closely pressed against the anterior wall of the chest. In this position the development of the face is completed. In the sixth week, the head gradually raises itself from this position. At this time the processes which form the face and palate are already half united.

The amnion at first arises from the lower border of the inferior maxillary process transversely above the rudiment of the heart, and surrounding what is subsequently the umbilicus. From this origin it extends closely over the fore end of the brain, and laterally over the areas of the branchial arches (also over the developing limbs). The face area and the area of development of the lips deeply bowed upon the breast, does not come into contact with the amnion, and so far the amnion cannot influence the development of the face.

During the period in which the cervical curvature persists, the amniotic membrane becomes dilated through the collection of amniotic fluid within it, and covers the inner surface of the uterus. By the development of the neck the face now becomes raised from the front of the chest, and is turned free towards the uterine cavity, and may thus come into contact with the amnion.

Epithelial rests or collections of cells of the same kind as those composing the amnion have been found in the cleft itself, or its neighbourhood, in certain cases of hare-lip. From the form and situation of the cleft, a relation of cause and effect of such "epithelial rests" with cleft formation has been inferred. In other cases, adhesions have been demonstrated between the amnion and the embryo, which it has been suggested by König (Bibl. No. 106) may have caused the defect in one or other of the following ways:

(1) The adhesion is attached to the embryo at the direct place where the cleft has remained.

(2) The adhesion is attached some distance from the cleft itself, upon the cheek. It acts by drawing the superior maxillary process towards the middle line, so that it does not reach forward far enough to unite with the inter-maxillary process. The cleft therefore remains open, and hare-lip results.

(3) The adhesion between the amnion and the embryo is attached to the embryo somewhere in the extremities. The band of adhesion stretches across one half of the face, and retards further development on that side, either by direct interposition, or by pressure, much as in the last case.

There is an additional, indirect, influence in some cases. Such cases are often associated with brain deformity, such as encephalocele, or hydrencephalocele. The malformation of the developing brain-rudiment resulting from the adverse influence of amniotic adhesions, retards the development of all the structures dependent upon it, the lateral and mesial nasal processes and the superior maxillary processes. Their development and union, therefore, cannot so well occur as in normal brains. Thus in them also, hare-lip and cleft palate are produced. Such is the outline of this theory. But an amniogenic origin is insufficient to explain completely the observed facts, even in cases in which amniotic adhesions are found (see the Tables, p. 101 *et seq.*), and in the majority of cases no adhesions are present (see the Tables, p. 93 *et seq.* and the accounts of family cases).

II. FREQUENCY IN MAN. OCCURRENCE IN LOWER ANIMALS.

(1) *Frequency in Man.*

Goring[1] has examined 1500 criminals and found amongst them no case of simple hare-lip, one case of hare-lip with cleft palate and no case of cleft palate without hare-lip. There were six cases of "deformed palate," but in none of these was the palate cleft. Pauline Tarnowsky[2] examined 150 prostitutes and found that 14, or 9·3 per cent., had cleft palate. Frobelius[3] examined 180,000 children in the Foundling Hospital of St Petersburg during 30 years. In 42 of these there was hare-lip with complete cleft palate. In 34 there was hare-lip without cleft palate. In 42 there was incomplete cleft of the hard palate, without hare-lip. That is to say, the deformity, in one form or another, occurred in 0·066 per cent. of this large number of children. Talbot[4] examined 1977 feeble-minded children without finding a case. Amongst 207 patients in an institute for the blind he found one case. He also examined 1935 deaf mutes and found two cases amongst them, i.e. about 1 in 1000, or 0·1 per cent. Knecht, according to Talbot, examined 1200 criminals and found cleft palate in 5·0 per cent. Amongst 495 criminals in the Illinois State Reformatory only one case was observed[4]. Amongst 1080 in the New York State Reformatory only one case was observed[4]. Langdon Down, according to Talbot[4], found that 0·5 per cent. of congenital idiots had cleft palate. Gresnor[4] found 9 cases amongst 14,466 children taken at random or 1 in 1607 (0·062 per cent.). From January 1st to December 31st, 1908, 67,945 cases of illness of various kinds in children of 7 years of age and under (of an age i.e. suitable for admission to the Children's Wards) attended the Receiving Room of the London Hospital. In this number there were 8 children with simple hare-lip, 19 with cleft palate alone, and 12 with combined hare-lip and cleft palate. That is to say, 39 individuals showed the deformity in one form or another. The 67,945 cases, however, do not accurately represent the number of individuals, being too great; for some doubtless attended more than once and how many these were cannot be stated. The exact total number of individuals cannot be determined, but the number 39 represents individuals. The percentage which these figures give (0·057) is too low, but furnishes an approximate figure of the frequency of the condition. This seems to agree with that of Frobelius, when the above qualification is considered.

The deformity is more common amongst the defective classes (deaf mutes, congenital idiots, prostitutes and criminals) than amongst foundlings and others. This is what might be expected, since it represents physical degeneracy, a fact which

[1] Goring, C.: Biometric Laboratory, University College, London, July 21st, 1909.

[2] Pauline Tarnowsky: *Étude anthropométrique sur les prostitutées et les voleuses*, Chap. vi. p. 41. Paris, 1889. Tarnowsky found amongst the 150 individuals of this class a high-arched, narrow palate 38 times and absence of lateral incisor teeth 10 times. In 15 of the 150 examined there was no physical defect, but 82·6 per cent. showed more than one. She found this percentage amongst other women to be: 14 in illiterate peasant women and 2 amongst the "educated" classes.

[3] 1864, see Bibl. No. 44.

[4] 1904, see Bibl. No. 127.

is more clearly shown than would otherwise be the case by its disproportionately frequent association with other defects, an association which is paralleled in other defective conditions (e.g. polydactyly in hermaphroditic stocks and deaf-mutism in albinotic stocks). The general percentage frequency of hare-lip and cleft palate children would be somewhat higher but for early deaths and therefore there is some difference between the number of these born and the number that live long enough to be included in the statistics of institutions. But even without early deaths the percentage would not be high, for gynaecologists do not report the condition as common amongst new-born children and early deaths are in modern times becoming of less and less frequency owing to the increasing care taken of such children in institutions.

Touching the frequency of various forms :

(1) The commonest variety of the deformity is unilateral cheilo-gnatho-uranoschisis. Thus in 270 cases, E. Müller[1] found it in 116. Gotthelf[2] in 56 cases found it 17 times, and Stobwasser[3] in 80 cases found it 15 times. The second commonest variety is unilateral cheiloschisis. E. Müller[1] in 270 cases found this 57 times, Gotthelf[2] in 56 cases found it 6 times, and Stobwasser[3] in 80 cases 12 times. The third in order of frequency is bilateral cheilo-gnatho-uranoschisis. E. Müller[1] in 270 cases 47 times, Gotthelf[2] in 56 cases 6 times, Stobwasser[3] in 80 cases 17 times. The other varieties are less common than these. It is unnecessary to state their exact order of frequency.

(2) The condition is more common in males than in females. Thus : E. Müller[1] in 270 cases found 170 males and 100 females. Fahrenbach[4] in 210 cases found 143 males and 67 females. Gotthelf[2] in 56 cases found 35 males and 21 females. Köllicker[5] states that in 277 cases of all kinds there were 153 males and 124 females ; that in 75 cases of all kinds in another series there were 45 males and 30 females ; and that in 132 cases of unilateral cleft there were 75 males and 57 females. Bartels[6] found in 20 cases of hare-lip healed *in utero* 14 males and 6 females.

(3) The cleft occurs more commonly on the left side than the right. Köllicker[5], in an analysis of 400 cases, found 271 unilateral and 129 bilateral clefts. Of the 271 unilateral cases the side was stated in 165 ; this was the left in 113 and the right in 52. Eigenbrodt[7], in 70 cases, found 55 unilateral and 15 bilateral clefts. Of the former there were 32 left and 23 right sided clefts. Bartels[6] found, in 15 cases of hare-lip that had healed *in utero*, 10 left and 5 right sided clefts.

There are many other statistics upon these points but it is unnecessary to give them. They are all in general agreement with the above figures.

(2) *Occurrence in lower Animals.*

Comparable defects have been observed in many species of mammals; it is possible that they also occur in vertebrates lower than these in the scale. In the Hunterian Museum of the Royal College of Surgeons there are specimens showing the condition in cattle, sheep and dogs ; in some of these specimens it is associated

[1] Bibl. No. 85. [2] Bibl. No. 81. [3] Bibl. No. 75. [4] Bibl. No. 109.
[5] Bibl. No. 71. [6] Bibl. No. 57. [7] Bibl. No. 80.

with other defects, such as anencephaly. Its occurrence in domestic animals and in wild animals born in captivity has been noted by many observers[1]. In the case of wild animals born in captivity the deformity is frequently associated with rickets. The influence of heredity in its causation has been shown in dogs by Bland Sutton[2], as well as by Meckel[3] and others. Whether the deformity occurs in wild animals born under natural conditions, or whether it is in them a new condition, only occurring when they are born in captivity, possibly as the result of bad environment and degeneration of the parents is uncertain. The evidence is insufficient for any conclusion to be drawn upon this point. In the Hunterian Museum there are specimens showing: (1) Complete congenital cleft of the lower jaw in birds (sparrow, cockatoo). (2) Congenital shortening, smallness or total defect of the cranio-facial axis, on one or both sides, in birds (chicken, pheasant, sparrow, duck) and fishes (salmon, trout, eel). The first of the latter conditions, congenital shortening of the upper jaws (micrognathia), is the normal condition in certain breeds of dogs (bull-dogs &c.), of cattle, of domestic pigeons and of fishes (carp). This condition is shown, combined with cleft palate, in a specimen in the calf. There are, however, no specimens of cleft palate in birds or fishes.

Congenital cleft of the lower lip and jaw[4] and congenital micrognathia occur in man, though very much less commonly than cleft palate. These three conditions, as the mode of development of the lips and jaws (essentially the same in all vertebrates) shows, only represent different phases of the same defect of development. In the development of these parts there seem to be two factors at work, (1) plastic or architectural, which moulds them to the shape they normally assume; (2) that which determines their growth in size, whether (1) has performed its work efficiently or not. Defect of (1) will produce hare-lip, cleft palate and cleft of lower lip and of face. Defect of (2) will produce micrognathia and defect of cranio-facial axis. Both (1) and (2) may be deficient. It is upon the nature of the association of the defect of these two factors that the exact form of the resulting deformity depends. In mammals defect of (1) is the more common occurrence. In lower vertebrates (2) or, (1) and (2) together would seem to be the more usual. It appears a reasonable hypothesis, though there are no specimens to support it, that in birds and fishes (1) alone may at times be defective, i.e. that cleft palate may occur in birds and fishes.

[1] E.g. lion and other cubs born in Zoological gardens. A. Keith, Royal College of Surgeons (Verbal Statement, July 21, 1909), Talbot, Bibl. No. 127.

[2] Bland Sutton: *Evolution and Disease*, pp. 189, 190–3, London, 1890.

[3] Bibl. No. 7, 1812.

[4] Meckel, Bibl. No. 7, cited p. 97 below. There is also a specimen in an infant in the Museum of St George's Hospital, London, and there is a case of cleft lower lip and jaw in an ass in the Museum of the R. C. of Surgeons.

III. HEREDITY IN HARE-LIP AND CLEFT PALATE.

The influence of heredity in the causation of this deformity was first shown in the year 1757, by Trew[1]. He described a family in which several members in two generations were affected. In 1795 James Lucas[2] described a family of four children, all with hare-lip. He did not look upon this association as evidence of heredity, however, for in discussing possible causes of the deformity he says: "The mothers could ascribe no cause for such unfortunate events and these may be deemed occurrences truly unsearchable." He appears to have looked upon the association in this case as an improbable chance combination. In 1805 Anna[3] published a family of this kind and when Meckel's[4] book upon Morbid Anatomy appeared, in 1812, several such were described in it, including those of Trew, Lucas and Anna. Meckel was the first observer to lay stress upon the hereditary factor in causation. In the course of the next few years family histories were published by Nicati[5], 1822, Steinhausen[6], 1836, Houston[7], 1842, and Canard[8], 1845. Roux[9], who in 1819 performed the first successful operation for the cure of cleft palate, has recorded several family histories, but he attached little importance to the part played by heredity in the causation of the deformity. I. Geoffroy-Saint-Hilaire[10] considered the evidence for an hereditary causation to be untrustworthy, but it is evident from his writings that he was ignorant of all the above recorded cases except that of Nicati. The first famous *surgeon* to attach due weight to the importance of the hereditary influence was Demarquay[11], 1845. He published several family histories and stated it as his opinion that the deformity is more frequently hereditary than had hitherto been supposed. About the same time Jardine Murray[12] in England and Trelat, Follin and Duplay in France[13] expressed similar views. From that time until now family histories have from time to time been recorded and lie scattered through the literature, so that over 200 of these now exist, but the question whether the condition is or is not "hereditary" continued to be debated in certain quarters almost up to the present day. Authoritative opinions have been expressed which have been formed, it would appear, largely without reference to recorded facts, upon personal observation and experience which, however wide, must have been limited. Conclusions drawn from such sources alone have therefore been to a large extent inaccurate. V. von Bruns[14] of Tübingen (1859) considered two causes to be possible, psychical influences (i.e. maternal impressions) and heredity. The latter he considered to be an infrequent cause, basing his opinion upon a very wide experience in which he had only once made sure of the influence of heredity. Sir Jonathan Hutchinson[15] expressed in 1881 the opinion that though often occurring in several members of a generation hare-lip is not hereditary; but since that time the record of cases showing hereditary origin has been very greatly increased in number. The view held by Darwin was as follows: "Although many congenital

[1] Bibl. No. 2. [2] Bibl. No. 5. [3] Bibl. No. 6. [4] Bibl. No. 7.
[5] Bibl. No. 11. [6] Bibl. No. 14. [7] Bibl. No. 18. [8] Bibl. No. 19.
[9] Bibl. Nos. 21 and 22. [10] Bibl. No. 13. [11] Bibl. Nos. 20 and 46.
[12] Bibl. Nos. 20 and 33. [13] Bibl. Nos. 20 and 127. [14] Bibl. No. 31. [15] Bibl. No. 70.

monstrosities are inherited, of which examples have already been given, and to which may be added the lately recorded case of Mr Sproule of the transmission during a century, of hare-lip and cleft palate, in the writer's own family, yet others are rarely or never inherited. Of these latter cases many are probably due to injuries in the womb or egg and would come under the head of non-inherited injuries or mutilations." (*Variations of Animals and Plants under Domestication*, p. 446.)

The hypothesis of psychical influences or "maternal impressions" as an important factor in the causation of the deformity held sway in the mind of Authority in certain quarters for long, the "face-saving" statements of the mothers concerning gruesome spectacles of various kinds observed during pregnancy being, in most cases, the only explanation forthcoming of the deformity in their children. Of such spectacles a favourite seems to have been the removal of a gaping fish from a pond; another was the sight of a vagabond of foreign nationality, Italian or gipsy for choice; but there were many others[1].

Of a case of this kind, the mother of which had herself had hare-lip, Sir William Fergusson[2], 1860, writes "One might have assured her that a glance in her mirror would have afforded her a sufficient explanation of the origin of her child's condition," and as is shown elsewhere in his records he was aware of the influence of heredity here; for he has said "I have seen four or five hundred cases of hare-lip and have operated upon three or four hundred. I have known the defect to occur in five generations."

Besides the two causes stated many others have been adduced for this deformity. Of mediaeval notions such as "the evil eye," terror &c., which are merely paraphrases of the idea of maternal impressions, nothing need be said. *Trauma* has been suggested as a cause of deformities and experimental evidence has been brought forward in support of the theory; it can only produce deformities in ova such as those of reptiles, birds, amphibia or fishes which develop outside the uterus, such ova as those employed for experimental purposes. Meckel and others advanced the theory that hare-lip and cleft palate are produced by developmental defects of the fore-brain, increased intra-cranial pressure &c., because of the frequent association with hydrocephalus; in no case however can these be shown to be anything more than associated conditions;

[1] The following case may stand as an example of the usual story of a "maternal impression":

A woman went before a police magistrate in London with her newly-born infant in her arms. She complained that its face was parrot-shaped. This regrettable circumstance was, she said, to be ascribed to the fact that during pregnancy a parrot belonging to a neighbour had alighted upon her shoulder, causing her great alarm; for, hence the remarkable physiognomy of her child. The information she wished to acquire from His Honour was whether she could bring an action for damages against the owner of the parrot for the grave injury that had been done by it to her child and to herself. (The parrot was, it appears, to escape scatheless.) The infant's face having been exposed to view, it was decreed, after some delay, that the mother's description of it was sufficiently accurate for practical purposes, but that no action for damages was advisable.

This was, probably, an infant having double hare-lip and cleft palate, with projecting os intermaxillare (praemaxilla) and prolabium. But whether this is true or not, the case may stand as a typical illustration of a "maternal impression."

[2] Bibl. No. 38.

no relation of cause and effect between them can be proved. The interposition of extremities, fingers and toes, which have occasionally been found in the clefts at birth has been looked upon as the primary cause of the defect; the hypothesis that such is secondary is more plausible. The tongue has been supposed to play a part in producing cleft of the palate, but on defective evidence. The adenoid tissues of the palate have been supposed to produce cleft palate. Their method of doing this is not clear, for the development of the palate is normally almost complete by the beginning of the third month, while the adenoid tissues do not appear until the fifth. Anomalous development of the teeth has been suggested as a cause of the persistence of the cleft. In some cases the teeth are normal. Development of the lips and palate is complete before the teeth appear. Defective dentition may therefore be a secondary or an independent association but cannot cause the cleft. In many deformities, including some cases of hare-lip and cleft palate, evidence of abnormality of the amnion, in the shape of bands and adhesions, is present. But the two conditions are not necessarily associated as cause and effect. The various ways in which these bands and adhesions are supposed to produce the deformity have already been dealt with (pp. 81, 82) and need no further description. The development of the face and of other parts depends as we have seen upon two factors (1) architectural, determining form, and (2) that determining increase in size. It is by defect of one or other or both of these energies that deformities are produced. There are the following possible explanations of these defects. They might arise *in utero* owing

(i) To an accidental influence, such as

(*a*) *Trauma.* This cannot produce any effect in mammals. Congenital deformities appear in the early embryo, to which efficient protection is afforded by the uterine walls and other parts. It is only in such ova as those of reptiles, birds, amphibians and fishes, in which development occurs outside the uterus, that trauma could be reasonably supposed to have any effect in producing deformation.

(*b*) *Psychical disturbance.* This signifies "maternal impressions." If such are a cause of deformities in man they must also be a cause of the same deformities in carnivora and domestic animals of various kinds, and may possibly also be their cause in birds and fishes. Owing to differences of character and environment, however, the nature of maternal impressions must be totally different in these different animals. Yet the deformities produced are the same.

(*c*) *Toxic influences.* Some toxic influence might reach the developing ovum from the maternal circulation (in mammals, or from other sources as well in the case of the ova of lower vertebrates). Of this nothing is known; but in such conditions as hereditary syphilis, where the nature of the toxic influence is known, there is defective growth in size but no deformity at birth.

(ii) To a formative defect on the part of the mother, e.g. an individual type of amnion, which may belong to her stock. In the latter case the deformity is hereditary through the mother. Cases in which the deformity passes from father to child, and these are frequent, cannot be accounted for in this way.

(iii) To a defective developmental determinant in either gamete which leads to an arrest in the development of the zygote.

Thus only two of the causes that have been suggested need consideration (1) an inner cause (inherent tendency), (2) amniotic influence, which again is secondary to some inner cause. Accidental influences need no further consideration. The cause of these defects lies in the family tendency, but it is only when the family is considered as a whole, in all its branches and when normal as well as deformed individuals are included in the records, that we shall begin to understand the mode of working of the hereditary influence. This fact has long been known to students of insanity, but in the case of hare-lip and cleft palate inquiry has usually gone no further than to ask how many relatives (usually in the direct line) showed this defect. This method assumes a fixed type of heredity, for which there is no warranty.

Tables showing the proportion of "hereditary" to other cases of hare-lip and cleft palate have been given by Hayman[1] and Schmitz[2] for cases in the German Clinics from 1878 to 1900. It does not appear what is meant by an "hereditary" case, nor how many normal relatives existed or were inquired about. The poorest of such data show 132 "hereditary" cases out of a total of 1857, or 7·1 per cent.; the following rather more reliable material gives 22·5 per cent.:

Fritsche	Total	52	"Hereditary"	10
Dissmann	,,	17	,,	5
Bein	,,	52	,,	9
Salomon	,,	21	,,	5
Friccius	,,	78	,,	22
Francke	,,	24	,,	4
	Total	244	"Hereditary"	55

This shows that improvement in investigation from this point of view raises the percentage of so-called "hereditary" cases. But the above percentage can only be looked upon as a minimum. Experience in other directions shows relatives wilfully denying heredity, unconsciously ignorant of it, or a complete absence of records even when the careful observer takes up the case.

For the student of heredity the *idée fixé* of the heredity of a single deformity α or of a deformity β as a unit character is to a considerable degree unscientific and must be largely modified, at least as data are increased. The origin of this idea lies in surgical classification, which exists for practical purposes. It is artificial, classifies conditions as is practically convenient but not necessarily as they occur in nature. If individual cases of hare-lip or cleft palate are considered it is to be noted that although in the majority the hare-lip and cleft palate deformity occurs alone, in a fairly large minority there are other deformities or defects, sometimes more than one[3]. If families[4] are considered, it

[1] Bibl. No. 124. [2] Bibl. No. 125.

[3] See the cases described by Demarquay (Bibl. No. 20, I. and III.), Jardine Murray (Bibl. No. 33), Cooper Forster (Bibl. No. 32), Roux (Bibl. No. 22, II.), Plicque (Bibl. No. 95), Thurston (Bibl. No. 136), Sutherland and Jackson Clarke (Bibl. No. 122), Bulloch (*Treasury*, Fig. 114 and Vol. I. p. 56), &c. Similar data, illustrating the same facts, in the case of another abnormality, viz. Hermaphroditism, are given in this *Treasury*, Figs. 106, 114, 119, 137, 143 of Plate XVI. and Figs. 152, 153 of Plate XVII.

[4] See footnote 3, above.

is to be observed that not all deformed individuals show the same deformity; for instance a case showing cleft palate plus another deformity had a brother with syndactyly; in the case of a boy with cleft uvula plus bilateral radius defect a relative of the mother had cleft uvula. It must be recognised that physical degeneracy is inherent in certain stocks just as other characters are; that in such stocks this is not always expressed in the same way in different individuals; one of the simplest and commonest expressions seems to be the hare-lip and cleft-palate deformity, in some stocks this seems to be the only one, in others it may be associated with other deformities in various ways. This seems to be as true of such stocks as of those showing, for instance, ability, in which, associated with ability, eccentricity, insanity, &c. may also occur, sometimes in the same, sometimes in different individuals.

Occasionally there is a general inheritance of physical degeneracy, as cases of multiple malformation show; such a case is the "siren" described on our p. 100, and there are many other cases of hare-lip with multiple defects which, like it, seem to represent a summation of this inherent tendency. Hare-lip or cleft palate seem to be only the α or ω of some symbolic writing; though it can be deduced that these symbols signify physical degeneracy it is mainly from knowledge derived from other sources that this can be done. In order to understand the sentence the whole language must first be learned. This at present we do not know, and study has largely taken the form of asking without that knowledge how α or ω came into use, a question which under such circumstances cannot accurately be answered. It is only by the study of a large number of complete family histories, in which all members, normal as well as deformed, are included, that any advance in the knowledge of the mode of production of congenital deformities and the mode of action of hereditary influence can be made.

IV. ASSOCIATION WITH OTHER DEFORMITIES.

Frequency. In the cases described with the plates this percentage is over 4·0 (imperforate rectum, morbus cordis, polydactyly, syndactyly, congenital sinuses of lower lips, &c.). E. Müller[1] found other deformities in 3·7 per cent. of 270 cases. Haug's estimate[2] is 3·6 per cent. of 285 cases, Gotthelf's[3] 8·89 per cent. of 56 cases. Stobwasser[4] found no other defect in 80 cases, Fahrenbach[5] many deformities, but gives no percentage estimate of his 210 cases.

Abnormalities. All these observers note the frequent occurrence of anomalies in the development and situation of the teeth. They also record the following conditions: angioma (several cases), pigmented naevus, melanotic sarcoma of praemaxilla, enchondroma of nasal septum, phimosis, encysted hydrocele of spermatic cord, unilateral inguinal hernia, bilateral inguinal hernia (several cases), umbilical hernia, ectopia vesicae, accessory auricles, microphthalmos, coloboma iridis, staphyloma retinae, strabismus, exophthalmos, hydrocephalus, meningocele, encephalocele, amputation of fingers, polydactyly, syndactyly, congenital talipes equino-varus, congenital stenosis

[1] Bibl. No. 85. [2] Bibl. No. 128. [3] Bibl. No. 81.
[4] Bibl. No. 76. [5] Bibl. No. 109.

of glottis by adhesions; one case showed the acquired deformity paralytic talipes equinus. The percentage 3·7 is probably too low. The cases from which the figures are derived are those attending clinics and they show for the most part only mild associated conditions. Cases showing gross deformities, such as many of those to be presently described, are for the most part either born dead or die soon after birth and do not figure in statistics of clinics. Though this is a small group it would raise the percentage of cases showing associated deformities above the figure 3·7 if it were included in the statistics. The above list does not accurately represent the variety of associated defects, nor their manner of combination, nor the relative frequency of this or that combination; the vast majority of cases of hare-lip and cleft palate (unassociated) have never been recorded and only a relatively small number of those with associated deformities are on record; nevertheless in cases of the latter kind that have been described some deformities occur more frequently than others and an estimate, which is probably broadly accurate, may thus be formed as to which are common and which rare; but no estimate of proportion between these and unassociated cases can be made. In many cases the associated deformities are multiple. Though usually those of defect they may occasionally be those of excess, e.g. polydactyly and accessory "auricles." Schorr[1] amongst 61 cases of cleft palate noted other deformities of the head in 7. These, however, possibly represent selected cases. Hayman[2] states that *congenital defect of radius* has been recorded in association with hare-lip and cleft palate 19 times, *polydactyly* in the same association 11 times, that in 1857 cases of hare-lip *accessory auricles* were found only once[3], though careful examination was made for associated deformities in all cases. According to Hayman[2] *accessory "auricles"* in association with cleft formation about the face have been recorded 22 times; in 9 cases the cleft was of the lip, in 3 of the palate, in 10 of the face. The family histories described by Jardine Murray[4] and by Richet[5] show hare-lip associated with congenital bilateral symmetrical submucous sinuses of the lower lip. Dun[6] has described four cases of this condition. In two the sinuses were bilateral and symmetrical, in one with papillary outgrowths; in two the sinus was single, in one in the middle line, in the other to the left of it (the latter case described by letter). Dun, quoting Clogg (*Brit. Jour. of Children's Diseases*, Feb. 1907) shows that this condition has now been described 39 times, usually, but not invariably, associated with hare-lip, sometimes with other deformities. A considerable proportion of this total occurred in related individuals. Cases have been described also by Bland Sutton, Arbuthnot Lane, Clutton, Ballantyne, Jardine Murray, Clogg and others in this country, and by Richet, Lannelongue, Demarquay, Fritsche and others on the Continent. Besides the conditions which are known as hare-lip and cleft palate there are several allied and rarer conditions about the face and neck which are too infrequent to be discussed here, e.g. lateral nasal proboscis, &c. For an account of these the reader is referred to Keith[7]. Any of these conditions may be associated with other deformities.

Since the influence of the amnion has sometimes been regarded as the cause of

[1] Bibl. No. 132. [2] Bibl. No. 124. [3] Friccius, Bibl. No. 111.
[4] Bibl. No. 33. [5] Bibl. No. 35. [6] Bibl. No. 134. [7] Bibl. No. 135.

hare-lip, cleft palate and other deformities, the following classification of associated deformities has been made in two groups, *A* and *B*. In no case in group *A* is there any evidence of an amniogenic cause of the deformity. In group *B* are placed cases showing the presence of bands or adhesions, which have by some been held to be the cause of the deformities with which they are associated. Of these cases it may for the most part be said that, granted that this influence may have caused one deformity, there is no evidence that it caused another (e.g. hare-lip or cleft palate), and in the cases showing multiple deformities most of these could not be produced in any such way. As regards many of these deformities, including hare-lip and cleft palate, adhesions may more plausibly be regarded as a secondary association than as a primary cause of deformity. If it be granted that amniotic bands and adhesions can in any case have produced hare-lip or cleft palate or other deformities (which are undoubtedly more usually produced by some inner cause, such e.g. as inherent tendency) such bands and adhesions are themselves secondary, and must be referred for their origin to some inner cause also. Thus in either case inherent tendency must be regarded as the primary cause of these deformities. Bands and adhesions may possibly act as secondary causes or may be a simple association without causal relation. Crede[1], in 10 cases of clefts of various kinds about the face, found 2 showing amniotic adhesions and 8 without these.

The mode of grouping of the associated deformities in the following list is arbitrary; it is employed for purposes of convenience. Deformities are grouped regionally, cases are placed (according to the main features of each) in what appears to be the order of relative frequency with which the deformity shown occurs in association with hare-lip and cleft palate; thus e.g. cases showing defects of brain and cranium are placed first. No attempt has been made in the Tables to give a complete list of all recorded cases of hare-lip and cleft palate associated with other malformations. Considering the number of these cases and the space available this was an impossibility, and it was unnecessary. The cases quoted are sufficient to show the variety of these associations and to indicate in a manner which is probably broadly accurate, which associations are common and which are rare. For the reasons given on p. 91, it does not seem possible to determine exact proportions at the present time.

[1] Bibl. No. 54.

Reference, Bibliography	General description	Abnormalities of brain, head or vertebral column	Special senses, especially eyes and ears	Trunk and viscera	Hands and feet	Upper and lower extremities other than these
133	Foetus with two faces united laterally above. On right face right H.L. and C.P. On left face left H.L. and C.P. One body and set of extremities.	Anencephaly.				
94	Two faces united laterally, &c. as above. H.L. and C.P. as above.	Anencephaly.	Cyclopism.			
12	A double female monster, heads united at vertex and occiput. Left H.L. and C.P. on smaller individual. Left oblique facial cleft extending nearly to inner canthus on this individual.					
4	Double oblique facial cleft.	Anencephaly.				
17	H.L. and C.P.	Anencephaly. Defect of 4 cervical vertebrae, only 3 present.				
17	H.L. and C.P.	Anencephaly. Only 3 cervical vertebrae. Laminae of all vertebrae as far as last lumbar lacking.				
17	H.L. and C.P.	Anencephaly.		No suprarenal bodies. Heart partially transposed.		
17	H.L. and C.P.	Anencephaly.		Congenital hernia of diaphragm on left side. No pericardium. Only one umbilical artery. A very small thymus.		
15	Left sided H.L. and C.P.	Partial anencephaly, or pseudo-encephaly.	Eyes bordering on root of nose. Optic nerves rudimentary.	Imperforate rectum. Recto-vesical fistula. No urachus. Only one umbilical artery.	Only 3 metacarpal bones each hand. Carpus correspondingly deficient. 3 digits on left hand. 1 digit on right hand, bifid at distal extremity. 3 phalanges to each digit.	Partial radius defect both forearms. Muscles and nerves of forearms show corresponding deficiencies.

GROUP A, *continued*

Reference, Bibliography	General description	Abnormalities of brain, head or vertebral column	Special senses, especially eyes and ears	Trunk and viscera	Hands and feet	Upper and lower extremities other than these
137	Median H.L. Bengali, aged 20 (see Plate K, Fig. 18).				6 fingers and 6 toes each side.	
137	Median H.L. Bengali, aged 5; brother of last.				7 fingers each hand and 6 toes left foot.	
Treasury, Fig. 114, p. 56. (Matthes' Case.)	H.L. and B.P. in two Hermaphrodites, ?sisters.	Hernia cerebri and occipital meningocele in both.		Partial transposition of viscera in both. Urethra no external opening, urine passed through a patent urachus in both. Testes undescended in both.	Polydactyly, hands and feet, in both.	
133	H.L. and C.P.	Hydrocephaly				
133	H.L. and C.P.	Hydrocephaly.				
133	H.L. and C.P.	Hydrocephaly.				
71	Double H.L. and C.P.	Hydrocephaly. Sacral spina bifida.			Doubling of little fingers and toes, both sides. Left congenital talipes equino-varus; right congenital talipes valgus.	
49	H.L. and C.P.	Hydrocephaly. Sacral spina bifida (meningocele).		Horse-shoe kidney.	Left hand only 4 digits. Congenital talipes valgus both feet.	Left radius rudimentary. Left ulna curved. Both hips ?dislocated. Knees flexed. Both tibiae curved. Lateral dislocation of patellae.
104	Very severe H.L. and C.P.	Hydrocephaly. Bones of vault of cranium completely lacking on both sides. Occipital hernia cerebri. Asymmetry of bones of base of skull.	Olfactory nerves and right optic nerve and chiasma lacking. Right eyeball rudimentary. No cilia. No eyebrows. Left eyeball normal.			
3	Bilateral oblique facial cleft.	Hydrocephaly.		Ventral hernia. Defect of ribs.	Defect of	extremities.
1	Bilateral oblique facial cleft. C.P.	Parietal hydrencephalocele.				
34	Bilateral oblique facial cleft.	Frontal encephalocele.				
43	Left oblique facial cleft. Cleft alveolus.	Occipital hydrencephalocele.				
55	Left H.L. Left oblique facial cleft.	Encephalocele.				
88	Right H.L. Left oblique facial cleft.	Hydrencephalocele (fronto parietal).				

88	Right H.L. and C.P. Left oblique facial cleft. Median cleft of lower jaw.	Two occipital encephaloceles.		A cystic appendage of vulva, the size of a hazel nut.	
88	Bilateral oblique facial cleft.	Hemi-crania. Encephalocele.	Ectropion of right upper lid. Cleft of right inner canthus.		
72	Bilateral H.L. and C.P.	Hernia cerebri.			"Deformity of hands and feet."
78	H.L. and C.P.	Meningocele.		Congenital microgastria.	
45	Bilateral H.L. and C.P. (Child born at full term.)	Hydrops ventriculi. Defect of olfactory lobes. Thickening of pia mater over anterior part of cerebrum. Synostosis of frontal bones at birth or only one centre of ossification.	Microphthalmos. Optic nerves normal. 3 small accessory auricles on left side.	Both testes undescended. Left kidney hydronephrotic. Only one umbilical artery.	
45	Large median cleft of palate. No praemaxilla.	Cranium bifidum totale. Encephalocele. Occipito-cervical spina bifida (myelocele).	Right microphthalmos. Left eye almost lacking. Left ear no ext. meatus and small deformed auricle. Right 3 accessory auricles.		
45	Bilateral H.L. and C.P.	Small round skull prominent in frontal region. Cerebrum not divided into lateral hemispheres. Lateral ventricles continuous anteriorly; marked hydrocephaly.	No olfactory nerves. No left optic nerve. Microphthalmos left. Defective left tympannic cavity, no ossicles. 2 accessory auricles on left side.		
133	H.L. and C.P.	Microcephaly. Cebocephaly.			
71	Bilateral H.L. and cleft of alveolus, defect of praemaxilla and vomer.	Microcephaly.			Supernumerary little toe, left foot. Supernumerary little finger, each hand.
114	Bilateral H.L. and C.P. A grooving of skin from ears to angles of mouth, double on left side, single on right.	Microcephaly.			Partial amputation of right thumb.
59	H.L. and C.P.	Small skull, without fontanelles; sutures united. No falx cerebri. No falx cerebelli. Pia mater adherent. Cerebrum very small. Not divided into lateral hemispheres. Bones of base ossified and united.			

GROUP A, *continued*

Reference, Bibliography	General description	Abnormalities of brain, head or vertebral column	Special senses, especially eyes and ears	Trunk and viscera	Hands and feet	Upper and lower extremities other than these
66	C.P.	Foetal synostosis of bones of base of skull. (Trigonocephaly.)				
66	C.P.	do.	Microphthalmos. Smallness of palpebral fissure.			
66	C.P.	do.	Rudimentary condition or a trophy of both optic nerves.			
133	H.L. and C.P.	Cranial asymmetry.				
5 *bis*	Bilateral H.L. and C.P.				Syndactyly both hands. ?? "Amputation" III left at metacarpophal. joint or ? syndactyly of digits. Right foot: ? defect of II and III metatarsal bones and digits or ? syndactyly. Left foot: ? syndactyly or ? defect of II and III metatarsal bones and digits. (Date 1804.)	
133	H.L. and C.P.	Asymmetry of bones of face.				
58	Left oblique facial cleft. Left alveolus indented. Palate irregular in shape.	Left temporal and frontal bones, maxilla and zygoma smaller than right.	Coloboma of left upper eyelid, extending upon forehead as far as hair margin.			
10	H.L.	Micrognathia (lower jaw).	Microphthalmos.			
71	Bilateral H.L. and C.P. Defect of praemaxilla.		"Defect of eyes."	"Stunted penis."		
134	Left H.L. and C.P.		Bilateral coloboma iridis, without coloboma of eyelids.	Hypospadias of glans penis.		
75	Bilateral H.L.		Bilateral coloboma iridis.			
87	Bilateral H.L. and C.P.		Bilateral coloboma iridis and of lower eyelids. A small tumour at corneal margin of each eye at canthi. (? Dermoid cysts.)			

120	Median cleft of jaw, defect of praemaxilla almost complete. Palate asymmetrical. Median cleft of nose. Right oblique facial cleft. Right macrostoma.		Coloboma both lower lids, ectropion both upper lids.			
53	Median H.L. and C.P. with cleft of alveolus.		Congenital cataract, both eyes.		Supernumerary digits on each hand.	
62	8 months' foetus. Bilateral H.L. and C.P.				Left club hand, four fingers, no thumb.	Defect of left radius.
63	Full term foetus. Left H.L.			Defect of ventricular septum of heart.	Both hands without thumbs. Left club hand.	Partial defect of left radius.
71	Right H.L. and C.P.				Club hands; the left without a thumb. Congenital talipes valgus, both feet.	Defect of both radii; forearms shortened. Defect of both fibulae.
71	Bilateral H.L. and C.P.			Hypospadias.	Right hand no thumb, no index finger. Left hand no thumb.	Bilateral defect of radius.
71	Median H.L.				Club foot. Doubling of great toes and little fingers.	Defect of fibulae.
71	Left H.L. and C.P.	Sacral spina bifida, i.e. defect of laminae of vertebrae.				
71	Left H.L. and C.P.	Sacral spina bifida.				
71	Right H.L. and C.P. Macrostoma.	Spina bifida.				Congenital defect of patellae. Rudimentary right lower extremity.
7	H.L. Cleft of lower lip.	"Very imperfect development of the vertebral column."				
8	Family of dogs. H.L.					Ectromelia; fore limbs lacking.
7	Bilateral H.L.			Uterus bicornis.	6 fingers and 6 toes on each side.	
7	Bilateral H.L. and C.P.			Uterus bicornis. Meckel's Diverticulum of small intestine.	6 fingers and 6 toes on each side.	
7	Two other cases like the last.			Uterus bicornis. Meckel's Diverticulum.	6 fingers and toes, each side.	
				Uterus bicornis. Meckel's Diverticulum.	6 fingers and toes, each side.	
71	Left H.L.; double C.P.				6 fingers and 6 toes on each side.	
71	Bilateral H.L. and C.P.				Doubling of little finger on left hand and of little toe on right foot.	

GROUP A, *continued*

Reference, Bibliography	General description	Abnormalities of brain, head or vertebral column	Special senses, especially eyes and ears	Trunk and viseera	Hands and feet	Upper and lower extremities other than these
71	Bilateral H.L. and C.P.				Doubling of little fingers and toes, both feet.	
71	Bilateral H.L. and C.P.				Doubling of little finger on left hand. Left congenital talipes equino-varus.	
71	Bilateral H.L. Cleft of alveolus. Defect of praemaxilla.				Both little fingers doubled.	
71	Bilateral H.L. and C.P.		"Deformity of external ear."			
113	Bilateral macrostoma. Fistulae of cheeks.		Small and deformed auricles on both sides. On right side 3 small accessory auricles containing cartilage.			Congenital dislocation of right hip.
9	A lamb and a pig each with C.P.		Each had double or cleft ears.			
134	H.L. or C.P.	Submucous sinuses of lower lip; usually bilateral and symmetrically placed about the middle line; occasionally single. 39 cases of this condition recorded, usually with H.L. and C.P. Nature and origin of this condition uncertain. Many cases show hereditary influences in the matter of both abnormalities.				
7	Calf with total defect of hard palate. Micrognathia (lower jaw).		Cleft of mouth prolonged through temporal bone and auricles. These divided into a large upper and small lower portion.			
42	C.P.		3 accessory auricles on each side (contained cartilage). Left middle ear—ossicles deformed, incus and stapes irregular and deformed.	Both kidneys on right side. One short and thick just below the normal one. Left suprarenal in normal position on left side. Ureters of both kidneys divided, but inserted into bladder normally. Only one umbilical artery.		

107	Left H.L. and C.P. Maldevelopment of left side of nose.		?"Accessory auricle" between mastoid process and occipital crest.			
112	Right H.L. and C.P.	Hypoplasia of left petrous bone. Labyrinth normal, but completely shut off by bone from tympanic cavity (which was rudimentary) on left side. Eustachian tube present, but did not communicate with this rudimentary tympanic cavity. Tympanic ring and tympanal membrane defective. Mastoid antrum deficient.	Deformity of right auricle, which was very small and nearly occluded external meatus. "Accessory auricle" on right side. Ossicles deformed.			
108	Left H.L. and C.P.		Complete defect of auricle and external auditory meatus. Auricle represented by two small cartilaginous nodules.	Horse-shoe kidney. Left ureter double. Right and left unite just before entering bladder. Heart, interventricular septum incomplete. Pulm. art. arises from aorta. Arteries to head and arms arise from right ventricle. Other abnormalities.	Abnormalities of joints of hands. Congenital talipes calcaneo-valgus, both feet. Abnormalities of the	Both forearms deformed. No right radius, ulna curved. Left elbow joint, defective movement. muscular system.
61	Total defect of palate.	Micrognathia (lower jaw). Only left mandibular joint present. Turbinates and lateral walls of nose defective.	Auricles deformed. Only lobule present on either side. No tympanic membrane. No external meatus but a blind diverticulum. No ossicles. Tympanic cavity rudimentary on each side. Promontory and foramen rotundum present, but stapes and foramen ovale lacking. Only on left side did Eustachian tube unite pharynx to middle ear.		No thumbs. No first metacarpal bones. Only 4 bones in each carpus.	Scapulae small. Right showed defect of a large part of infraspinous portion. Shape of articular surfaces of gleno-humeral joints reversed. Both humeri small. Right forearm only one bone (? radius). Cubitus valgus. Left forearm, both bones deformed.
		←	Several other	defects.	→	
52	Left macrostoma. On both upper and lower lips fine but marked depressions at sites of normal commissures.	Facial asymmetry (left side of face smaller than right).	Two large ? accessory auricles.		A brother, 3 years old, webbing of several toes on both feet.	

GROUP A, *continued*

Reference, Bibliography	General description	Abnormalities of brain, head or vertebral column	Special senses, especially eyes and ears	Trunk and viscera	Hands and feet	Upper and lower extremities other than these
71	Bilateral H.L. and C.P.			Hernia of diaphragm.		
71	Bilateral H.L. and C.P.			Vasa omphalo-mesenterica.		
71	Bilateral H.L. and C.P. Defect of praemaxilla and vomer.			Inter-ventricular septum of heart incomplete.		
32	C.P. in several members of family.			Imperforate rectum in several members of family.		
123	H.L. First child. No others had then been born.	Defective development of gums.		Congenital heart disease.	6 digits on each hand.	Shortening of all long bones of extremities.
122	H.L.	Defective development of gums.		Congenital heart disease.	6 digits on each hand.	Shortening of all long bones of extremities.
122	A brother of last; H.L.			Cyanosis of lips, nose and ears since birth. Died aged 9 months of heart disease and congestion of lungs.	6 digits on each hand.	Shortening of all long bones of extremities.
84	8 months' foetus. A case of siren-formation (mermaid-like monstrosity). Bilateral H.L. and C.P.	Facial asymmetry. Lumbar portion of vertebral column defective. Several vertebrae lacking, others deformed. A broad thick short stump in lumbar region attached to buttocks (roughly fin-like; ? rudiment of second lower extremity).	Left ear no lobule. Double microphthalmos. Right palpebral fissure smaller than left	Trunk shaped like inverted egg. Abdomen asymmetrical. Umbilicus to left of middle line. No external genitals. Anal orifice, rima and nates completely lacking. A pigmented area, uncovered by epidermis on left lower abdominal wall. Pelvis small, cartilaginous, abnormally movable on vertebral column. Auricular septum of heart deficient. One pulmonary vein. Pulmonary artery abnormal; divided into 3, the middle, biggest = patent Ductus Botalli; this represents pulmonary artery. Both lungs collapsed, airless. Right only 2 lobes. Kidneys represented by 2 membranous sacs. No suprarenal bodies. No ureters. No urinary bladder. No pancreas.	Syndactyly IV and V fingers of right hand. No feet.	Both arms and hands perfect, except for above right syndactyly. No legs (or only a rudiment of one). A single lower extremity the rudiment of both fused (only one femur). Movable on trunk, but not by a joint. Cone-shaped with base upwards. A rudimentary knee joint but with flexor surface forwards (i.e. reversed).
			Several other	defects		

83	Right H.L. and C.P.	Teratoma sinus frontalis.				
74	C.P.	Fibro-myomatous tumour of tongue.				
74	Right H.L.	Periosteal tumour of alveolus, cystic; partly composed of erectile tissue.				
115	Median H.L. and C.P. Cleft of nose.	In nasal cleft a cystic tumour (striated muscle and fibrous tissue, &c.)				Left forearm defective except in upper fourth. On stump 2 appendages, one of skin, one containing a phalanx-like bone united to that of forearm.

TABLE OF ASSOCIATED DEFORMITIES. GROUP B.

25	Third month embryo. Cleft of face from inner canthus to angle of mouth.	Membranous adhesions between forehead and placenta, about which adhesions spinal cord wound spirally.				
25	Full term child. Bilateral H.L. and C.P.	Very large hernia cerebri, to which placenta and amnion adherent.		Ectopia cordis. Heart in middle line, exposed in a broad cleft from upper thorax to umbilicus Heart globular, the shape of auricles, apex defective. No pericardium.	Amputations of digits of right hand. Adhesion to distal extremity of middle finger.	
25	C.P. A large oblique cleft of face, extending from forehead to upper lip, through inner angle of left orbit.	Acrania. Hernia cerebri. Nose proboscis-like, with very small nostrils.	Both inner canthi ununited. Bilateral microphthalmos. Left eyelids defective.	Partial defect of thoracic and abdominal wall on right side; a partial hernia of abdominal contents, covered by peritoneum showing in this defect. Umbilical cord winds round the tumour this forms.	Left congenital talipes equino-varus. A pseudo-membrane from II to IV toes of right foot; inserted into distal extremity of IV transversely.	Whole of right upper extremity lacking. Whole of left forearm lacking.
105	Bilateral H.L. and C.P.	Defect of bones of vault of skull. Only base present. Hernia cerebri; amnion adherent over it.			Bilateral congen. talipes equino-varus. Only thumb separate on left hand; four other digits webbed, but bones separate from one another. Fingers shorter than on right hand.	

GROUP B, *continued*

Reference, Bibliography	General description	Abnormalities of brain, head or vertebral column	Special senses, especially eyes and ears	Trunk and viscera	Hands and feet	Upper and lower extremities other than these
30	Bilateral H.L. and C.P.	Defect of most of soft parts and bones of cranium. No parietal bones; parts of frontal, temporal and occipital present. Rudiments of brain tissue present. Nerve origins normal. Hydrencephalocele. Placenta adherent to this.				
82	Congenital oblique cleft of face and nose.	Hemicephaly. Hydrencephalocele, to which amniotic adhesions attached. Double falx cerebri, &c. Gross defects of cerebrum.			Right foot II and III digits amputated (terminal phalanges). IV digit, marked constrictions (partial amputation). Right hand, II digit amputated at base of second phalanx. Left hand, thumb through proximal phalanx, II, III and IV at base of first phalanx. Stumps of these fused into one. Traces of adhesions. Fifth finger normal.	
54	Bilateral H.L. Left oblique facial cleft.	Amniotic adhesions to margins of clefts. Defect of left frontal bone. Hernia cerebri. Amniotic bands adherent to this.	Orbits small. Eyelids fused externally.	Right floating ribs rudimentary. Ventral hernia. Abdomen shortened so that xiphi-sternum and symphysis nearly in contact. A finger-like appendage 1½ cm. above and behind anus, bearing a nail. A thick bridge of skin attached to right thigh. Placenta and amnion attached to back of child in a bridge-like manner at points 17 cm. apart.	Amniotic bands adherent to left wrist. Right little finger only 2 phalanges, but bears a nail. Syndactyly of IV and V left foot, all nails rudimentary. Syndactyly of II and III and of IV with V, right foot. All nails rudimentary.	Right arm defectively developed. Forearm ⅓ length of left. Flexed and adducted. Thighs abducted. Left knee over left shoulder, left leg dislocated at knee and almost completely webbed to back of left thigh. Left foot over left buttock dorsum to front. Right leg lies across symphysis pubis, plantar surface to front.

73	Right H.L. and C.P.	Hydrencephalocele. Cranium contained much fluid. Bones very movable and sutures wide open.	Eyes asymmetrically placed. Very small left palpebral fissure. No left eyeball. Eyes asymmetrically placed. Very small left palpebral fissure. No left eyeball.	No right hand. Constrictions, nearly to bone, on second phalanges of all digits of left hand. Thin adhesions on thumb and index finger. Right foot, I and II shortened and without nails. I united to II; III, IV and V also united. Marked constrictions on III, IV and V.	
94	H.L. and C.P. Oblique facial cleft.	Hernia cerebri. Amnion adherent to this.	Coloboma of lower eyelid.		
36	H.L. and C.P.	Hernia cerebri. Amnion adherent to this.		Numerous adhesions about the	and constrictions extremities.
41	H.L. and C.P.	Hernia cerebri. Amnion adherent to this.		Numerous adhesions about the	and constrictions extremities.
16	Bilateral oblique facial cleft.	Hydrencephalocele. Amniotic adhesions to the head.			
50	Bilateral H.L. and C.P. Bilateral oblique facial cleft, from angles of mouth to inner canthi.	Hydrencephalocele. Cleft of face extends upon forehead at fronto-parietal junction on both sides. Temporal and parietal bones and superior maxillae deficient in development.	Absence of eyelids on both sides.	Complete syndactyly of III and IV, both hands. Right foot, terminal phalanx of I amputated. Left congenital talipes equinovarus.	
48	H.L.	Cranium small. Frontal, parietal, temporal bones almost completely lacking. Two herniae cerebri covered by normal skin, but amnion adherent to this in places.		Terminal phalanges of III, IV and V of left hand amputated. Terminal phalanx of right V amputated. Remnants of amniotic adhesions. Marked constriction of extremity of little finger of right hand. Feet normal.	A circular constriction, lower third of left leg, bones here interrupted in their continuity. Right leg showed an excavation on internal surface near ankle, with considerable loss of tissue.
56	Bilateral cleft of alveolus. No palate. Bilateral oblique facial cleft, reaching to orbit on left side.	Small cranium. No fontanelles palpable. 3 Herniae cerebri, nasal, parieto-occipital, orbital.	Bilateral exophthalmos. No eyelids on either side.	Right hand, all fingers webbed, thumb free. No nail on V. Left hand, middle finger shortest, middle and index united by a web attached to terminal phalanges. Left foot normal. Right foot, I—IV webbed. Nail of V (free) very small.	

GROUP B, *continued*

Reference, Bibliography	General description	Abnormalities of brain, head or vertebral column	Special senses, especially eyes and ears	Trunk and viscera	Hands and feet	Upper and lower extremities other than these
103	Rudimentary H.L. Double oblique facial cleft.	Hydrocephaly.	No eyeballs. Coloboma of left lower eyelid. Several epithelial adhesions to cleft.	Ventral hernia. Skin appendages over sternum containing cartilage.	Terminal phalanx of left thumb doubled, polydactyly.	Right clavicle and scapula lacking. No right upper extremity. In its place a skin excrescence with amniotic rests at apex.
88	Bilateral oblique facial cleft, healed *in utero*.		Small epithelial excrescences on conjunctiva of left eye at inner and outer canthi.		Left hand. Amputations of II and III digits through middle phalanges, of IV and V through terminal phalanges. Right hand. IV digit incurved at distal extremity and somewhat shortened. Right foot, I lacking, II and III partially amputated.	
37	Right H.L. Bilateral oblique facial cleft.	Several depressions on forehead, to which pseudo-membranous strands attached.			Fingers and toes show membranous strands. Syndactyly. Patches on palm devoid of epidermis. Amputations. Right congenital talipes equinovarus.	Several hollow patches on forearms, as it were eroded.
92	Left H.L. and C.P. Left oblique facial cleft. Right horizontal facial cleft.	Amniotic adhesions at borders of clefts.	Left coloboma of eyelid. Auricles deformed, horizontally directed, and abnormally low in situation.		Congenital talipes equinus of both feet.	
74	Complete congenital cleft of lower lip and jaw. Fistula from mouth to cleft.	Adhesions between tongue and bones of mandible. Adhesion between jaw and sternum, very thick; caused flexion of head on trunk.				

60	Bilateral C.P.			Right hand; no metacarpal bones. Only one carpal bone (?trapezium). 2 digits (thumb with 2 phalanges and little finger with 3). Inner side of right foot touches internal condyle. Sole directed inwards.	Amputation of left forearm (through lower end of humerus). No right tibia. Right fibula articulates with outer surface of femur. Bent upon itself at middle nearly at right angles.
				Abnormalities of muscles, vessels and nerves.	
67	Right H.L.		3 accessory "auricles" on left side. Overlapping of sclera on left cornea.	No right thumb.	Congenital defect of right radius.
69	Right H.L.	A long cicatricial adhesion from right temporal region to right upper eyelid producing eversion of the lid (ectropion).	Right ectropion. Lower and upper lid of right eye united by a similar adhesion.	Amputations: Right hand; III and IV digits through middle of 1st phalanx, V through first interphalangeal joint. Left hand; all except I at first inter-phalangeal joint. IV depressed beneath the others. Stumps webbed together. Right foot; I, II and III at first interphalangeal joints. Stumps webbed. Left foot; all through terminal phalanges at roots of nails. Not webbed.	A circular deep furrow just above ankle, on left leg, constricting the limb.
106	Right H.L. and C.P. Nose and praemaxilla deflected to left.			II and III of both hands amputated, III and IV webbed. Deep constrictions on II toes. Syndactyly of III, IV and V both feet.	
124	Left H.L. Cleft of left alveolus. Born with left hand in cleft, held there by a firm adhesion surrounding index finger and bridging middle and ring fingers.		2 left accessory auricles.	Left hand, all fingers shortened. Constrictions on II, III, IV and V fingers. Phalanges of deficient growth. Right congenital talipes equinovarus. Constrictions on II, III and IV toes. Left foot, syndactyly of I, II, III and IV.	Right genu valgum.

GROUP B, *continued*

Reference, Bibliography	General description	Abnormalities of brain, head or vertebral column	Special senses, especially eyes and ears	Trunk and viscera	Hands and feet	Upper and lower extremities other than these
110	Partial C.P.		Small and contracted right auricle. Right accessory "auricle."		Bilateral congenital talipes calcaneo-valgus.	
110	Bilateral H.L. and C.P.		3 left and 1 right ?accessory "auricles" or ?rests of amniotic adhesions.			
106	Left H.L. and C.P.		Lagophthalmos (left). Left ectropion, upper lid drawn upwards and outwards, lower downwards and outwards, by amniotic strands. An excrescence in upper and outer quadrant of bulbar conjunctiva and one at outer canthus (left eye). Canthus externus nearer auricle than on right side. Both auricles flattened and contracted. No external meatus on either side.			
106	H.L.	Small skin appendages on face. ?Accessory "auricles."			Syndactyly. Small skin appendages.	
110	Right H.L.				Right hand; digits II and III amputated at metacarpo-phalangeal joints. Stump of III fused with IV. Constrictions between I and II. Left hand; II and III amputated at 1st inter-phalangeal joint. Stumps of II and III fused with IV. Both feet, II lacking; III, IV and V webbed together.	
106	Bilateral H.L. and C.P.	Rests of amniotic adhesions both cheeks. ?Accessory "auricles."				

In Groups A and B there are 122 cases, 91 in Group A, which showed no traces of amniotic adhesions, and 31 in Group B, where such were seen. In the 122 cases there are the following numbers of associated deformities:—

a Abnormalities of brain, cranium and other parts of head	*b* Defects of the vertebral column (spina bifida, etc.)	*c* Abnormalities of eyes, eyelids, etc.	*d* Abnormalities of ears	*e* Congenital "amputations"	*f* Defect of bones other than by congenital "amputations"	*g* Abnormalities or defect of viscera, etc.
Total 71* Anencephaly, 8 Hydrocephaly, Hernia cerebri, Meningocele } 36 Microcephaly, 6 Foetal synostosis, 3 Micrognathia, 2 Other cranial and facial defects, 12 New growths, 4 * To these add submucous sinuses of lip. Of this condition there are 39 cases usually with H.L. or C.P., but exact number is uncertain.	*Total* 10	*Total* 48 Microphthalmos, 12 Coloboma or lack of union of inner canthus, 11 Dermoids or (?) Dermoids, 5 Congenital atresia of palpebral fissure, 4 Defect or rudimentary condition of optic nerve, 4 Ectropion, 4 Displacement of eyes, 2, other than exophthalmos, 1 Congenital cataract, 1 Cyclopism, 1 Other conditions, 3	Defect, 13 Accessory "auricles," 12	*Total* 19 + 1? Polydactyly *Total* 21 Syndactyly *Total* 14 Talipes (congenital) *Total* 11	*Total* 20 Radius, 9 Fibula, 1 Tibia, 1 Patella, 1 Other bones, 9	*Total* 62 Heart, 10 Kidney, 6 Suprarenal body, 2 External genitals including hypospadias and undescended testes, 10 {6 4 Uterus, 4 Stomach, 1 Meckel's diverticulum, 3 Ventral hernia, 4 Diaphragmatic hernia, 2 Imperforate rectum, 6 Only one umbilical artery, 5 Other conditions, 9

The number 122 is that of individuals, the other figures refer to the number of associated deformities. In 48 out of the 122, or 39 %, of cases there was only one abnormality associated with the H.L.-C.P. This might be almost any one of those noted above. The distribution of these single associated abnormalities is as follows:

a	*b*	*c*	*d*	*e*	*f*	*g*
15 New Growths, 3	3	1	Defect, 4 Accessory "auricles" (or amniotic adhesions), 5	Polydactyly, 7 Syndactyly, 1	—	Viscera, 9

To this may be added some 30 cases of submucous sinuses of lips giving a total of about 78 cases showing one associated abnormality out of about 152, or rather more than 50 %. Thus the correct percentage figure is probably between 40 % and 50 %. Of course the terms used in the above tables are very vague, e.g. polydactyly here indicates anything from a bifid terminal phalanx on one digit to seven fingers on each hand and six toes on one foot, and this is true of some other terms used.

Some of the anomalies in these tables (coloboma, oblique facial cleft, macrostoma, cleft of lower lip and jaw, micrognathia and possibly some cases of hernia cerebri or meningocele in the fronto-parietal region) must be regarded as phases of the same developmental defect as that which produces hare-lip and cleft palate; but the others are of a different nature. There are many family histories in other parts of the Treasury, showing nearly all of these abnormalities (e.g. cataract, hypospadias, polydactyly, defect of bones, congenital heart disease) in various members. Like hare-lip and cleft palate each usually occurs alone in such stocks but not invariably. In these cases of multiple deformities, which represent over 3 per cent. of "hare-lip and cleft palate" cases, the connecting link between one deformity[1] or type of physical degeneracy and another is to be found, and the essential identity of origin of all of them in an inherent tendency is shown. Thus additional evidence of their hereditary causation is afforded. It is evident that the determinantal defect which involves hare-lip is definitely and closely associated with other determinantal defects, that the lesion of the ovum is, at times, a gross one and not necessarily of an entirely different character in the case of one deformity from that in another. These cases show the importance, from the aetiological standpoint, of studying these deformities comparatively and not as mere unit characters.

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[1] It must be understood that only *congenital* deformities are referred to here.

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Further instances of hare-lip and cleft-palate associated with other anomalies are to be found in the following papers:

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VI. HEREDITARY CASES.

Fig. 194. *Guthrie Cayley's Case.* Of I. 1 and 2 no statement is made. II. 2 had hare-lip, married a normal woman, II. 1, and had two children, III. 1 and 3. II. 3 was normal, married II. 4, also normal. They had five children, one, III. 4, with hare-lip, the other four normal. II. 5, the remaining nine children of generation II., were normal, but nothing is said of their descendants. Of IV. 1—7, the children of III. 1, four had cleft palate, viz. IV. 1, 3, 6 and 7. Three, viz. IV. 2, 4 and 5, were normal. (Bibl. No. 129, p. 17.)

Fig. 195. *Passavant's Case.* It is possible that I. 1 was affected by some form of the deformity. Passavant states that seven members of the family were affected, and he begins his account of the family with her. Without I. 1 there would be only six affected. But he does not definitely state that she showed any defect. Of her children two were affected; II. 2 had complete cleft palate and hare-lip (cheilo-gnatho-uranoschisis); II. 3 had hare-lip. In the next generation three were affected; III. 4 had hare-lip, III. 6 had cheilo-gnatho-uranoschisis. III. 13 had hare-lip. In the fourth generation IV. 3 had cheilo-gnatho-uranoschisis. It is noteworthy that the females are more affected than the males and the elder children than the younger. (Bibl. No. 39, p. 305.)

Fig. 196. *Demarquay's Case* (i). IV. 1 was brought to Demarquay in 1844 by the mother, III. 2. He had double hare-lip with considerable projection of the praemaxilla. The vomer was inclined to the right so that the right cleft was less marked than the left, and the right nostril very small compared to the left. The praemaxilla was covered by a quadrilateral fleshy strip. The median incisor teeth appeared at the proper time, but grew horizontally and fell out soon. The lateral incisors were normal. The lower lip was more prominent than normal and formed a semi-circle, projecting forwards. On each side of the median line there were two follicles, similar to those described below. III. 2, aged 45, had been born with double hare-lip and still showed traces of it. There was no division of the palate, but behind the upper lip there was a space which formed a communication between the mouth and nostrils. The lower lip was raised in its middle part and formed a semi-circle. It was much thicker than the upper. The lower median incisors were very large and the corresponding upper incisors were wanting. On each side of the median line of the lower lip were two depressions constantly filled with liquid mucus. They were the openings of two cavities which penetrated $1\frac{1}{2}$ cm. into the lower lip and were formed by a fold of the mucous membrane. They were hypertrophied glandular follicles. This woman stated that her father, II. 1, and grandfather, I. 1, showed the same condition, but gave no details to confirm her assertion that their deformity was the same as hers. She also said that many of her brothers and sisters, III. 1, had the same condition and died young; that she herself had seven children; that four of them, IV. 1, 2, 3 and 4, showed hare-lip, like herself. Of all her children only one, IV. 1, was left. Most of the others had died of convulsions. Richet, however (Bibl. No. 23, p. 174), states that in 1856 a young girl, IV. 6, aged

8 years, with double hare-lip, projection of the praemaxilla and cleft palate, was brought to him. On her lower lip there were two narrow openings, one on each side of the median line, which would admit a probe. Her mother stated that she herself had also had double hare-lip and a similar condition of the lower lip. She brought him to see her mother, the child's grandmother, who was similarly affected. Richet was afterwards informed by another medical man that IV. 6 was a sister of Demarquay's patient, IV. 1. From the dates and ages given this appears to be possible, but if so there is some confusion of sex in Gen. II. in the two accounts. The sex of II. 1 has therefore been left undefined. Richet states that he examined the mother and saw the grandmother. Demarquay saw only the mother. (Bibl. No. 20, p. 52, and Bibl. No. 35, p. 174.)

Fig. 197. *Plicque's Case.* I. 1, 2, 3 and 4 were normal. II. 2 had a supernumerary thumb. Nothing is said of his descendants if any. II. 3 and 4 were normal and not related. They had nine children of whom five, III. 2, 3, 5, 6 and 7, showed hare-lip. In three of these this was reduced to a simple indentation; in the other two cases, and especially in III. 7, there was prominence of the median tubercle; but there was no palatal defect in any of the five children. II. 5 was normal but had two children, III. 9 and 10, with complex lesions affecting the whole palate. II. 7 had hare-lip. This is the only case of this deformity known in the ancestry. He, II. 7, had two normal children, III. 11 and 12. Thus in the three branches of the family there was only one in which no child had hare-lip. But in that branch the father had hare-lip. In the third generation III. 2 and 3 married, but their five children were normal. (Bibl. No. 95, p. 294.)

Fig. 198. *Jardine Murray's Case.* I. 2 had a very narrow and high-arched palate. II. 1, her son, had double hare-lip. This had been cured by operation. He also had two sacculi in the lower lip. These sacculi were situated about $\frac{1}{4}$ inch from the margin of the mucous membrane of the lip. They were crescentic in form and symmetrical in position, one on either side of the middle line. The horns of the crescent were directed forwards and a little outwards. A probe inserted into one of these openings passed downwards on the inner side of the lip, beneath a considerable thickness of mucous membrane, to the depth of half an inch. The pouches did not communicate. They secreted a glairy mucus but caused no inconvenience. A split pea could readily be introduced into each pouch. His brothers and sisters were all normal. II. 2 was well formed in every respect. III. 1 had two sacculi in the lower lip. III. 2 was normal. III. 3 had double hare-lip and two sacculi in the lower lip. III. 4 was normal. III. 5 had a very narrow and high-arched palate which exactly resembled that of her paternal grandmother, I. 2. There was a miscarriage between the births of III. 5 and III. 6. III. 6 was normal. III. 7 had webbed fingers on both hands. The middle and ring fingers of the left hand were united by a web as far as their distal extremities. The middle and ring fingers of the right hand were united by a web which extended to about the middle of the second phalanx. The bones and tendons of each finger were normal. III. 8 had hare-lip and two sacculi in lower lip. He was a well developed and vigorous child. The fissure of the lip was confined to the left side, but extended into the nostril. There was a cleft involving the anterior part of the hard palate between the praemaxilla and the maxilla on this side (left). The praemaxilla was twisted on its base, so that the incisor teeth were placed at an angle to the line of their normal direction and partly bridged over the gap in the alveolar margin of the jaw. This twisting caused the portion of the alveolar margin formed by the praemaxilla to overlap the portion formed by the maxilla. (Bibl. No. 33, pp. 502—509.)

Fig. 199. *Ferrier's Case.* Of I. 1, 2, 3 and 4 nothing is stated. II. 2 was a very strong agricultural labourer, who did not drink, and never had done so. He absolutely denied syphilis. He had several brothers and sisters, nephews and nieces, all healthy. He had never been ill and had served his time in the army without a day at the infirmary. II. 3, a handsome brunette, health excellent, had never had an illness. At 24 years of age married II. 2, then aged 25. She had twelve brothers and sisters, almost all married and with healthy children. II. 2 and II. 3 had six children, III. 2—7. III. 2 was born normal. Had infantile paralysis at 2 years of age, followed by paralytic talipes equino-varus. III. 3 was normal, "a superb specimen." III. 4 was normal, "a very big and fine girl." She and her brother, III. 3, had never been ill, were well made and of average intelligence. III. 5 was a fine girl at birth. No hydramnios. She had cheilo-gnatho-uranoschisis. The cleft of the palate was bilateral. The nasal cavities and mouth formed a sort of cloaca. Suction was impossible, and she died at the age of 2 months from malnutrition. III. 6 also a girl. No hydramnios. She had hare-lip with considerable prominence of the median tubercle. Suction almost impossible, spoonfed. At age of 15 months she looked at least $2\frac{1}{2}$ years old. There were two lower incisors and an enormous upper incisor on the left side of the tubercle. III. 7, a boy, was very big at birth. He weighed probably about 10 lbs. No hydramnios. He showed exactly the same malformation as III. 5. On the third day after birth he fell sick of some "diphtheritic affection" and died seven days later. (Bibl. No. 130, p. 93.)

Fig. 200. *Sproule's Case.* Of I. 1 and 2 there is no definite statement. It is stated that the deformity had been handed down from one generation to another for 100 years, and that in some of the

later instances it had seemed to gain vitality. II. 1, the author of the pedigree, does not say whether he himself showed the defect or not. There is no note of his descendants, if any. II. 2 had hare-lip with complete division of the palate and alveolus. He had seven children, of whom three, III. 1, 2 and 3, showed the same degree of deformity as himself. One had double hare-lip and cleft palate on one side; it is not stated whether this was right or left. There were two girls and one boy. II. 4 had a daughter, III. 5, who showed the deformity, but he himself seems to have been normal. There was "a cousin" who showed the defect. He had a daughter who was similarly affected, but there is no exact statement as to degree of the defect or exact relationship of the cousins. (Bibl. No. 40, p. 412.)

Fig. 201. *Clement Lucas' Case* (i). Of I. 1 and 2 there is no definite statement. But no defect had previously been noticed in any of the relatives. II. 2 had three sisters, II. 3, 4 and 5, all normal. At the age of 25 years she married II. 1. There were five children in seven years. The first four were normal. II. 2 showed the following condition:—Her upper lip was normal, showing no fissure, notch or scar. But the right lateral incisor tooth of the upper jaw was lacking. She was positive that no tooth had ever been extracted in this situation. She was aware that she had a front tooth short of the proper number. All the other teeth were well formed. III. 2, her fifth child, was born at full term. It had hare-lip on the right side, an umbilical hernia and an inguinal hernia on each side. (Bibl. No. 91, p. 64.)

Fig. 202. *Clement Lucas' Case* (ii). Of I. 1 and 2 nothing definite is stated. But no member of the family had shown any deformity. II. 1 died of phthisis. "An aunt" died of the same disease. (Whether paternal or maternal aunt is not stated.) II. 2, a pale, delicate looking woman, one of a large family, had no notch in her lip but the left lateral incisor tooth of the upper jaw was lacking. She was positive that no tooth had been extracted from the front of her mouth. She knew of no deformity in any member of her husband's family. III. 1, her first child, had hare-lip and cleft palate. (Bibl. No. 91, p. 64.)

Fig. 203. *Bartel's Case.* I. 1 and 2 were normal. No hare-lip had occurred in either family. They had five sons, II. 1—5. II. 2 had unilateral cheilo-gnatho-uranoschisis, but the parents could not remember on which side the cleft was situated. II. 5 showed, at birth, hare-lip on the left side, which had healed *in utero*, being represented by a linear cicatrix. This scar extended from the left nostril, in a direction parallel to the philtrum, through the whole of the left upper lip as far as the margin of the mucous membrane. This was somewhat retracted. In continuation of the scar there was a cleft right through the red of the lip. The mucous membrane covering this cleft or notch was very thin. The hard and soft palate were normal. The alveolar margin of the upper jaw showed no trace of notch or scar. (Bibl. No. 57, p. 596.)

Fig. 204. *Sutherland and Jackson Clarke's Case.* I. 1 and 2 were normal and healthy. They knew of no abnormalities in themselves or amongst their relatives. II. 1, their first child, a girl, was healthy and of normal development. II. 2 had partial hare-lip, six digits on each hand, and blueness of lips, nose and ears. He died suddenly at the age of 9 months from congestion of lungs and heart disease. II. 3 presented the following abnormalities:—(1) congenital heart disease, (2) partial hare-lip, (3) six digits on each hand, (4) shortening of all the long bones of the extremities, (5) defective development of gums. The mother stated that he had been cold and blue since birth. Had had measles at the age of 8 months. He had always been subject to bronchitis. He was now two years of age. His parietal and frontal eminences were well marked, the occipital protuberance rudimentary. The anterior fontanelle was almost closed. The circumference of head was $18\frac{1}{4}$ in. The hair of the eyebrows and that over the scalp was scanty. The eyelashes were well developed, the palpebral fissures small, the nose pug-shaped. The upper lip sloped upwards from the sides with a small notch in its centre. In addition to this partial hare-lip, the lips were both drawn inwards. The only explanation of this condition was the imperfect development of the gums and jaws in their anterior portion. Here the teeth were defective. There was only one incisor in the lower jaw. There were none in the upper. The canine and molar teeth were present and apparently normal. The palate was narrow and high-arched. The arms and legs were symmetrically developed, markedly short, thick and somewhat shapeless. The hands were broad, square and shortened. There was a more or less complete sixth digit on each hand. This was attached to the little finger. It was of about half the length of that digit. In each supernumerary digit there was a metacarpal bone and two phalanges. The second and third digits were of equal length, broad and thick, while the first and fourth were considerably shorter than these. The feet were broad and thick, with a tendency to pes cavus. The first and second toes were of the same length. There were no supernumerary digits on the feet. (Bibl. No. 122, pp. 25—28.)

Fig. 205. *Demarquay's Case* (ii). I. 2 stated that her daughter, II. 1, was born with simple hare-lip, but that no other member of the family had been born with that deformity. I. 2 brought her granddaughter, III. 1, the daughter of II. 1, to the hospital. III. 1 had, at birth, double hare-lip and complete cleft palate, but the cleft between the palatine processes of the two maxillae was a very narrow one and

soon disappeared. When the child came under observation she had double hare-lip. There was also division of the vault of the palate posteriorly (partial cleft palate). (Bibl. No. 20, p. 53.)

Fig. 206. *Clement Lucas' Case* (iii). I. 1 was successfully operated upon for hare-lip and cleft palate. She married I. 2, who showed no deformity, and had six children, II. 1—6. The second, II. 2, and sixth, II. 6, of these presented the same deformity as their mother. (Bibl. No. 91, p. 64.)

Fig. 207. *Clement Lucas' Case* (iv). I. 1 was noticed as a boy to have no lateral incisor tooth in the upper jaw on left side. It is certain that no incisor tooth of the second dentition was ever extracted. He had only one child, II. 1, a daughter. She presented the same defect, on the same side. (Bibl. No. 91, p. 64.)

Fig. 208. *Lacazette's Case.* This case is described by Demarquay. I. 1, a carpenter, had hare-lip. II. 1, his son, had the same deformity. (Bibl. No. 20, p. 53.)

Fig. 209. *Steinhausen's Case.* I. 2 had a wide cleft of the upper lip and jaw. II. 1, a well nourished boy, aged three months. The upper lip was cleft to the nasal opening; the lower border of the alveolus was partially cleft. Both edges of the cleft in the lip were turned upwards and outwards; they were almost an inch apart. (Bibl. No. 14, p. 73.)

Fig. 210. *Höring's Case.* I. 1 and 2 were normal. II. 3 had simple hare-lip. During an operation upon this it was observed that the brother, II. 1, aged 10 years, who was present, had unmistakeable traces of hare-lip which had united. The parents said that the cleft was closed at birth, but that the scar had been more obvious at that time. The upper lip was thinner in the region of the scar than elsewhere. The cicatrix was brownish-red in colour. On the free margin of the upper lip, on the right side, a small notch was still to be seen. From this a reddish scar, about $1\frac{1}{2}$ lines wide, ran up to the nostril. The nostril itself was drawn down on this side. It looked as if an operation for hare-lip had been performed. The gums and palate were normal. The boy was late in learning to speak and then he learned with difficulty. (Bibl. No. 24, p. 32.)

Fig. 211. *Lebert's Case.* This case is described by Demarquay. I. 1 had hare-lip. Her daughter, II. 1, also had hare-lip. There is no statement about any other members of the family. (Bibl. No. 20, p. 53.)

Fig. 212. *Müller's Case* (i). I. 1 had hare-lip and cleft palate, for which he had been operated upon in his fifth year. The cleft was on the left side. The nose was slightly flattened and the cheek sunken on that side. The alveolar margin of the jaw did not form a regular arch. The cleft in the palate was triangular. This individual had eight children. One of these, II. 2, had bilateral cheilo-gnatho-urano-schisis. Another, II. 3, had unilateral cheilo-gnathoschisis. The other six were presumably normal. Some were dead. (Bibl. No. 85, pp. 252 and 278.)

Fig. 213. *Müller's Case* (ii). I. 1 had unilateral cheilo-gnatho-uranoschisis. He had three children, II. 1, 2 and 3. All three showed this same deformity. (Bibl. No. 85, p. 252.)

Fig. 214. *Bellingham's Case.* IV. 2 and 3 were brothers. IV. 2 had unilateral hare-lip, the fissure being on the left side. There was no fissure of palate or flattening of the nostril. In IV. 3 the fissure of the upper lip was on the right side. The right nostril was flattened. A wide fissure extended through the alveolus of the maxilla to the palate; but there was no projection of the alveolus. The edges of the fissure were divaricated and there was great deformity. The mother, III. 4, stated that she had three other children, quite normal, but that relatives of the children on both sides had been similarly affected, and that these were all males. Thus I. 2, the paternal grandfather, had hare-lip, and III. 1, a second cousin of their father, was similarly affected. On the mother's side two of her second cousins, III. 5 and 6, both males, had hare-lip. (Bibl. No. 28, p. 161.)

Fig. 215. *Trew's Case.* I. 1 had single hare-lip. II. 3 had double hare-lip. Five of her sons, III. 4—8, had the same defect. Her two daughters, III. 9 and 10, were normal. Her sister, II. 2, herself normal, had a son, III. 2, whose palate, although not cleft, was very uneven, and, as it were, with a tendency to the defect. He himself was unaware of this fact. He married III. 1, who was normal. There were seven children born of this marriage in 24 years. The first three, IV. 1—3, were normal. IV. 4 had hare-lip, and the whole palate was wanting. She died at the age of 32 weeks. IV. 5 was normal. IV. 6 and IV. 7 showed the defect in some form. Exact details of these two individuals are not given. (Bibl. No. 2, pp. 445—447.)

Fig. 216. *Demarquay's Case* (iii). I. 1 and 2 were healthy, but I. 1 came of a stock with peculiar antecedents. No details are recorded, but it is stated that they will be given at a later date. I. 1 and 2 had eight children. Two of these, II. 2 and 3, had hare-lip. II. 2 married. She had two normal children. II. 3 died at the age of 8 months. Of the six normal children two, II. 4 and 5, were

unmarried; II. 6 and 8 had normal children; II. 10 had three children, III. 4—6. III. 4 had double hare-lip and "was very delicate." The space between the two maxillae, which were of deficient growth, was considerable. The praemaxilla, which bore two teeth, projected anteriorly and was deflected to the right as the result of fracture of the vomer produced by a fall. The median lobule was very small; it was adherent to the extremity of the nose. III. 5 had hare-lip. III. 6 showed two sacculi in the lower lip similar to those described in previous cases. II. 12 had eight children; all had either hare-lip or sacculi in the lower lip. Some of them showed both conditions. (Bibl. No. 51, p. 111.)

Fig. 217. *Meckel's Case.* I. 1 bore a child, II. 1, with cleft palate. II. 2, daughter of II. 1, had a child, III. 1, with cleft palate. No other details recorded. (Bibl. No. 7, p. 20.)

Fig. 218. *Houston's Case.* Of I. 1 there is no note. I. 2 was robust and healthy. She had four children, II. 1—4. II. 4 had double hare-lip and bilateral cleft of the palate. II. 1 had shown the same deformity at birth. II. 2 and 3 were normal. (Bibl. No. 18, p. 129.)

Fig. 219. *Bramann's Case.* I. 1 was a healthy man. He had been operated upon for hare-lip in early life. II. 1 and 2, his twin children, had hare-lip on left side with incomplete cleft palate. (Bibl. No. 96, p. 132.)

Fig. 220. *Anna's Case.* I. 2 had two relatives with hare-lip. He married twice. There were nine children by his first marriage, seven of them still-born. The other two had hare-lip. Of the children of the second marriage II. 4 and 7 had hare-lip. Of. II. 5 nothing is recorded. In II. 6 the median portion of the upper lip was wanting. Only the lateral parts of it, about the angles of the mouth, were present. These were everted and projected over the lower lip. The nose was flat and triangular. From its tip a process hung down between the rudiments of the upper lip above described. This was freely moveable. Two parts could be distinguished in it. The smaller, directed to the right, was connected with the columella of the nose by cutaneous tissue. Its upper surface had the usual coverings, and, like the upper lip, it was covered with fine hairs. On it there was a small groove. It was soft to the touch; the edge was free and almost semi-circular. The larger portion lay under the smaller, to which it had become adherent. It was directed towards the left rudiment of the upper lip above described. It was thick, rounded, of reddish brown colour, and appeared to be inflamed on the left side. It felt like cartilage, but was sensitive to the slightest touch. It was attached above to the columella and to the septum of the nose. The whole of the hard palate was wanting, the nasal and buccal cavities being continuous with one another. The soft palate and uvula were also wanting. In the region of the anterior pillars of the fauces, on each side, there was a small fleshy structure, probably a remnant of the arcus pharyngei (palato-glossus and palato-pharyngeus muscles) of the soft palate. Above this, between the posterior nares, there was a body, of bony hardness, covered with a mucous membrane like that of the mouth. The child was seven months old. There were no traces of teeth, but it was stated that at birth there were two "eye-teeth" which became black after a few days and fell out. (Bibl. No. 6, pp. 209—212.)

Fig. 221. *Perthes' Case.* This case is described by Haymann. I. 1, 2, 3 and 4 were normal. II. 2 and 3 were normal. III. 2 had right-sided hare-lip. III. 3 had hare-lip. IV. 1 had right-sided hare-lip and fissure of the uvula. IV. 2 had left-sided cheilo-gnatho-uranoschisis. IV. 3 were all normal. (Bibl. No. 124, p. 1073.)

Fig. 222. *Cooper Forster's Case.* I. 1 had hare-lip. He married and had eleven children. The first two, II. 1 and 2, had hare-lip and imperforate rectum. The third, II. 3, had imperforate rectum and defective palate, but no hare-lip. The next five were free from external deformity. The ninth, II. 5, had imperforate rectum. After its birth the operation for hare-lip was performed on the father. Two normal children were born later. (Bibl. No. 32, p. 30.)

Fig. 223. *Kirmisson's Case* (i). III. 1 had a vertical congenital cicatrix on the left side of the upper lip. His maternal grandmother, I. 1, had a similar congenital cicatrix. (Bibl. No. 116, p. 103.)

Fig. 224. *Kirmisson's Case* (ii). II. 1 had a congenital cicatrix on the left side of the upper lip. His father, I. 2, married a second time. One child of the second marriage, II. 2, had a complicated cleft of the upper lip. (Bibl. No. 116, p. 103.)

Fig. 225. *J. Lucas' Case.* II. 1, 2, 3 and 4 were born with hare-lip. The mother, I. 2, normal, "could ascribe no cause for the deformity." (Bibl. No. 5, p. 101.)

Fig. 226. *Eigenbrodt's Case.* I. 1 had hare-lip. II. 1 had hare-lip. II. 2 had double hare-lip and complete cleft palate. He was a very small and weakly child, very subject to diarrhoea. (Bibl. No. 89, p. 90.)

Fig. 227. *Eigenbrodt's Case.* I. 1 had left-sided hare-lip and a partial cleft of the alveolus. Her left lateral incisor tooth in the upper jaw was wanting, and its left canine tooth was deformed. She had been operated upon. II. 1 had double hare-lip and complete cleft palate. On the left side the cleft in the lip and that in the alveolus were broad. On the right side the cleft in the lip was incomplete and that in the alveolus was very narrow. (Bibl. No. 89, p. 89.)

Fig. 228. *Maurel's Case.* II. 1 had a congenital cicatrix. This was linear, extending on the upper lip from the nostril to the free margin of the lip. There it terminated in a slight depression. I. 1, the mother, stated that her husband had a sister, I. 3, who had shown the same condition. She died on the day of her birth. (Bibl. No. 26, p. 302.)

Fig. 229. *Rennert's Case* (i). I. 3 had simple hare-lip. His niece, II. 2, was operated on for double hare-lip. The cleft extended, on each side, into the nostrils. II. 3, his daughter. Her nostrils were very small, her upper lip very long and flat. Rennert therefore suspected that this also represented a condition of hare-lip which had healed before birth, though he found no congenital cicatrix. (Bibl. No. 23, p. 117.)

Fig. 230. *Rennert's Case* (ii). I. 1 had the deformity of the upper lip called "double lip"; that is to say its thickness was augmented by an internal fold of mucous membrane running parallel to the opening of the mouth. She had five children. II. 1 was a fine girl free from deformity. II. 2, born after a troublesome pregnancy, showed simple division of the upper lip on the left side. There was a bad cicatrix after operation. The mother feared that the next child would be deformed. This was a girl, II. 3. She was fine and robust, but was disfigured by a congenital cicatrix of hare-lip. This was on the right side and was very like that of II. 2. But the disfigurement was even greater, for the scar extended to the corresponding nostril. The right nostril was narrower than the left, and the cartilaginous septum of the nose deviated to the right. The palate was not cleft and the voice was natural. The two children born subsequently were normal. (Bibl. No. 23, p. 117.)

Fig. 231. *Vrolik's Case.* I. 1 bore three children in succession, probably by different fathers. II. 3 had hare-lip and an oblique facial cleft (from the inner angle of the orbit to the angle of the mouth). II. 1 and 2 were said to have had a similar cleft. (Bibl. No. 25, Tab. XX. 3.)

Fig. 232. *Mauclaire's Case.* I. 1 had a nose which deviated to the right. The left nostril was enlarged, but the lips and teeth were normal. I. 2 had no malformation. II. 1 also had a nose which deviated to the right. The left nostril was enlarged, but the teeth and lips were normal. There was no history of syphilis in the ancestors. II. 2 had the following malformations at birth. The nose was slightly deviated to the right. Viewed from the front it appeared to be slightly bilobate. The left nostril was enlarged and deformed. There was a notch on the lip in the usual situation of a left-sided hare-lip. On attaining maturity the division of the hairs of the moustache was to the left of the normal position. There was no cicatrix on the skin. On lifting the upper lip a slight depression of the mucous membrane, corresponding to the direction of the notch, was observed. The teeth were normal on the right side, but on the left one tooth was displaced in a backward direction. Whether this was a canine tooth or a supplementary incisor was doubtful. Mauclaire considers this to be a case of hare-lip which had healed *in utero.* (Bibl. No. 126, pp. 92—94.)

Fig. 233. *Trendelenburg's Case.* I. 1 had hare-lip. II. 1 had cleft uvula. III. 1 had hare-lip. There is no note about any other member of the family. (Bibl. No. 86, p. 35.)

Fig. 234. *Hardurcke's Case.* This case is quoted from *La Presse Médicale*, Belge. II. 2, whose lip was irregular in shape, said she had been operated upon for hare-lip in infancy, and that her mother, I. 1, and sister, II. 1, had been born with the same deformity. Her child, III. 1, also had hare-lip and was operated upon successfully. (Bibl. No. 65, p. 129.)

Fig. 235. *Stobwasser's Case* (i). I. 1 had hare-lip. His child, II. 2, showed the same deformity. (Bibl. No. 76, p. 12.)

Fig. 236. *Stobwasser's Case* (ii). This case is exactly similar to the last. I. 1 had hare-lip. His child, II. 2, also had hare-lip. (Bibl. No. 76, p. 12.)

Fig. 237. *Stobwasser's Case* (iii). I. 1 had hare-lip. His two sons, II. 1 and 2, showed the cicatrices of hare-lip which had healed *in utero.* (Bibl. No. 76, p. 12.)

Fig. 238. *Stobwasser's Case* (iv). II. 1 and 2 had hare-lip. The parents were normal. (Bibl. No. 76, p. 12.)

Fig. 239. *Gotthelf's Case* (i). II. 1 had hare-lip. II. 2 had cheilo-gnatho-uranoschisis. There is no note concerning any other relatives. (Bibl. No. 81, pp. 360 and 600.)

Fig. 240. *Gotthelf's Case* (ii). I. 3 had hare-lip (?double or single). II. 2 had double hare-lip. No other details are recorded. (Bibl. No. 81, pp. 360 and 602.)

Fig. 241. *Gotthelf's Case* (iii). I. 1 had hare-lip. Her child, II. 1, also had hare-lip. No other details are recorded. (Bibl. No. 81, p. 360.)

Fig. 242. *Gotthelf's Case* (iv). I. 3 had hare-lip. II. 2 had hare-lip. No other details are recorded. (Bibl. No. 81, p. 360.)

Fig. 243. *Fronhofer's Case* (i). I. 1 had hare-lip on the right side. Her daughter, II. 1, had double hare-lip and cleft palate. (Bibl. No. 110, p. 888.)

Fig. 244. *Fronhofer's Case* (ii). I. 1 had hare-lip on the left side. Her daughter, II. 1, had left-sided hare-lip and cleft palate. (Bibl. No. 110, p. 888.)

Fig. 245. *Fronhofer's Case* (iii). I. 1 had hare-lip on the right side. Her daughter, II. 1, had left-sided hare-lip and cleft palate. (Bibl. No. 110, p. 888.)

Fig. 246. *Fronhofer's Case* (iv). II. 2 had hare-lip. II. 1 had cleft palate. (Bibl. No. 110, p. 888.)

Fig. 247. *Fronhofer's Case* (v). I. 1 had hare-lip on left side. Her daughter, II. 2, had hare-lip on right side. Her grand-daughter, III. 1, had hare-lip on right side. Her great-grand-daughter, IV. 1, had hare-lip and cleft of the alveolus, on the left side. (Bibl. No. 110, p. 888.)

Fig. 248. *Roux's Case* (i). I. 1 and II. 2 had hare-lip. III. 1, 2 and 3 showed traces of linear scarring with indentation at the free margin of the upper lip. This showed a vertical depression such as sometimes occurs after operation. III. 4 had cleft of the alveolus and hare-lip. (Bibl. No. 22, p. 274.)

Fig. 249. *Roux's Case* (ii). II. 2 had simple congenital division of the upper lip. The brother, II. 1, had been operated upon for double hare-lip. (Bibl. No. 22, p. 45.)

Fig. 250. *Roux's Case* (iii). I. 1 had been operated upon for double hare-lip. He had six fingers on each hand and six toes on each foot. His child, II. 1, had double hare-lip and the same deformity of hands and feet. (Bibl. No. 22, p. 46.)

Fig. 251. *Mason's Case* (i). II. 2 had double hare-lip, without cleft palate. The mother, I. 1, had been successfully operated upon for single hare-lip on the left side. (Bibl. No. 64, p. 20.)

Fig. 252. *Mason's Case* (ii). II. 2 and 3 both had hare-lip. (Bibl. No. 64, p. 20.)

Fig. 253. *Mason's Case* (iii). I. 1 and II. 1 had cleft palate. II. 2 had cleft palate and hare-lip. (Bibl. No. 64, p. 64.)

Fig. 254. *Mason's Case* (iv). I. 1, the father, and one of his children, II. 1, had cleft palate. The other three children were normal. (Bibl. No. 64, p. 64.)

Fig. 255. *Mason's Case* (v). III. 1 had cleft of the soft palate. III. 2 had double hare-lip, and complete cleft of hard and soft palate. The father and mother were normal, but the mother's aunt, I. 3, had cleft palate. (Bibl. No. 64, p. 64.)

Fig. 256. *Rose's Case.* IV. 1 had hare-lip. It was ascertained that the father, III. 2, grandmother, II. 2, and great-grandfather, I. 1, all showed hare-lip of greater or less extent. (Bibl. No. 100, p. 23.)

Fig. 257. *Talbot's Case* (i). II. 1, 2 and 4 had cleft palate. No instance of cleft palate could be found among the ancestors or collateral branches. II. 2 married and had a family, all normal. (Bibl. No. 127, p. 745.)

Fig. 258. *Talbot's Case* (ii). I. 1 had cleft palate. Her son, II. 2, was normal. He had five children. III. 1, 3 and 5 were normal. III. 2 had single hare-lip and cleft palate. III. 4 had double hare-lip and cleft palate. (Bibl. No. 127, p. 745.)

Fig. 259. *Mason's Case* (vi). I. 1, 2 were normal. They had six children. II. 1, 2, 4 and 5 had cleft uvula. II. 3 and 6 were normal. (Bibl. No. 64, p. 64.)

Fig. 260. *Tübingen Case* (i). I. 1 had hare-lip. Four of his children, II. 1—4, had the same deformity. Further details are not recorded, and the sex of only two was known. (Bibl. No. 128, pp. 270 and 271.)

Fig. 261. *Tübingen Case* (ii). I. 1 and his two sons, II. 2 and 3, had hare-lip. No other details recorded. (Bibl. No. 128, pp. 270 and 271.)

Fig. 262. *Tübingen Case* (iii). I. 1 had simple hare-lip on one side. Her son, II. 1, had double hare-lip and cleft palate. (Bibl. No. 128, pp. 270 and 271.)

Fig. 263. *Haug's Case* (i). I. 1, 2, 3 and 4 were normal. I. 2 had two children with the deformity. II. 1 had cheilo-gnatho-uranoschisis. II. 2 had cheilo-gnathoschisis. The other four surviving children were normal. Of the children of I. 3 the two first were normal. II. 6 had simple left-sided hare-lip. The next four children were normal. II. 8 had simple right-sided hare-lip. (Bibl. No. 128, p. 270.)

Fig. 264. *Haug's Case* (ii). IV. 4 was operated upon for hare-lip complicated by gnathoschisis. His mother, III. 2, was normal, but her cousin, III. 4, a female, had hare-lip. Of his nine children, one son, V. 3, was operated upon for double incomplete hare-lip of the second degree. V. 4 had complete hare-lip of the third degree on the left side. Both died. Seven other children, free from deformity, survived. IV. 2, a sister of IV. 4, was herself normal, but had a daughter, V. 2, with left-sided complete hare-lip of the third degree. No malformations had previously been known in the family. (Bibl. No. 128, p. 270.)

Fig. 265. *Tübingen Case* (iv). I. 1 had hare-lip. Two of his children, II. 1 and 2, had a similar deformity. The other six were normal. (Bibl. No. 128, pp. 270 and 271.)

Fig. 266. *Tübingen Case* (v). I. 1 had right-sided hare-lip. II. 2 had double hare-lip. Her child, III. 1, had right-sided hare-lip and cleft palate. (Bibl. No. 128, pp. 270 and 271.)

Fig. 267. *Haug's Case* (iii). I. 1 and 2 were normal. None of their relatives showed any abnormality. They had seven children, II. 1—6. II. 1 had left-sided incomplete hare-lip of the second degree. The cleft was continued as a congenital cicatrix. The second, third and sixth children were normal. II. 3 was operated upon, at the age of nine months, for right-sided cheilo-gnatho-uranoschisis and died. $1\frac{1}{2}$ years later II. 4 was born, and showed a similar deformity on the left side. She died at the age of $1\frac{1}{2}$ years. II. 6 had hare-lip. No further details are recorded. (Bibl. No. 128, p. 270.)

Fig. 268. *Fein's Case.* There is no note concerning ancestors or collaterals of I. 1. Of I. 2 nothing is recorded. I. 1, grandmother of Fein's patient, showed a triangular indentation of the hard palate, and in the middle line of the soft palate an exceedingly thick white raphé. The condition was similar to that observed in her son, but was of less severe degree. II. 1, aged 37 years, son of I. 1, speech nasal, but not markedly so, uvula cleft, soft palate not cleft but showed an elevation, triangular in outline like the cleft shown in the case of his daughter, III. 1. Digital examination revealed the presence of a defect of the bones of the hard palate like that in his daughter, III. 1, but this did not reach so far forward as in her case. II. 2 not examined, but from her statements it was to be judged that her upper jaw was normally developed. II. 3 and 4, sisters of II. 1, normal. III. 1, Fein's patient, a girl aged 8 years, daughter of II. 1. Apart from defect in her palate she showed no abnormality, was well grown and of average intelligence. Soft and hard palate cleft in the middle line as far forwards as the alveolar margin of the jaw. The cleft was triangular in shape, with its apex forwards, reaching to the alveolus. Many adenoid growths in the naso-pharynx. Septum deflected to the right posteriorly, somewhat deficient anteriorly. A small excrescence of epithelium, about the size of a hemp-seed, at the anterior angle of the cleft. Speech markedly nasal and with difficulty understood. III. 2 and 3, brethren of III. 1, sex not stated, normal. (See Bibl. No. 131.)

Fig. 269. *E. Schwalbe's Case.* A man, whose palate was complete, but asymmetrical and irregular, as though scarred, married a normal woman. There were seven children, four boys and three girls. All four boys were normal. All three girls had hare-lip and cleft palate. The sister of his mother also had seven children, five sons and two daughters, of whom the daughters were normal and the sons were deformed in the same way. Of I. 1 and 2 there are no details. Of II. 1, 2, 3 and 4 there are no details. III. 1, palate complete but irregular, as though scarred, and asymmetrical. III. 2, his wife, normal. IV. 1, 2, 3, 4, their four sons, were normal. IV. 5, 6 and 7, their three daughters, had hare-lip and cleft palate. III. 3, 4, 5, 6, 7, sons of II. 3, sister of II. 1, mother of III. 1, that is to say first cousins of III. 1 through sisters, showed hare-lip and cleft palate. III. 8 and 9, brothers of III. 3, 4, 5, 6 and 7, normal. (See Bibl. No. 132, the above account follows Schorr, Bibl. No. 133.)

Müller (Bibl. No. 85, S. 251—2) and Haug (Bibl. No. 128, S. 269—270) describe a considerable number of cases in which they state that relatives possessed the defect, but they do not give the number of relatives in each class. Of this vague "heredity" they assert the occurrence in 12 °/₀ of cases. These are distributed as follows:

Relative	No. of cases
Father	5[1]
Father's brother	5
Child of Grandfather	1
Two father's cousins	1
Cousin	1[2]
Brother or sister	49[1]
2 brothers or sisters	2[1]
3 brothers or sisters	3[1]

Relative	No. of cases
Mother	10[3]
Mother and grandmother	1
Mother's brother	6
Mother's brother and his two children	1
Mother's sister	4

It has not been considered necessary to put these very incomplete pedigrees on the plates.

DESCRIPTION OF PLATES.

PLATE G. *a—i* Diagrams varieties of cleft palate (the soft palate is not shown). The common conditions are *a*, *b*, *c*, *f* and *h*. *i* shows cleft of alveolus of jaw (*Kieferspalte*) without cleft of palate. *h* shows cleft of palate (*Gaumenspalte*) in incomplete form. Except after operative removal of praemaxilla *e* is an extremely rare condition; shown, however, in Plate I, Figs. 10 and 13. It may be questioned whether *d* and *g* occur in man, except in individuals in whom the lateral incisors are lacking. The condition as shown certainly occurs in mammals other than man, *e.g.* in the carnivora. Diagram *j* shows the development of the face in later stages, and should be compared with Diagram *k*. Normally the lateral lobes of the fronto-nasal processes (shown in *j*) unite to form *a* (shown in *k*); persistence of the condition shown in Diagram *j* produces median hare-lip (see Plate J, Fig. 14) and median cleft of nose, which may occur without median hare-lip. If the fronto-nasal process (shown in *j*) is of defective growth as a whole the condition shown in Plate I, Figs. 10 and 13, or a more extensive median defect, is produced. Normally the fronto-nasal process forms prolabium, praemaxilla, septum of nose, and median part of nose itself (Diagram *k*, part *a*); the lateral nasal process (Diagram *k*, part *b*) forms the lateral walls of the nose; it unites with the maxillary process (Diagram *k*, part *c*) to form the face. Failure of union between *b* and *c* (Diagram *k*) produces oblique facial cleft through the nose (see Plate H, Fig. 7, especially clear on left side, and Plate H, Fig. 6, on left side). In some cases of normal development[4] the lateral nasal process (Diagram *j*) grows down between fronto-nasal process and maxillary process, and enters into formation of upper lip. In such a case *b* (Diagram *k*) is interposed between *a* and *c* in the normal formation of the lip. When during this growth *b* and *c* fail to unite, an oblique facial cleft is produced, which does not involve the nostril but passes from near the angle of the mouth to the inner canthus, a condition shown Plate H, Fig. 6, right side (see also Rose and Carless, *Textbook of Surgery*, p. 789). This is said to be the usual kind of oblique facial cleft, but probably incorrectly. Failure of union between *a* and *c*, or under the last circumstances between *a* and *b* (Diagram *k*), produces the common kinds of hare-lip and cleft palate; and for reasons still unknown this failure of union is much the most frequent[5]. The maxillary process and the mandibular process (Diagram *k*, parts *c* and *d*) do not unite except by their superficial parts, which form the cheek. The surfaces of contact of the deep parts are kept apart by an articulation—the lower jaw joint. Failure of the superficial soft parts of processes *c* and *d* to coalesce to a normal extent produces an abnormally large mouth or cleft of cheek (*macrostoma*: see Plate H, Fig. 5. This is very often associated with defect of external and middle ear, parts developed in connection with the mandibular process). Defect of union between *d* and *d* (Diagram *k*) produces cleft of lower lip and jaw. This is an excessively rare condition in man.

Diagram *l* represents the normal palate at birth with its component parts *a*, *c* and *c'*. *a* is the praemaxillary portion of hard palate, origin shown in *a* of Diagram *k* and the fronto-nasal process of Diagram *j*; *c* is hard palate, *c'* soft palate, which have their origin in *c* of Diagram *k* and the maxillary process of Diagram *j*.

Reference to the following Plates and to the Tables, p. 93 *et seq.*, will show that all combinations of defect of union along the lines indicated in Diagrams *j—l* may occur in man.

[1] In two cases of siblings with hare-lip, the father also had hare-lip.
[2] In this case there was also a sibling.
[3] Including 6 cases intrauterine healing.
[4] It will be obvious that in different normal individuals the relative rate of growth of these processes may not be the same and variations in physiognomy would result. This must tend to be even more marked in abnormal cases.
[5] [Granted the heredity of these conditions, an explanation may be found in the fact that this failure of union leads to more viable cases and therefore to perpetuation. ED.]

Diagram *m*. For purposes of comparison a sketch of the hard palate in a human subject at about five years of age is given, showing the completion of the palate and palatine fissures; the soft palate is not shown, and for the sake of clearness the ossa palati have not been delineated.

PLATE H. Fig. 1. Left *cheilo-gnatho-uranoschisis*. Bulldog puppy, showing left complete hare-lip and cleft of jaw; there is also cleft palate. *Museum of R. C. of Surgeons*, Teratology, 191 A.

Fig. 2. Bilateral *cheilo-gnatho-uranoschisis*. Bulldog puppy, showing bilateral hare-lip and cleft of jaw; there is also bilateral cleft palate. *Museum of R. C. of Surgeons*, Teratology, 191 A.

Fig. 3. Head of Hare, showing formation of upper lip in normal animal; photographed for this work. There is a median cleft extending from the upper lip into the nostril on each side. The only known conditions resembling this are those shown on Plate J, Fig. 14, and Plate I, Fig. 13. The cleft in Fig. 14 is median, but does not extend into the nostrils; that in Fig. 13 is median, and extends into both nostrils, but the jaw behind the cleft is totally lacking. The term "hare-lip" is thus an inaccurate one.

Fig. 4. Bilateral hare-lip of a very severe degree; palate almost entirely lacking. Praemaxilla present in a very rudimentary form as a tag attached beneath tip of nose; it bears, however, two rudimentary incisor teeth. *Associated defects.* Bilateral microphthalmos (left eyeball and palpebral fissure almost completely lacking). Both auricles deformed. *Museum of R. C. of Surgeons.*

As regards the palate Figs. 4 and 7 represent an extreme degree of the condition shown in Plate G, *b*. Fig. 6 shows a very extreme degree of Plate G, *f*.

Fig. 5. Horizontal facial cleft—*macrostoma*. Palate completely lacking; maxilla very small—*micrognathia*[1]; buphthalmos secondary to this. *Associated defects.* Very small external auditory meatus; defect of auricle almost complete, only a rudiment of lobule present. *Museum of R. C. of Surgeons.*

Fig. 6. Hare-lip and cleft palate, left side. Praemaxilla displaced forwards to right. On left side oblique facial cleft through the nose, *i.e.* of same kind as in Fig. 7 (less extensive than that on left side, but about the same as that on the right side of that figure). Between actual cleft and inner canthus of left eyelids (still non-united) is a faint linear scar of an old cleft. On right side is a scar of oblique facial cleft of a different kind; it extends from inner canthus to angle of mouth and does not involve nostril at all; it is the usual variety of oblique facial cleft. *Associated defects.* Facial and cranial asymmetry, hydrencephalocele; latter occipital and not shown. *Museum of the London Hospital.*

Fig. 7. Hare-lip and cleft palate, oblique facial cleft through nose, more extensive on left than on right side. Cleft of right upper eyelid. Cleft of face extends as palpebral fissure on left side; lower eyelid almost lacking. The condition on this side is that shown in Plate G, *j*, which is exactly reproduced, and is an extreme grade of the condition given on Plate J, Fig. 17. *Associated defect.* Ectromelia, left arm completely lacking: cf. Aucante's case, Bibl. No. 8, and the Table, p. 97. *Museum of the London Hospital.*

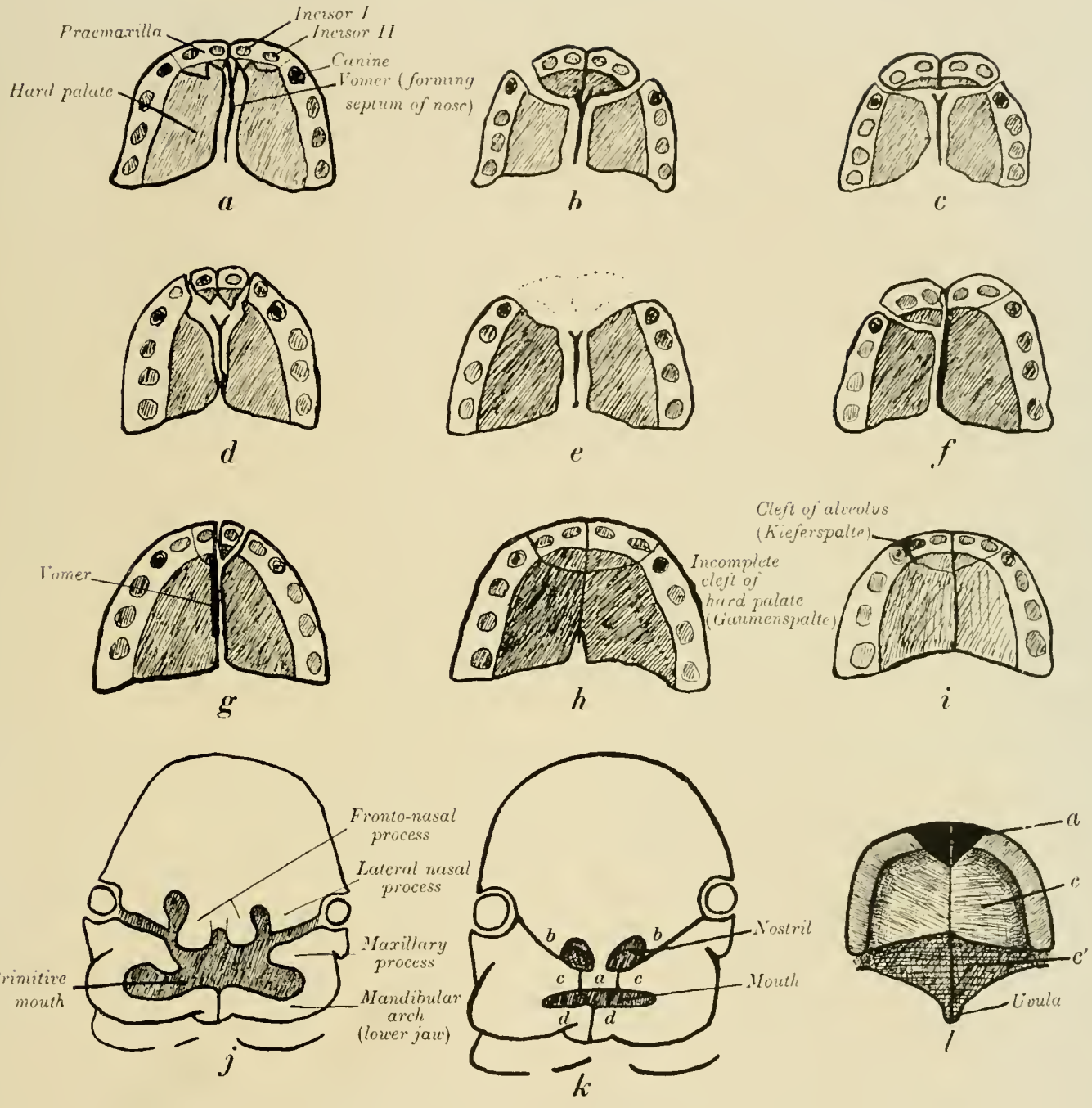
This Plate is intended to illustrate oblique facial cleft and horizontal facial cleft, phases of the same defect as hare-lip and cleft palate, and the occurrence of the latter in mammals other than man. Figs. 4, 5, 6 and 7 show other defects as well, similar to those enumerated in the Tables on p. 93 *et seq.* which occur in association with hare-lip and cleft palate. It is obvious that the individuals shown in these figures were not viable. This is chiefly owing to the extreme grade of their defect and to the number of the associated defects. Any of the conditions described on this Plate may occur alone and be compatible with life. These extreme cases are shown here, because they illustrate the nature and origin of these clefts more clearly than less marked cases would do, and further because they illustrate some of the conditions of a different character which are found in association with hare-lip and cleft palate deformities: see the Tables, p. 93 *et seq.*

PLATE I. Fig. 8. Normal palate of child about 18 months old. Praemaxilla, palate processes of maxilla, palate processes of palate bones and vomer. To be compared with Plate G, *m*, also with *a* and other figures on the same plate. *Museum of R. C. of Surgeons*, Osteology, 55.

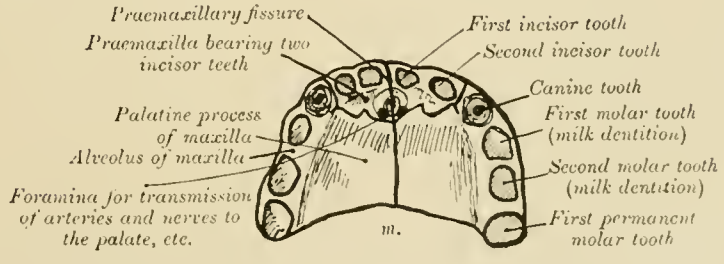
Fig. 9. Adult skull, showing complete bilateral cleft of palate. The palate is not united to its fellow of the opposite side nor to the vomer, which forms the septum of the nose. On the left side the praemaxilla is completely lacking. Compare with Plate G, *e* and the present plate Figs. 10 and 13, in which both praemaxillae are lacking. It is possible that the defect of praemaxilla in this skull is the result of operation. *Museum of R. C. of Surgeons*, Teratology, 201.

Fig. 10. Bones of the face of an infant showing complete defect of praemaxilla on each side. The bones of a case of so-called median "hare-lip." The case itself is shown in Fig. 13. Cf. Plate G *e*. *Museum of R. C. of Surgeons*, Teratology, 208.

[1] Apart from micrognathia, palate represents extreme case of Plate G, *a*.



a to *i*. Diagrams showing varieties of Cleft Palate. (The soft palate is not shown.)



m. Diagram of Hard Palate in subject of about 5 years.

(*For the sake of clearness the ossa palati have not been delineated.*)

See separate descriptions of plates.

FIG. 1. Bulldog puppy, left complete hare-lip and cleft of jaw; also cleft palate. Museum, R. C. of Surgeons.

FIG. 3. Head of Hare, showing formation of the upper lip in the normal animal.

FIG. 2. Bulldog puppy, bilateral hare-lip and cleft of jaw; also bilateral cleft palate. Museum, R. C. of Surgeons.

FIG. 4. Bilateral hare-lip and cleft palate. Only rudiment of praemaxilla; etc. Museum, R. C. of Surgeons.

FIG. 5. Horizontal facial cleft (macrostoma). Total defect of palate; etc. Museum, R. C. of Surgeons.

FIG. 6. Left hare-lip, cleft palate, traces of facial clefts; etc. Museum, London Hospital.

FIG. 7. Bilateral hare-lip, cleft palate, double oblique facial cleft, etc. Museum, London Hospital.

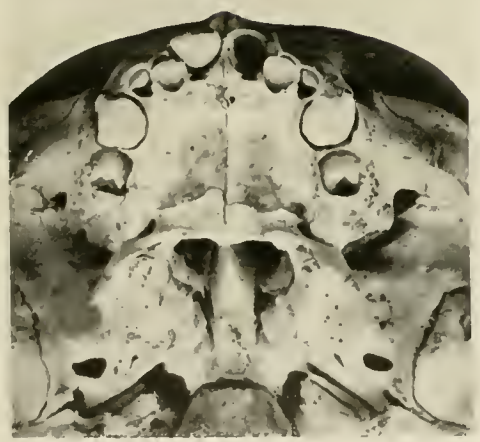


FIG. 8. Normal palate of child about 18 months old. Museum, R. C. of Surgeons.

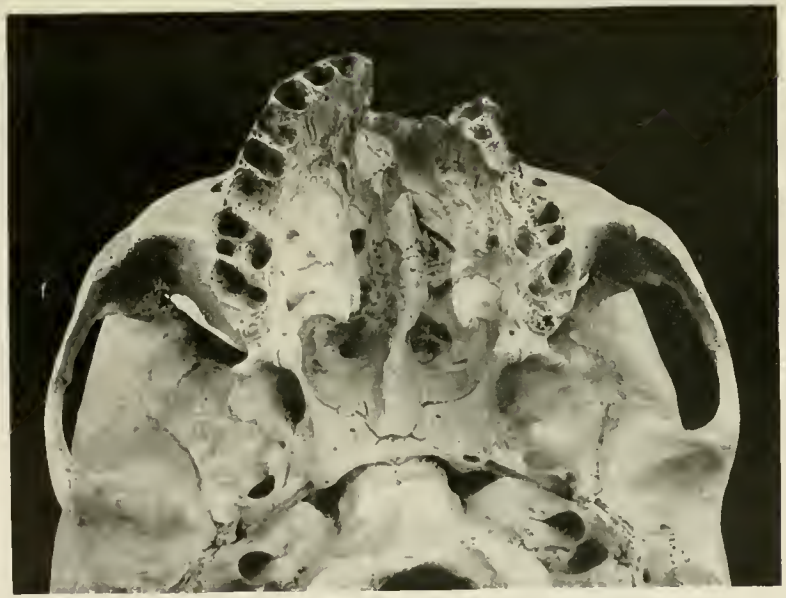


FIG. 9. Adult skull. Complete bilateral cleft of palate. Absence of left praemaxilla. Museum, R. C. of Surgeons.

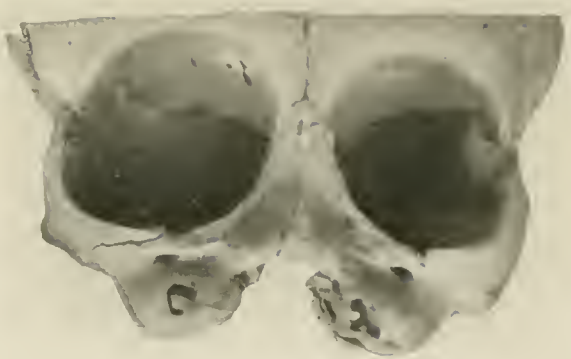


FIG. 10. Bones of face of Fig. 13. Complete defect of praemaxilla. Museum, R. C. of Surgeons.

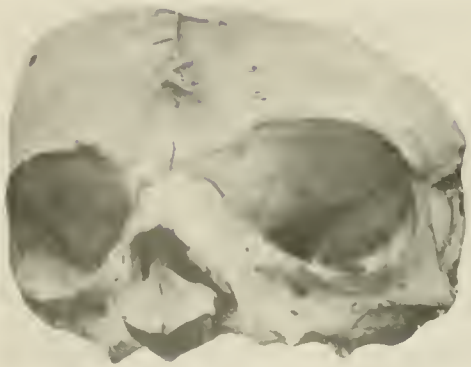


FIG. 11. Bones of face of an infant with bilateral hare-lip and cleft of palate. Museum, R. C. of Surgeons.

FIG. 12. Palate of Fig. 11, showing cleft of alveolus on each side and cleft of hard palate.

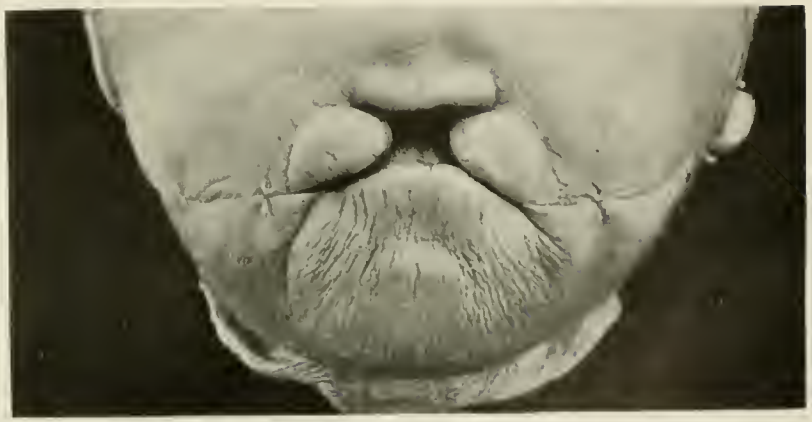


FIG. 13. So-called median "hare-lip." Bones shown in Fig. 10. Museum, R. C. of Surgeons.

See separate descriptions of plates.

FIG. 14. Median hare-lip proper. Mr R. C. Dun's case.

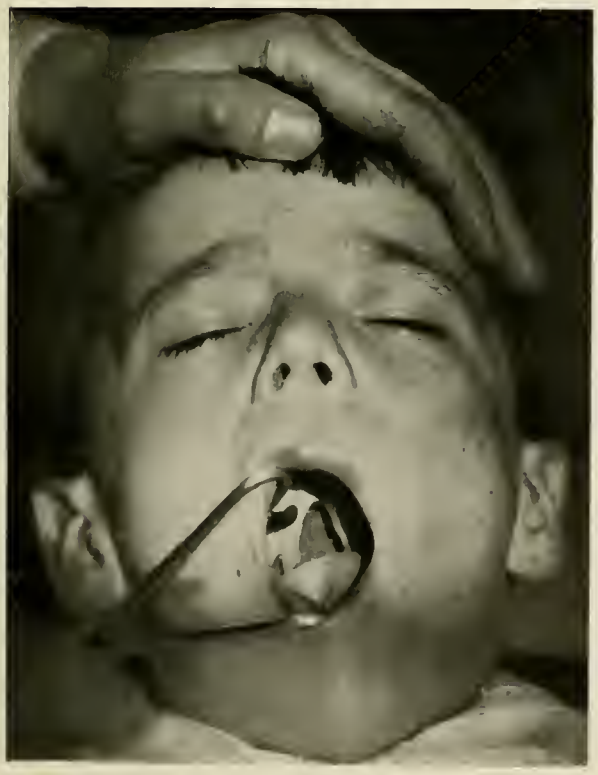


FIG. 15. Incomplete cleft palate (cleft of soft palate and uvula). Mr J. Sherren's Case.

FIG. 16. Partial hare-lip, right side. Mr J. Sherren's Case.

FIG. 17. Bilateral hare-lip, cleft of alveolus and palate. Mr F. S. Eve's Case.

See separate descriptions of plates.

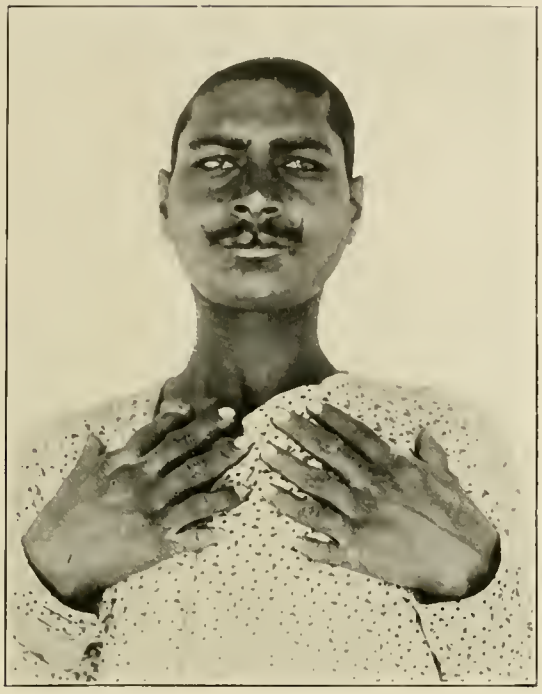


FIG. 18. Hindoo showing median hare-lip associated with polydactyly. Mr E. O. Thurston's Case.

FIG. 19. Hare-lip, cured by operation, associated with bilateral submucous sinuses of the lower lip. Mr R. C. Dun's Case.

FIG. 20. Bilateral hare-lip, cleft of alveolus and palate associated with bilateral sinuses of the lower lip. Mr R. C. Dun's Case.

FIG. 21. Bilateral hare-lip, cleft of alveolus and palate, with median submucous sinus of lower lip. Mr R. C. Dun's Case.

Mr R. C. Dun's Cases are reproduced from photographs kindly sent by him. See his account: "Congenital recesses of the lower lip," *Liverpool Medico-Chirurgical Journal*, July 1909. Mr E. O. Thurston's Case is reproduced by kind permission of the Editor of the *Lancet*, who provided the block. *See separate descriptions of the plates.*

Fig. 11. The same skull as in Fig. 12 viewed from the front. Alveolus cleft on each side. Praemaxilla completely separate from maxilla on both sides, but attached to vomer above. See references under Fig. 12 for comparison.

Fig. 12. Palate showing cleft of alveolus on each side, cleft of hard palate (palate not united to its fellow nor to vomer on either side), with forward projection of praemaxilla. Condition shown in Plate G, *b* and *c*, and Plates H, Fig. 2, J, Fig. 17, and K, Fig. 20. In most cases the cleft is confined to one side, usually the left, as in Plates G, *f*, and H, Fig. 1. *Museum of R. C. of Surgeons*, Teratology, 194.

Fig. 13. So-called median "hare-lip," a very rare condition, almost as uncommon as Fig. 14, Plate J, which represents median hare-lip proper. The prolabium is completely lacking; the praemaxillae are also completely lacking, and the palate is cleft. Thus the term "hare-lip" is not accurate. The bones of this case are shown in Fig. 10. Cf. Plate G, *j* and *k*, and the condition in *e*, and mark the difference from Fig. 3, Plate H. *Museum of R. C. of Surgeons*, Teratology, 207.

This Plate is intended to illustrate some of the bone conditions of cleft palate.

We have to express our sincere thanks to the authorities at the Royal College of Surgeons for much of the material used in the illustrations of Plates H and I.

Plate J. Fig. 14. Median hare-lip proper. The cleft extends along the septum of the nose as far as its extremity. There is thus a median cleft of the nose as well as of the lip. (Cf. Plate K, Fig. 18, in which the cleft involves the lip alone, and is partial even in that case.) The nose itself is very broad and flat. A very rare condition. Cf. Plate I, Fig. 13, and Plate G, *j*. We owe this photograph to the courtesy of Mr R. C. Dun.

Fig. 15. Defect of about the same intensity as Fig. 16 involving the palate. Incomplete cleft palate; cleft of soft palate and uvula (*staphyloschisis*). Cf. Plate G, *h*, which represents a condition a little more extensive than this, the hard palate being partially cleft as well as the soft; see also Plate G, *l* for illustration of these parts in normal condition. Mr James Sherren's Case.

Fig. 16. Partial hare-lip (*cheiloschisis*), the second commonest variety of the deformity; here on right side, usually on left. Note unequal size of nostrils and their difference in level; ala of nose flattened on defective side. Mr James Sherren's Case.

Fig. 17. Bilateral complete hare-lip, cleft of alveolus and of palate, with projection of praemaxilla forwards (bilateral *cheilo-gnatho-uranoschisis*, unilateral being the commoner). This is the third most frequent variety of the defect. Condition is shown Plate G, *b* and *c*, Plates H, Fig. 2, I, Figs. 11 and 12, and K, Fig. 20. Mr F. S. Eve's Case.

This Plate is intended to illustrate varieties of hare-lip, two common and one extremely rare. A simple partial cleft of palate is also shown. In Fig. 17 there is also cleft of palate which cannot be seen. Fig. 13, Plate I, should be taken in conjunction with those on this plate.

Plate K. Fig. 18. Hindoo showing median hare-lip associated with polydactyly. A brother showed a similar condition. For another family case of hare-lip associated with polydactyly see Pedigree 250. Mr E. O. Thurston's Case. See *Lancet*, p. 996, October, 1909, reproduced by the kindness of the Editor of the *Lancet*.

Fig. 19. Child showing hare-lip (cured by operation) associated with bilateral submucous sinuses of the lower lip, one on each side of the median line. Cf. Jardine, Murray, Demarquay, etc. for family cases. Mr R. C. Dun's Case.

Fig. 20. Child showing bilateral hare-lip, cleft of alveolus and palate, with projection of praemaxilla forwards, associated with bilateral sinuses as in Fig. 19. In this case, however, each showed as well a proboscis-like structure capable of extrusion on movement of the lip. Mr R. C. Dun's Case.

Fig. 21. Child showing hare-lip, cleft alveolus and palate, associated with a condition like that in Figs. 19 and 20. In this case, however, the sinus was single and situated in the median line. Mr R. C. Dun's Case.

We owe photographs of Figs. 19, 20 and 21 to the kindness of Mr Dun.

This Plate shows cases in which hare-lip or cleft palate is associated with other abnormalities, not so extensive in character as those on Plate H.

SECTION VI γ (*see* Vol. I. pp. 27 and 69). HEREDITARY DEAF-MUTISM, *continued.*

(EUGENICS LABORATORY.)

The following unpublished pedigrees of congenital deaf-mutism have been compiled in the Eugenics Laboratory from material most kindly provided by members of the staff or by workers among the deaf and dumb connected with the *Royal Association in Aid of the Deaf and Dumb.* We have especially to thank the Rev. F. W. G. Gilbey for his most willing help amid many more pressing duties. Mr E. Bates James and the Rev. W. Raper have gone into individual cases and visited and written to members of the stocks with whom they were not personally in touch. The pedigrees have gone to and fro with additional inquiries, and personal and written communications have been made to many individuals both on the part of the above workers among the deaf and dumb and from the Eugenics Laboratory itself. The actual preparation of the pedigrees, of which the present are only a first instalment, has been due to Amy Barrington and Ethel M. Elderton. We cannot sufficiently acknowledge the ready manner in which Mr Gilbey and his co-workers have appreciated the scientific aspect of this inquiry and given every assistance within their power to further it.

PLATE XXVII. Fig. 270. *Eugenics Laboratory Case I. Family C.* I. 1 and I. 2 were normal, their sons II. 1, and a daughter II. 2, were also normal; one daughter II. 3 was, however, a congenital deaf-mute, she married II. 4, a deaf-mute, the son of deaf-mute parents I. 3 and I. 4; he had a normal brother II. 5, and a normal sister II. 7, who died aged 12. II. 4, made a good living as a hawker but sometimes drank too much. II. 3 and II. 4, had seven children. Of these III. 2, normal, married III. 1, who was "hard of hearing," and had two children IV. 1—2, both normal. III. 3, unmarried, was a deaf-mute. He was intelligent, inherited his father's hawking business and was said to drink. III. 4, unmarried, was normal and died of some unspecified cause, he served as a soldier in S. Africa. III. 5, normal, was unmarried. III. 6, deaf-mute, was unmarried and when he had money drank. III. 7, normal, married III. 8, normal, and had two normal children, IV. 3—4. III. 9, normal, married III. 10, normal, and had four normal children, IV. 5—8. II. 4, married a second time a widow, II. 11, who was a deaf-mute, her father I. 5, normal, died in an alms-house, her mother I. 6, normal, died in a workhouse infirmary. She had two normal sisters, II. 8 and II. 9, one of whom was married, but no particulars were available of her or her descendants, and a normal brother II. 10, who may have had descendants, but no statement concerning them was available. There were two children of this marriage, III. 12, who died in his second year and was said to be able to hear and speak, and III. 13, a deaf-mute, who is stated to have become deaf and dumb from the effects of a fall at the age of $3\frac{1}{2}$, which caused discharges from the ears and deafness. II. 11, by her first husband II. 12, a deaf-mute, had seven children, five of whom, III. 14, died in infancy, of the other two, III. 15, normal, died aged 14, and III. 16, normal, married a normal man III. 17, and has had two normal children IV. 9. II. 12, was a horse slaughterer who was stated to have lost his speech and hearing by a fall from a window, he was the only one of his family so afflicted and had a normal brother II. 13, and two normal sisters II. 14, and II. 15, of whose descendants, if any, nothing is known. No statement was made with regard to his parents I. 7 and I. 8. II. 11, married a third time a congenital deaf mute, II. 16, who had a normal brother, II. 17, and two normal sisters, II. 18 and II. 19. Nothing was known of his parents, I. 9 and I. 10, nor of the descendants of his brother and sisters if any. There was no issue by this third marriage. II. 5, normal, married a normal wife, II. 6, and had a son, III. 11, who was practically a deaf-mute although he could speak a few words, "having been encouraged to do so at school." (*Names preserved in Eugenics Laboratory.*)

Fig. 271. *Eugenics Laboratory Case II. Families H. + M.* I. 1, and I. 2, were normal, they had four sons and a daughter. Of these, II. 1, normal, married II. 2, normal, and had four normal children, III. 1—2. II. 3, normal, married II. 4, normal, and had two normal sons, III. 3—4. II. 5, normal, married II. 6, normal, and had three normal children, III. 5—8. II. 7, a deaf-mute, married II. 8, normal, and had two children, III. 8, a deaf-mute imbecile, and III. 9, normal, who married III. 10, normal, and had three normal children, IV. 1—3. II. 9, a deaf-mute, married II. 14, also a deaf-mute, whose parents I. 3, and I. 4, were normal. She had four brothers, II. 10, unmarried and partly deaf; II. 11, normal, married II. 12, normal, and had four normal sons, III. 11; II. 13, unmarried, was deaf, but spoke a little; II. 15, a deaf-mute, married II. 16, normal, and had normal children, III. 17—21. II. 9 and II. 14, had six children, of whom four, III. 12—14, and III. 16, were deaf-mutes, and the remaining two, III. 15, were normal. (*Names preserved in Eugenics Laboratory.*)

Fig. 272. *Eugenics Laboratory Case III. Family N.* I. 1, and I. 2, were normal. Of their children, II. 1, II. 3, and II. 9, were normal and childless, II. 5, and II. 7, were normal, married normal persons, II. 6 and II. 8, and had normal children, III. 1—4. II. 4, deaf-mute, was unmarried. II. 2, deaf-mute, became nearly blind some years before his death, he married II. 11, also a deaf-mute and much younger than himself. Her father, I. 3, normal, deserted her mother, I. 4, normal, after the marriage of II. 11, and I. 4 married another normal man, I. 5, by whom she had several normal children, II. 13. II. 11 had ten children, the first four, III. 5—8, died too young to ascertain whether they were deaf-mutes or not, III. 9, III. 10, III. 13 and III. 14, were deaf-mutes, III. 11 and III. 12 were practically deaf-mutes, but were said to hear a little. II. 10, deaf-mute, sister of II. 11, married a normal man, II. 12, and had four children, III. 15—18, all deaf-mutes. (*Names preserved in Eugenics Laboratory.*)

Fig. 273. *Eugenics Laboratory Case IV. Families D. + P. + R.* Nothing is known of I. 1 and I. 2, they had six children of whom three daughters, II. 1, were normal, but no statement was available with regard to their descendants if any. Another daughter, II. 2, was a deaf-mute and died. II. 3, the son was unmarried, deaf-mute, and "hopelessly diseased in face." II. 4, another deaf-mute daughter, married II. 5, also a deaf-mute, the son of normal parents, I. 3 and I. 4, who had other five normal offspring, II. 6. It is stated that II. 5 had no deaf-mute relatives. II. 4 and II. 5, had six children, III. 1—4; III. 1, deaf-mute, married III. 12, deaf-mute, but there was no issue of this marriage, III. 2 had partial hearing, III. 3 was a deaf-mute, and the other three children, III. 4, could all hear. I. 5 and I. 6 were normal and were not known to have any deaf-mute relatives. Two of their children were deaf-mutes, II. 8 and II. 9. II. 9, a mattress-maker and alcoholic, died aged 68 after a week's illness, cause unknown. He married II. 10, the congenital deaf-mute daughter of normal parents, I. 7, and I. 8. She had several brothers and sisters, II. 11, all normal, whose children, III. 16, were all normal. II. 10, had ten children. Of these, III. 5, normal, died aged 17 months. III. 6, normal, died aged 4 years. III. 7 were stillborn. III. 9, deaf-mute, married three times; by her first husband, III. 8, who was also deaf-mute, she had twins, IV. 1—2, who died in infancy before any knowledge as to deafness could be ascertained. Her second husband was III. 17, the deaf-mute son of deaf-mute parents, II. 12 and II. 13, he had seven hearing brothers and sisters. His grandparents, I. 8 *a* and I. 8 *b*, were also deaf-mutes. By III. 17, III. 9 had three children, IV. 3—5; IV. 3 was deaf-mute, IV. 4, normal, and IV. 5, died aged five months. The third husband of III. 9, was III. 21, a deaf-mute "natural," and by him she had twins, IV. 7—8. IV. 7, died in infancy, IV. 8, met with an accidental death, caused by the drunkenness of its mother, at the age of two months. III. 10, deaf-mute and alcoholic, in an Inebriate Home for three years, married III. 11, also a deaf-mute, much older than herself, but had no children. III. 12, deaf-mute, has been mentioned before. III. 13, a soldier and normal, married a normal wife, III. 14, and had a normal child, IV. 6. III. 15, was a deaf-mute imbecile, unmarried. I. 9 and I. 10 were normal and in good circumstances, they had three normal children, II. 14—16, but II. 16 was ruined by drink and lost all his money, he married his first cousin, II. 21, who was also alcoholic, but not a deaf-mute; her parents, I. 11 and I. 12, and her brothers and sisters, II. 17—20, and II. 22, were normal. There were five children of this marriage, III. 19—23. III. 19 was normal. III. 20, stillborn. III. 21, a deaf-mute "natural." III. 22 was normal but went wrong, and III. 23 was normal. (*Names preserved in Eugenics Laboratory.*)

Fig. 274. *Eugenics Laboratory Case V. Family M.* I. 1 and I. 2, were normal. II. 1 and II. 3, were normal, married normal wives, II. 2 and II. 4, and had normal children, III. 1 and III. 2. II. 5, a deaf-mute, married II. 6, also a deaf-mute, and had one child, a deaf-mute son, III. 3. II. 7, deaf-mute, married a deaf-mute man, II. 8, and had a hearing daughter, III. 4. II. 9, normal, married a normal man, II. 10, and had a normal son, III. 5. II. 11, was normal, but there is no further record of her. III. 3, had work in the engineering line, he married III. 6, normal, whose parents, II. 12 and II. 13, were deaf-mutes. She had one normal sister, III. 7, who married III. 8, normal, and had normal children, IV. 8, and two normal brothers, III. 9, who married III. 10, normal, and had normal children, IV. 9, and III. 11, unmarried. III. 3 and III. 6, had eleven children all normal. Five died, IV. 1. IV. 3,

unmarried, was a little weakminded. IV. 2, were unmarried. IV. 4, married IV. 5, normal, and had three children, V. 1—3, of whom two, V. 2—3, died in infancy. IV. 6, married IV. 7, normal, but there was no issue of this marriage. (*Names preserved in Eugenics Laboratory.*)

Fig. 275. *Eugenics Laboratory Case VI. Family D. + D.* I. 1, I. 2, I. 3 and I. 4, were normal, and all their children were normal. II. 2 and II. 3, had seven children. Of these, III. 1 became insane and died in a lunatic asylum. III. 2 and III. 4, normal, married and had normal children, IV. 1 and IV. 2. III. 6, normal, married a normal man, III. 7, and had four children, two of whom, IV. 3—4, died of consumption. III. 9 and III. 11, were also normal, married, and had normal children, IV. 6 and IV. 7. III. 8, deaf-mute, married III. 30, also deaf-mute, her grandparents, I. 5, I. 6, I. 7 and I. 8, were normal, as was also her mother, II. 7, but her father, II. 8, developed deafness after his marriage. It was said to be the result of a cold. III. 30, was one of a family of fourteen, four of whom, III. 13, died young. Her surviving brothers and sisters with the exception of III. 31 were all normal, married, and had normal families. III. 31, was slightly deaf at times, but not always so, he was married but childless. III. 30 and III. 8 had eight children, of these: IV. 16, normal, married, IV. 17, normal, and had five normal children, V. 1—5. IV. 18, normal, was married but childless. IV. 20 and IV. 22, were normal and single. IV. 23, normal, was married and had three normal children, V. 6—7. IV. 25, deaf-mute, was unmarried, and there is no record available of IV. 26. IV. 21, was partially deaf, she married IV. 27, a deaf-mute, who had deaf-mute parents, III. 36 and III. 37, III. 37 being also insane; IV. 27's paternal grandparents, II. 9 and II. 10, were normal, but he had two paternal uncles, III. 33 and III. 34, and one paternal aunt, III. 35, deaf-mutes, and of his brothers and sisters, two brothers, IV. 28 and IV. 29, were deaf-mutes, the others, IV. 30 and IV. 31, were normal. IV. 21 and IV. 27, had only one child, a daughter who died in infancy and had a cleft palate. (*Names preserved in Eugenics Laboratory.*)

SECTION XIIIα. CONGENITAL CATARACT.

By N. Bishop Harman, M.A., M.B. Cantab., F.R.C.S.Eng.

(i) *General Account.*

For the benefit of the non-medical reader it will be well to define at the outset the terms that appear in this paper. In the title: the word "congenital" is used in its strictly literal sense "born with one; dating from birth"; cataract is used in its acquired and restricted technical sense, as connoting opacity of the crystalline lens of the eye. Congenital cataract therefore indicates a lens opacity believed to be existing at or before the time of birth.

The forms of cataract which appear in the pedigrees collected in this monograph may be classified as follows:

1. Lamellar cataract.
   - (*a*) Lamellar cataract proper.
   - (*b*) Discoid cataract.
2. Coralliform or axial cataract.
3. Stellate cataract.
4. Anterior and posterior polar cataract.
5. Then follows a group of pedigrees of "congenital cataract" of which the form of opacity was not strictly defined, or not stated by the authors of the pedigrees.

1. (*a*) *Lamellar cataract proper* is by far the most frequent form of cataract found in children. The appearance of the opacity is distinctive. Most will retain

in their memory the appearance of the "alley marble" beloved in boyhood days, it was a sphere of glass, clear for the greater part, but within its core was embedded a device of threads of coloured glass arranged in spherical or spiral fashion. The lamellar cataract is something like that. Instead of a wholly clear transparent lens there is within it a layer, a "zone" or "lamella" of opaque substance. (Plates L and M, Figs. 9, 10, 11 and 16.)

The lamellar cataract is usually, but not invariably, bilateral. The opacity varies in density and in size; in some cases it obstructs vision completely, in other cases fair vision may be obtained either through its substance when it is thin, or around the edges of the opacity when it is of small diameter. The varying form and size of the lamellar cataract will be best understood by a reference to the figures in Plate L and the description of the figures which will be found later.

With this form of cataract there are not infrequently associated other defects, of the teeth, of the bones, of the skull, and sometimes of the mental capacity of the subject; the import of these associated defects will be discussed later.

1. (*b*) *Discoid Cataract*, to use the name suggested by Mr Nettleship, is quite a recent discovery, the first case being described by Doyne in 1888. It is now known to be allied to the lamellar cataract, at any rate the two forms have been found to occur within the same family, and in the same sibship; and intermediate forms between the extremes are described by Nettleship (Plate M, Figs. 20—26). The opacity is of the most delicate order. Usually it appears as a small disc of less than 4 mm. diameter, let in as it were between the nucleus of the lens and the posterior layers of fibres (Plate M, Figs. 17 and 19). The opacity is so thin, that Nettleship qualified his description of it as an "opacity, or to use a better term intransparency, for the opacity is never complete." And in the cases I have recorded (Ped. 344) the opacity was so thin that one was not aware of any intervening obstruction during examination of the fundus of the eye with the direct ophthalmoscope.

The disc is always bilateral. There is never a second or anterior disc in the same lens; it varies very little in size and density in individual cases. The subjects of it are found to be of normal intelligence, often of superior intelligence, and their teeth are remarkably good.

2. *Coralliform Cataract.* This is the name given by Marcus Gunn to a peculiar form of congenital opacity. It has been described as shaped like a "spindle" by v. Ammon, as "fusiform" by Pilz, and "axial" or like "the sails of a windmill" by Knies. The figure of Marcus Gunn's case drawn by Holmes Spicer (Plate M, Fig. 27) accurately depicts its features. There are a number of opacities springing from a nebulous core. The opacities take the form of dense blunt-ended processes radiating obliquely forwards and outwards towards the equator of the lens, but not reaching the capsule. Each spoke or process ends peripherally in a sort of trumpet-shaped enlargement or "mouth," not unlike the mouth of a madrepore coral. The history of these cases indicates that the opacity is present at birth. The cataract is usually stationary.

If the axial cataract described by continental observers be of one and the same type as the coralliform cataract, then lamellar opacities sometimes occur in the same cases as axial opacities, see Müller's case (Plate M, Fig. 28). Also in one pedigree, that of the Betts family described by Nettleship (Fig. 318), some of the cataractous subjects were found by Sinclair to be affected by ordinary lamellar cataract.

But since sections of a coralliform cataract have not been made up to this date the relationship of coralliform to lamellar cataract must be left indeterminate.

3. *Stellate Cataract.* This is a rare form of cataract of which only two pedigrees are to be found. The opacity takes the form of lines disposed in more or less geometrical figures in the anterior and posterior layers of the lens fibres. The figures take the form of a Y or ✳, and suggest a defect in the union of the lens fibres. Since similar markings appear very frequently in lamellar cataract both in the large well-defined form and in the thinnest and most delicate form (cf. Plates L and M, Figs. 14, 29), it is possible that the stellate cataract may be allied to the lamellar cataract.

4. *Anterior and posterior Polar Cataract.* These forms of cataract differ essentially both in form and origin from the lamellar cataracts and the varieties associated with them. They consist of more or less dense opacities in, and beneath, the capsule of the lens: see Plate M, Figs. 30—34. In very many cases, indeed in most cases, the eyes in which these congenital forms of opacities are found are small eyes, they are immature or defective in all their structures. Some writers exclude them from the category of cataract proper on grounds explained later; but there seems every reason to include them in a monograph on congenital hereditary cataract, for the pedigrees given, though few in number, are conclusive of the hereditary character of the defect.

5. The last of our groups of pedigrees of "congenital cataract" comprises those where the type of cataract is not strictly defined, or not described by the author of the pedigree. There are a considerable number of these pedigrees. In some cases the form of cataract is not sufficiently definitive to allow of classification, or else the cataract had been removed from the eye of the subject before the family history was investigated; in the bulk of these pedigrees the authors of the pedigrees have described the condition found as "congenital cataract" only, but this lack of definitiveness will not invalidate their evidence in the study of heredity.

(ii) *The Origin of Cataract.*

1. (*a*) *Lamellar Cataract.* The crystalline lens of the eye, the teeth, the hair, the nails and the numerous skin glands of the body, although so different in their final forms, have each a similar origin and mode of development. They spring from the surface epiblast, the embryonic tissue that makes the epidermis or scarf-skin. They commence their growth by pushing down as a number of pits into the underlying mesoblast—

the embryonic tissue that makes the general skeleton and musculature of the body. These pits become flask-like depressions, and from the bottom of them there spring up by an overgrowth of the cells lining the cavity and particularly the bottom of the pits, the essential structures of these appendages of the scarf-skin. In the case of the glands—the sweat, the grease and the milk glands—the output is fluid or semi-fluid. In the case of the nails, the hair and the teeth, the outcrop is of a more permanent order. In the case of the lens of the eye there is no return outgrowth, for the connection of the lens pit with its mother surface cells is cut off by the rapid growth of the surrounding mesoblast. In this way an island-like globe is buried to continue an isolated development. Within the limits of this sphere of cells the solid lens is produced.

The cells of the deeper layer grow and continue to grow so as to fill the cavity of the buried sphere. The cells elongate, become fibrous, assume a clear, transparent character, and spread over each other in neatly arranged layers. Each fibre takes a definite and almost mathematically accurate course, so that in the end a "lens" of clear, elastic and highly-refracting material is produced. The fibres are cemented together by a fine glue, and the whole lens is enveloped by a clear elastic capsule; both the glue and the capsule are the secretions of the lens fibres themselves.

The figures below give a good idea of the arrangement of the lens fibres.

Fig. i. Fig. ii. Fig. iii. Fig. iv.

Taken from F. Arnold: *Tabulae Anatomicae*, Turici and Stuttgardiae, 1842 (Figs. 17, 18, 20 and 24).

Fig. i. Anterior surface of lens from newly-born child; showing star figure where the fibre ends unite (× 6).

Fig. ii. Ditto from adult; showing many rayed star with increase in layers of fibres (× 4½).

Fig. iii. Side view of foetal lens; showing curving of fibres and alternation of rays of anterior and posterior stars.

Fig. iv. Lens hardened in alcohol; the layers of fibres have split asunder exposing the denser nucleus.

If a fair idea of the growth of the lens is held in mind the conditions that go to make a cataract will be easily followed. Since the lens is made up of many parts or fibres, which are closely glued together both along their contour, and also at their extremities where they meet in radiating lines; it follows that a very little

defect in this delicate joinery will result in flaws of the transparency of the lens. Indeed, no lens is absolutely perfect, as anyone can demonstrate by the subjective impressions of his own eye. To the eye with the most perfect vision a distant point of light appears as a "star," that is a point of light with several radiating limbs; these rays are produced by the dispersion of the light along the flaws of the joints of the ends of the lens fibres, and each ray of the "star" coincides with a line of joinery in the lens. Appropriate illumination of almost any lens will demonstrate objectively these radii as faint milky lines (Tweedy). When these lines are grossly marked then one form of cataract is present.

The nails of the digits and the lens of the eye have an identical source and mode of origin. The likeness extends yet further. The outgrowth of the nails and the enclosed growth of the lens are both specialised in that their substances become clarified and transparent. A very little injury to the nail bed, from trauma or from disturbance of its growth by fever or other severe illness, will produce flaws in the outgrowing nail which appear as white marks in the substance of the nail. So also in the lens, serious disturbance of its nutrition will cause a whole layer of fibres produced at that or some earlier period to be altered in character; the fibres are badly formed or they shrink, and fluid is deposited in minute drops along their lines. These defective layers of fibres are no longer transparent, they are more or less opaque, and form what is known as a "lamellar" or "zonular" cataract.

Similar defects produced by similar causes appear in the growing enamel of the teeth. A line of irregular enamel may not infrequently be seen in the permanent dentition and persist throughout life as the record of a defect in nutrition, or some severe illness, occurring in infant life. In worse cases the whole of the enamel of the teeth may present a rugged, "pitted" or "honeycombed" appearance[1].

Defects in the nails speedily disappear since the nails grow rapidly, but defects of the teeth and of the lens are permanent.

We know the average dates, both during intra-uterine and extra-uterine life, of the laying down and development of the enamel organs of the teeth, and can therefore date fairly accurately the time when the disturbance occurred that produced these permanent marks in the teeth. It follows then that the correlation of the condition of the dental enamel and of the lens of the eye forms an important

[1] These defective teeth are known variously as "rachitic," in the belief that they are the result of rickets (Horner); or as "mercurial," in the belief that the enamel organ is injured either by "teething fits" or "convulsions" occurring in infancy, or by the mercurial powders which are administered to check the recurrence of the fits (Hutchinson). But Norman Bennett has shown that neither the one nor the other of these explanations is sufficient to meet all cases found; and he concludes that the primary cause of these disorders of dentition, and of the cataracts that may be associated with them, lies with a general defect of health due to such causes as hand-feeding, or from attacks of acute febrile disease. In view of these conflicting theories it would be better therefore if these teeth were always given a simple descriptive name, such as "ridged," "pitted" or "honeycombed."

Before leaving this point I might add a caution against putting two much stress upon the presence of honeycombed teeth and their association with cataract. The association is often found, but much more frequently congenital cataract is found without defective dental enamel, for proof examine the notes of the subjoined pedigrees. Further, honeycombed teeth are very common: I have in the past few months examined a group of 613 school-children, aet. 7—14, of a Higher Grade London school, of these 25 out of 432 boys, and 16 out of 181 girls had honeycombed or pitted teeth; one of each sex had syphilitic teeth; at the same time I carefully examined the lenses of their eyes (the pupils were dilated with a midriatic), and only in one case did I find any trace or suggestion of a cataract, and in that one case the vision of each eye was perfect, i.e. Snellen's type $^{6}/_{6}$ was read with ease.

diagnostic indication of the period of growth during which the disturbing influence affected the lens of the eye.

This point has been very ably dealt with by Norman Bennett (Bibl. No. 22[b]), and his observations and conclusions should be examined. So far as dentition is concerned, he concludes: "That any cause tending to inhibit calcification during the first three years of life might be expected to show its effects on some part of the crowns of the first permanent molars, and on corresponding parts synchronously calcified of the incisors, cuspids, and bicuspids. Any cause operating later than the second year might affect either these latter or the second permament molars."

The coexistence of honeycombed teeth and lamellar cataract points to an affection after birth, and the condition of particular teeth may help to date the occurrence of that affection.

There has been much discussion concerning both the time and the mode of origin of production of the lamellar cataract; and although any elaboration of pathological evidence would be out of place here, yet some note must be made on these points, for they have a considerable bearing on the hereditary nature of the cataract.

It has been variously held: (1) That the opacity is a pre-natal or congenital, and therefore probably developmental defect. (2) That it is a post-natal or infantile defect, and due to pathological shrinkage of the lens. If the cataract be pre-natal and developmental in origin, then inheritance is of special importance. If the cataract be post-natal and pathological, then the conditions of infant feeding and health are likely to be of more importance than heredity.

The determination of these points has been thought to rest with: (1) The relation of the size of the lamellar opacity to the whole lens at different periods of growth. (2) The nature of the opacity forming the lamellar or zonular cataract.

On both these points the evidence is inconclusive. (1) The size of the lamellar opacity seen in children of varying ages has been measured, and compared with the size of the lens at birth by many workers. Almost all agree that the diameter of the lamella is smaller than the average diameter of the lens at birth. The natural deduction is that the defective lamella was laid down before birth. (2) The lamella of opacity is practically confined to one layer of lens fibres; but it is not wholly so confined, for evidence of shrinkage of the nucleus of the lens, in the presence of minute vacuoles and clefts, has been found by all observers (see Lawford's figure and others, Pl. L, Figs. 1, 2, etc.). The supporters of the pre-natal theory of origin allow that the nuclear changes are due to shrinkage in the nucleus subsequent to the faulty construction of an overlying layer of lens fibres. Their antagonists reply: If there be shrinkage of the nucleus subsequent to its proper formation, why may not the whole lamella itself be produced by a similar shrinkage also, just indeed as shrinkage produces cataract in old age? It is noteworthy also that the actual formation of lamellar cataract has been observed in young children; in a girl aged seven years by De Wecker, and in a boy aged three years by Grimsdale (Bibl. No. 25).

The argument on these lines is "stale mate." The opaque lamella may be pre-natal in site, but if it be caused by the pathological shrinkage of a layer of fibres previously normal in character and not by faulty development, then the site of the opaque lamella goes for nothing, for the shrinkage might take place in any part of the lens. On the other hand, the most ardent supporters of the post-natal origin cannot but agree that at any rate some of these cataracts are of pre-natal origin, for the cataract has been seen immediately after the birth, both by parents and by surgeons, in human infants; and similar cataractous lenses have been the subject of dissection immediately after birth in the cases of animals by Bowman and von Hippel (Bibl. Nos. 2 and 20).

It would appear that the congenital or pre-natal origin of many forms of cataract is best proved by the following considerations: (1) The distinctly hereditary nature of lamellar cataract, particularly of the small sized opacity. (2) The association of the lamellar type with other forms of lens defect in the same pedigree. (3) The occurrence of these several forms of cataract within families who are free from evidence of nutritional defects, from rickets, and from faulty dentition.

Rickets does undoubtedly coincide with the occurrence of lens changes, defects of dentition, and of deformities of the boney skeleton; and all the changes may be seen in the same child, or in several children of one pair of parents. It is even conceivable that both parent and child may be subject to the same conditions of life, and suffer the same disease (e.g. my case, Fig. 300). But it is wellnigh inconceivable that such a disease as rickets or any other disorder, acting for a limited period in the early months or years of infancy, should produce such eye defects as are tabulated in the pedigrees of lamellar cataract of Nettleship, or in one of mine (Figs. 289, 303 and 307). In the last of these pedigrees considerable variation in the size and density of the lamellar defect was found (see Plate L, Figs. 8 and 14), and these lens defects appeared in several generations and in many sibships; and it was a point of special remark that signs of rickets were absent, and that the teeth were particularly good. Such a physical defect recurring in several sibships living under dissimilar conditions of environment and risks to infant health, requires that the condition common to them must depend upon some stable character such as germ distinction, rather than on the effect of a more or less temporary disturbance such as rickets or infantile disorders.

1. (*b*) *Discoid Cataract.* Concerning the mode of origin, or defect in development, that is responsible for the appearance of discoid cataract we know nothing. If, as it would seem from its association with lamellar cataract in some pedigrees, the discoid cataract is a minute form of the grosser type called lamellar, then we may have little to learn concerning it; but the singularity of this minute, circumscribed, oddly placed and exceedingly delicate opacity remains a puzzle.

2. *Coralliform Cataract.* Of the origin of coralliform cataract little can be said, for no section has yet been made of such a cataract. It has been conjectured by Treacher Collins that the tube-like opacities that make the characteristic feature of this cataract lie in the planes of suture between the lens fibres.

3. *Stellate Cataract.* Similarly, no section has been made of a stellate cataract. But it is clear that the linear opacities affect the cortex of the lens only, and that the geometrical pattern they assume is exactly that of the lines of union of the extremities of the lens fibres, these same markings can frequently be made out in faint milky lines in healthy lenses, they appear to each one of us subjectively in examining distant points of light, and they are common as the denser markings of lamellar cataract (see Plate L, Figs. 9, 10). But how this singular opacity is produced we do not know.

4. *Anterior and Posterior Polar Cataract.* The lens fibres are surrounded by an elastic sheath known as the capsule of the lens, this has been shown to be formed from the lens proper as a secretion from its cells. During the growth of the lens there is surrounding it another sheath of fibro-vascular tissue. This second sheath has a main vessel of supply from the central artery of the retina, it passes straight from the optic papilla to the back of the lens by way of the hyaloid canal of the vitreous in the optical axis of the eye (Pl. M, Fig. 34). The sheath in front of the lens is connected with the iris and forms the "pupillary membrane." In the natural course of events this fibro-vascular sheath is absorbed and disappears before the birth of the human child; so that every trace of the sheath, the central artery, and the pupillary membrane is lost, and the optical axis becomes quite clear.

In some cases however this natural absorption is checked, or is incomplete, so that there may persist, either in front or on the back of the lens, fragments or a plaque of opaque tissue.

If such a plaque did not involve the lens substance it would not, strictly speaking, constitute cataract, and therefore it has been excluded from the category by some (Parsons). But in my cases, Figs. 339 and 441 (see Plate M, Figs. 30 and 33), the body of the lens was affected. Treacher Collins has brought evidence to show that the persistence of these remnants of the fibro-vascular sheath is due to a faulty growth in the proper capsule of the lens. Gaps are left in it, and these gaps are plugged, as it were, by the retention of the fibro-vascular tissue. If this explanation be true we should expect to find that these polar cataracts are hereditary, at least in some cases. For the fault in the growth of the lens capsule is of early date in the development of the child, and considerably anticipates such faults as produce lamellar cataract. Such pedigrees are to be found; they are not numerous, but they are sufficiently striking even in their small number. It is worth noting that in most cases the eyes affected with anterior and posterior polar cataracts are small eyes; the particular defect in development has been followed by a more or less general arrest of growth of the eye. Further, the subjects of these defects are, in the particular pedigrees I investigated, stunted in body and feeble in mind, so the determining factor of this failure in development must have been of a grave order.

In the succeeding pages and Plates XXVIII—XXXII details of 95 pedigrees are given, embracing 36 pedigrees of lamellar cataract proper, 5 of discoid cataract, 11 of coralliform cataract, 7 of polar cataract, 2 of stellate cataract, and 36 of "congenital cataract" of which the type of opacity was not defined or not stated. These pedigrees

cover some 2685 persons and include 614 certainly and 26 probably affected subjects, who are distributed in the two classes as follows: 264 + 7 male and 257 + 13 female, 93 + 6 sex unstated[1]. Some of the pedigrees are small and obviously incomplete: others are of splendid proportions, and have been worked out with the greatest care and in the strictest detail; their value cannot be over-estimated in the study of heredity. In these pedigrees the facts of the inheritance of cataract are spread open before the reader; but it does not lie with me to draw conclusions therefrom, or to point the moral.

In concluding this section, I would seize the opportunity to express my indebtedness to Mr Nettleship, not only for the broad base which his earlier work in collecting and classifying pedigrees of cataract has made for me to build upon, but also for the help and guidance he has drawn for me from the wealth of his experience.

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Supplementary Pedigrees.

Several pedigrees have been obtained since Plates XXVIII—XXXII were engraved. Consequently they could not be placed in their appropriate groups, but follow as a supplement. The nature of the cataract is not clearly stated in some; in others, especially *Norrie's Case*, it is probably congenital. Further cataract pedigrees, unpublished congenital cases and published hereditary but non-congenital cases will appear in a later number of the *Treasury*.

HEREDITARY CASES.

GROUP I *a*. *Lamellar Cataract.*

PLATE XXVIII. Fig. 276. *Horovitz's Case.* Lamellar cataract. II. 1 was seen, aet. 50, with lamellar cataract, her right eye had been weak from early age, the left from aet. 13. Her mother had good sight, but her father always had bad sight, cause unknown. III. Four children, two were normal, of these: III. 1 had seven children all normal; III. 4 had four all normal, the youngest had rickets. Of the two cataractous siblings: III. 2 had cataract (diagnosed, aet. 16), but not rickets, and gave birth to two cataractous children, of whom one had rickets, but good teeth; the other neither rickets nor fits. III. 3 had cataract (diagnosed, aet. 35), and had four children, all short-sighted, but not cataractous; the eldest had "elephantiasis," and the youngest rickets. No note made of consanguinity. (Bibl. No. 78.)

Fig. 277. *Appenzeller's Case*, is that of a father and two sons. The father I. 1 was born with cataracts, which were operated upon about aet. 10, and he still saw pretty well at date of record (1880), when his age must have been 60, more or less. He had six children, of whom II. 3 and 4, both male, were operated on for lamellar at the ages of nine and six years respectively, in 1872. The rest of the sibship was normal. No family history of eye disease or congenital anomalies. (Bibl. No. 58, (vi).)

Fig. 278. *Sir Wm. Adams' Case.* Four siblings, of whom the first-born, II. 1, a girl, had good eyes. The second, II. 2, a girl, had "slate-coloured cataracts, with transparent edges" (doubtless lamellar cataract), when seen at aet. 13. II. 3 (boy) and II. 4 (girl, aet. seven), had "fluid" congenital cataracts; in II. 3 the opacity was discovered when he was a month old. All operated upon with success. (Bibl. No. 34.)

Fig. 279. *Nettleship's Case.* M—— family. II. 3 and 7, sister and brother, in a sibship of seven had small lamellar cataracts, diagnosed in each case at aet. 14; no fits, teeth very good. II. 1, 2, 4 and 6 had good sight; II. 5 died a few weeks after birth. (Bibl. No. 82, Case 72.)

Fig. 280. *Galezowski's Case.* Cataract in four generations, proved to be of lamellar type in last two. Females only affected; uncertain if males were recorded in generations I. to III. I. 1 and II. 1 operated for cataract. III. 2, "incomplete zonular cataract," III. 3 described as having incomplete zonular cataract. IV. 2, 3, 5 described as having cataracts like their respective mothers. No consanguinity recorded. (Bibl. No. 54.)

Fig. 281. *Nettleship's Case.* Pickett family. Pedigree shows only those who had cataract, number and order of siblings not known. Lamellar cataract. I. 1 had cataracts. II. 1 bad sight as a child, and consequently learnt but little at school. Operated for cataract, aet. 30. III. 2, aet. 40 (1883), vision never good enough to allow of reading newspapers; getting worse up to eight years; now sees letters of 20 Jäger; sharply defined anterior and posterior cortical spokes and "indications of clear margin as in lamellar." Left operated upon with good results. Teeth very bad, most of upper ones gone. No history of fits. III. 3, vision always defective; operated for cataracts, aet. 16. IV. 1, aet. ten. Small lamellar cataracts just filling, moderately small pupil; no spicules, cortex quite clear, but rather too visible. Double iridectomy performed. Teeth not honeycombed. (Bibl. No. 82, Case 75.)

Fig. 282. *Hosch's Case.* I. 1, an only child. Operation for cataracts, aet. eight; good result in one eye, moderate in other. II. 1, male, one of five, but which one not noted. Born with cataracts, diagnosed as lamellar by Horner when aet. 38, at which time artificial pupils made. Teeth slightly honeycombed. II. 2, boy, died, aet. four. II. 3, 4, 5, sisters with good eyes. Have between them 14 children, all with good eyes. III. 1, female, born 1872, six toes to each foot, walked at 12 months; dentition normal; cataracts seen by mother, aet. two days, by Horner, aet. six weeks, and again six years, and had not increased; diameter of opaque shell about 4 mm. III. 2, female, born 1882; cataract seen by mother just after birth; no fits; no rickets; lamellar cataracts with clear cortex. III. 3, female, born 1885. History and condition same as III. 1 and 2. Double shell lamellar opacity. III. 4 to 11, seven others, sex and position in sibship unknown; free from cataract. (Bibl. No. 71.)

Fig. 283. *Saunders' Case.* The description and illustration given by the author indicate typical lamellar cataract in three brothers, II. 1—3, and one sister, II. 4. In the same publication he mentions three other familial cases, but, as he gives no description of the opacity, we cannot say positively that these cases were of the lamellar kind. (Bibl. No. 33.)

Fig. 284: see p. 158.

Fig. 285. *Storbeck's Case.* Familial lamellar cataract. The author gives the scheme of a family tree, and states that there was consanguinity on both sides, and in more than one generation. II. 2 and 3 are definitely stated to be brother and sister; II. 1 and 6 had the same name; and III. 2 and II. 4 also had the same name; II. 5 and 6 are connected with III. 4 and 6 by dotted lines in author's chart. Cataract occurs only in generation IV.; in which are seven cases of lamellar cataract, in three sets of siblings, containing 18 children. All the 18 appear in the original to be of the same generation, and all descended from a brother and sister (II. 2 and 3), who had good eyes, and were grandparents to the affected generation. In the youngest (IV. 18), a child aet. 14 months, the cataract was diffuse in one eye, and typically lamellar in the other. In another, aet. 22 (IV. 2), one eye showed a double shell of opacity and opalescence of the cortex, whilst the other eye contained a simple, rather small lamellar cataract. All the others had typical lamellar cataract in both eyes. Of IV. 11 to 18 it is stated that the children with cataract were like the father and the normal children like the mother. Storbeck suggests that the consanguinity had some bearing on the appearance of the cataract in the fourth generation. (Bibl. No. 49.)

Fig. 286. *Von Hippel*, quoted by *Daust*, knew of a mother, I. 1, with lamellar cataract, of whose five children three, II. 1—3, were similarly affected. No further details. (Bibl. No. 74.)

Fig. 287. *Nettleship's Case.* Dearsley family. Lamellar cataract. A small pedigree of two affected in a sibship of six. It is of interest on account of the history: the cataracts had been seen in IV. 2, within one day of birth; an operation was performed at 11 months. Permanent teeth good. IV. 4 was operated (iridectomy) aged five years for small, very dense cataracts, 4 to 5 mm. diameter. Permanent teeth good. The parents III. 3 and 4 were first cousins, at marriage father was aet. 20, mother 24; the father is an only child, the mother fourth of a sibship of seven, of whom the sixth and seventh died under a year old, the third died aet. 40, and the rest aet. from 63 to 72. The marriage between III. 3 and 4 is the only consanguineous one known. In giving this pedigree Nettleship places beside it a pedigree of a family named Deasley or Darsley who show many cases of pre-senile cataract. The two families originally came from neighbouring districts in the eastern counties of England, but no certain connection between the families can be traced. (Bibl. No. 88.)

Fig. 288. *Harman's Case.* B—t family. Lamellar cataract. This family is scattered widely. I., II. and the right wings of III. and IV. reside in several villages in the West of England; for information concerning them I have to thank the Rev. M. A. Bere, vicar of Wareham, Dr Du Boulay of Weymouth, and Dr Morgan, late of Puddletown; the left wings of III. and IV. I have investigated myself. I. 1 and 2 presumed normal: I. 3, male, dead, had cataract, his daughter says he had no brothers or sisters affected; I. 4, his wife, good sight. II. 1 and 2, two brothers and two sisters of 3, presumed to be normal, the brothers migrated to Australia, one sister died of cancer, the other of "consumption." II. 3, male, aet. 69, alive, perfect eyes. II. 4, his wife, the daughter of a cataractous father, aet. 71, "both eyes operated by Dr Fox, 50 years ago at Weymouth Eye Infirmary, for congenital cataract, since had fairly good sight in right eye, left never so good; examined, aet. 59, probably detached retina in right, left fair vision with glass." II. 5, female, died aet. 45, had "beautiful sight"; has three normal children, III. 6—7, and one grandchild, IV. 7. II. 8, female, aet. 66, operated on many years ago for cataract, now nearly blind; married first, normal male II. 7, had five children, one weak-minded, two very bad sight, cause uncertain; married secondly, II. 9, normal male, no children. II. 10, female, aet. 65, "born with cataract, operated aet. 12, not married. II. 11, reported by nephew (III. 2) to have had bad vision just like his three cataractous sisters; he cannot now be traced. III. 1, 2 offspring of affected mother. III. 1, male, died aet. 16, operated for cataract as a child. III. 2, a skilled workman, aet. 46, with excellent vision—$\frac{6}{6}$ and Jäger 1 with each eye—yet he has the faintest order of lamellar cataract with a few denser spots in the anterior polar region; married a normal eyed woman; has six children, three of whom had dense cataracts and bad vision after successful operations. III. 8—14, the sibship of five by affected mother, II. 8. III. 8, female, normal, has three normal children, though two, male, aet. 16, and female, aet. 25, have "weak eyes" and blepharitis. III. 10, female, very bad sight, cause not certain, not married. III. 11, female, good sight; has one normal infant son. III. 13, male, good sight, "weak-minded," unmarried. III. 14, male, nearly blind, not seen for some years, has several children. IV. 1—6, sibship from affected father, normal mother. IV. 1, male, now aet. 21, cataracts operated years ago, teeth good. IV. 2, male, died aet. 15 months of rheumatism. IV. 3, male, aet. 17, cataracts operated early, fair vision, good teeth. IV. 4, male, aet. 15, eyes and teeth normal. IV. 5, male, aet. 13, eyes and teeth normal. IV. 6, female, aet. 11, cataracts removed several years ago. IV. 1, 3 and 6 were operated by McHardy, he writes: "I well remember the series as examples of lamellar cataracts, one child had three dense white specks in a small and otherwise uniformly opaque lamella, with some radiating striae or faults passing into the cortex; another had a double lamellar cataract in each eye, an inner and outer shell of opacity." See Plate L, Fig. 12. (Bibl. No. 93.)

Fig. 289. *Nettleship's Case.* Everett family. Lamellar cataract. Four generations affected. The cataract was uniform with slight variations in all those affected. The shell of opacity was dense, seldom transmitting any red light from the fundus with an undilated pupil; sharply defined and circular, except in one or two, where it was bluntly triangular, sometimes with a few projecting spokes, but never with any separate riders or other evidence of a second layer of opacity. It measured 3·75 to 4·5 or 5 mm. in diameter. The cortex was clear, except in one case where it was slightly dim. In no case was there a history of fits, and there were few, if any, defects of dental enamel. No early deaths are recorded. No consanguinity of any parent. Associated with the lens defect was congenital ptosis of the upper lid in two cases (IV. 2 and 5). Goitre was grafted into the family by III. 7 and IV. 18, but only one case of the disease resulted, V. 10. In some of the cases cataracts were seen very early in life. IV. 3 told Nettleship she had been able to see the cataract by opening her baby's eyes (V. 9) almost as soon as the nurse had washed it, immediately after birth. The mother was intelligent and observant, and disconcerted by having borne so many children with imperfect sight (six affected out of nine) and very solicitous to have normal ones; she had therefore looked at the eyes of her younger children critically, immediately after birth. I. 1 and 2, no information except that there was no blood relationship. II. (all dead at time of enquiry), II. 4, male, one of about 11 brothers and sisters, always had bad sight, but did not go blind; no report of bad vision in his brothers and sisters. The man married twice, neither wife bore blood relationship to him or each other; by the first wife he had one child who had cataract; many years later he took to wife a woman twenty years younger than himself, and had three children, two of whom had cataracts. III. 3, now aet. 70, fine tall man; sight always defective, small dense lamellar cataracts with a few radii, opacity 4 mm. diameter. III. 4, male, similar cataracts, unmarried. III. 5, male, normal eyed, has five normal children. III. 6, female, cataracts removed "in her teens," unmarried. IV. 2, male, small lamellar cataracts with wide clear cortex, teeth good; has decided congenital ptosis, not married. IV. 3, female, cataracts, married and has nine children, of whom six have cataracts. IV. 4, male, circular sharply defined lamellar cataracts, 4 to 4·5 mm. diameter, cortex faintly opalescent, sharply defined Y-shaped opacity at anterior pole of each lens; lower incisor teeth good; married, no children. IV. 5, male, small sharply defined cataracts, 3·5 to 3·75 mm., a broad limbed Y-shaped opacity of dense white on anterior surface of opaque zone; slight congenital ptosis of upper lids; has three children, all cataractous. IV. 6, female, and IV. 7, male, normal; the latter has one child, normal. IV. 8, male, small dense clean edged lamellar cataracts; fairly good vision, served seven years in the army; upper incisor teeth good, lower slightly defective; has two children, both cataractous. IV. 9, male, small dense lamellar cataracts; teeth good; unmarried. IV. 10, female, like preceding, has four children, two cataractous. IV. 11, female, similar, but with anterior polar denser spot of opacity; has two children cataractous. V. 1 to 9, children of IV. 3; the first four (two males, two females), and last two (one male, one female), had cataracts; in the last two the mother noticed the defect within a short time after birth. V. 5 and 7, males, and V. 6, female, were normal. V. 10 to 13, children of IV. 5. V. 10, female, lamellar cataract 4 to 4·5 mm. diameter, with white anterior polar spot and some spokes. V. 11, died aet. 11 months, but mother is sure he had the family cataract. V. 12, aet. six, small cataracts, and Y-shaped anterior polar opacity like his father. V. 14, male, aet. ten, cataracts, subtriangular in outline with dense anterior polar opacity, noticed aet. three months. V. 14*a*, male, seen by Nettleship, aet. seven days, typical cataract. V. 15, female, aet. six, cataracts removed. V. 16 and 17, males, normal. V. 17*a*, female, died seven months after birth of wasting and convulsions, good evidence from parent and aunt of cataract. V. 18, female, cataract noticed by mother within an hour or two after birth. V. 18*a*, female, seen by Nettleship 14 days after birth, typical cataracts. (Bibl. No. 88.)

Fig. 290. *Fromaget's Case.* Congenital, probably lamellar cataract. Cataract affects five generations. Transmission, except for first generation, always took place through the mother. The opacities in the lenses of the children (V. 1, 4, 5) consisted of "alternate opaque and clear layers." I. 1 and 2, males, reported to have had congenital cataract. II. 1—4, four daughters who had cataract from birth, only one could be traced; she had two children. III. 1, sex not stated, cataractous; III. 2, female, cataract was complete. III. 2 had fourteen children, of whom two were affected. IV. 1—3, sibship of preceding, IV. 1 male, IV. 2 female, both born with cataract; son is unmarried; the daughter has married, and at aet. 28 has six children. V. 1, female, aet. 11, operation aet. eight; V. 2 and 3, normal, sex not stated; V. 4, male aet. five, and V. 5, female, aet. $3\frac{1}{2}$, had double congenital cataract. V. 6, sex not stated, died aet. three weeks, believed to have been free from cataract. No consanguinity. (Bibl. No. 65.)

Fig. 291. *Hirschberg's Case* (i). H. operated on a girl, II. 1, aet. 15, who had double-layered lamellar cataract of both eyes. Her mother, I. 1, had complete uncomplicated cataract in one eye, and congenital strio-punctate cataract in the other. No other details recorded. (Bibl. No. 47.)

Fig. 292. *Hirschberg's Case* (ii). H. narrates the case of a child, III. 1, aet. five, with double cataract, whose father, II. 1, aet. 30, had suffered from cataract since the fifth year of life, and had been operated upon by von Graefe at the age of 20; whilst a brother of the father, II. 3, now aet. 32, who had had iridectomies in 1864 by von Graefe, was now found by Hirschberg to have lamellar cataracts. No other details recorded. (Bibl. No. 47.)

Fig. 293. *Hirschberg's Case* (iii). H. found lamellar cataract in both eyes of a child, III. 1, aet. one year, and ascertained that cataract becoming complete at the twentieth year of life had occurred in the two previous generations. No other details recorded. (Bibl. No. 47.)

Fig. 294. *Nettleship's Case.* Hawkins family. II. 8, female, seen 1878, aet. 17, with very small stationary lamellar cataracts; high myopia about 10 D. each eye; V. $\frac{6}{6\cdot0}$; teeth perfect; iridectomies improved vision; she is seventh of eight born. II. 1 and 9, both male, said to be short-sighted. III. 2, male, aet. 12, had had cataracts removed from each eye; diagnosis, "lamellar cataracts," but descriptive notes say "irregular axial opacity from centre of lens to posterior pole"; small myopic crescent; vision only slightly improved by operation; marked nystagmus; enamel of teeth defective; is second born and only living of three. II. 1 said to have had one eye affected; no consanguinity recorded. (Bibl. No. 82, Case 74.)

Fig. 295. *Bresgen's Case.* I. 1, male, aet. 58, right eye perinuclear cataract; left, recently complete cataract; sight bad in both eyes all his life; of his eight children, six had cataract. II. 1, eldest daughter, aet. 25, could never see enough to read or write; now, left partial cataract, and right total cataract, which has become rapidly complete during the last year. II. 2, second daughter, aet. 23, not able to read or write since aet. 5, owing to lamellar cataracts with central capsular opacities in addition. II. 3 to 6, cataracts, no details given. II. 7 and 8 had good eyes. No other details or note of consanguinity. (Bibl. No. 46.)

Fig. 296. *Nolte's Case.* Steitz-Heppel family. I. 1, female, aet. 47, nuclear and general cataracts, age at onset not given; extracted successfully. I. 2, her brother, blind from cataract, aet. 43. II. 1—3, seven children of I. 1. II. 1, female, had "indications of hare-lip." II. 2, female, was extremely fair and the fundus of the eye lacking in pigment (? partial albinism—this word not used by author); others normal. II. 4—8, children of I. 2. II. 4, female, posterior polar and zonular cataracts both eyes. II. 5, female, and 6, 7, males, congenital deficiency of pigment (like II. 2, ? partial albinism); no note of vision. II. 8, idiot boy, over-ripe cataracts. (Bibl. No. 68.)

Fig. 297. *Nettleship's Case.* S—— family. I. 1, no note. 2, mother, light flaxen hair, good vision. II. 1, male, aet. 34 (when seen in 1890, date of record), good vision; dark striped choroids; fair hair and skin. II. 2, female, aet. 30, lamellar cataracts; defective intellect; honeycombed teeth; many infantile fits. II. 3, female, aet. 28, faintest possible degree of lamellar cataract; honeycombed teeth. II. 4, male, aet. 27, 5 D. myopia when aged 16; honeycombed teeth; no fits; no note of lenses, but visual acuity good. II. 5, male, aet. 26; right eye myopic, left, emmetropic at aet. 16. II. 6, male, aet. 24, myopic, vision good at aet. 19; very fair hair and white skin, but dark striped choroids; no consanguinity recorded. (Bibl. No. 82, Case 71.)

Fig. 298. *Nettleship's Case.* M—— family. No note of parents. Family of four sons, daughters not mentioned. II. 1, aet. 21, when seen by Nettleship; lamellar cataract operated aet. 13; bad teeth, and history of convulsions. II. 2 and 3, no cataract; perfect sight; good teeth; no convulsions. II. 4, aet. 16; R. 9 D., L. 3 D. myopia; lamellar cataracts, faint and rather large, but opacity more dense at posterior pole; had convulsions, and one incisor tooth deficient in enamel. (Bibl. No. 82, Case 73.)

Fig. 299. *Nettleship's Case.* Poulton family. No note of parents or other children than the three with cataracts. II. 1, female, aet. 13 (in 1882), small, very dense lamellar cataracts, smaller than the ordinary undilated pupils; projecting spokes, cortex otherwise clear; both operated on and did well; teeth honeycombed; screaming convulsions during teething, and many "powders" taken; always delicate. II. 2, female, aet. seven (in 1882), rather dense lamellar cataracts; no note of size; operations did well; was treated for fits in infancy. II. 3, male, aet. 12 (in 1892), large, dense lamellar cataracts, seven to eight mm. in diameter, with spicules; teeth honeycombed; had fits aet. eight months, and was seen for eyes aet. 14 months. (Bibl. No. 82, Case 75.)

PLATE XXIX. Fig. 300. *Harman's Case.* M—— family. II. 1—5, one sibship; all said to have normal eyes, all married. II. 1—4 had several normal children each. II. 5 was a tailor, and had "perfect sight"; he married the sixth of a sibship of eight; this woman, II. 11, is still alive (1909), aet. 80 years. Of the rest, II. 6 and 10 had normal families; II. 7, 9 and 13 died unmarried; II. 8 and 12 died young. III. 2, male, aet. 49, has lamellar cataracts, worse in right eye; teeth badly honeycombed; he had fits very badly as a baby. III. 3, 4, 5, 6 are all normal; they married late in life, so have small families, except III. 5, who has no issue; the children are normal. IV. 1—5, offspring of III. 2, father affected, mother normal. IV. 1, male, died aet. two, did not have fits, eyes thought to be good. IV. 2 and 3 died shortly after birth. IV. 4, male, aet. 14, sight found defective one year ago, right eye fine lamellar opacity, left eye well-marked lamellar cataract of large size; central incisors and first molars badly honeycombed; he had fits in infancy. IV. 5, male, died shortly after birth. The occurrence of the same order of defect of eyes and teeth in father and son is suggestive of a germ inheritance; defect in germ plasm may be held supported by the feeble viability of this sibship, one survives in five; but there are no similar defects in other members of a fairly large tree. (Bibl. No. 90.)

Fig. 301. *Grimsdale's Case.* I. 1, sight "bad," cause unknown. II. 1, unknown. II. 2, operation for cataract in youth. Of sibship III. 1—8 four were affected, four were normal-eyed; of sibship III. 9—15 four were affected, three were normal-eyed. IV. 6, male, had lamellar cataract and ridged teeth; nine others in his sibship were normal-eyed. The order of the siblings in the various sibships not known; no details concerning consorts, but probably normal. (Not previously published.)

Fig. 302. *Kunn's Case.* I. 1, cataract in early life, no details of its character. II. 1, operation for lamellar cataract aet. six; square "rickety" skull. III. 1—4, order of birth not given; two boys, aet. seven and five, had lamellar cataract, diagnosed early; the younger, III. 2 (first seen aet. $1\frac{1}{2}$ years), showed a three-fold layer of opacity; they had had no fits, but their bones were rickety and they had "rachitic" teeth (the youngest boy's teeth would still be the milk set). (Bibl. No. 60.)

Fig. 303. *Nettleship's Case.* Smith family. Comprises 58 members; all the living members were seen. I. 1—2, nothing known. II. 1—10, ten siblings, of whom five, 2, 5, 7, 8, 9 died in early life; five are alive, all see well except one, II. 1, male, who had operations by Sir Wm. Bowman at Moorfields, first in one eye then in the other, between the ages of 10 and 13 (1847, etc.); the entry in the Register on each occasion is "Congenital cataract"; he has now been blind for 15 years. II. 6, female, normal, is married, and has three normal children, all living; II. 10, a Thames pilot, is normal, and has ten children all living and normal. III. 1—7, children of II. 1; III. 1, male, cataract diagnosed by George Lawson, aet. 18; now aet. 40 (1908), seen by Dawnay; he has dense lamellar cataract in each eye, the right showing a double shell of opacity. III. 2, female, lamellar cataracts removed in 1886, aet. 18; upper incisors gone, lower imperfect enamel. III. 6, female, was operated on for lamellar cataract (aet. 9 in 1886) by Treacher Collins; her first molar teeth were deficient in enamel. III. 3, 4, 5 and 7 are free from cataract. III. 7 married a cousin, but has normal children. Generation IV. has contained 25 persons. The first sibship (IV. 1—9) of nine was the issue of III. 1, who had cataract. IV. 1, 2, 3, 7 died early of measles, &c. IV. 5 was born dead; IV. 4, male, aet. 11, IV. 6, male, aet. eight, IV. 8, female, aet. seven, and IV. 9, female, aet. four, are living; Nettleship examined all and found their lenses perfect. Only the eldest has permanent teeth, and they are normal. They are healthy, well-grown children, and very intelligent. Sibship IV. 10 to 22, children of III. 2, who was cataractous, contains 12 children, including twins twice. Five only are living, and were seen. IV. 10, male, IV. 11—12, females, IV. 16, male, IV. 17, female, all have cataract; three have had operations, but IV. 10, aet. 14, and IV. 17, aet. seven, have typical lamellar cataracts and need operation (March, 1906). Of the other siblings, IV. 13, died aet. three years; IV. 18, died aet. five months, and was probably an idiot; IV. 19, died aet. 11 or 12 months. In each of the two twin-pregnancies, one miscarried at four months, the other (a boy in each instance) was born at full term, one of them dying aet. five months, the other aet. 12 months. Thus every one of the five who were born alive and survived had lamellar cataract. In regard to the teeth of these five children, the permanent incisors are quite sound and well shaped in all who have cut the permanent set (in the youngest, IV. 16 and 17, only the lower permanent incisors are up); the first permanent molars show deficient enamel in IV. 10 and 11, but are perfect in the other three. Some of the younger children had convulsions. All the five living children are bright, intelligent, and well nourished, but the first and second are very under-grown. III. 6, cataractous, has only one child, IV. 23, believed to be normal. The normals III. 3 and 7 have normal children. No consanguinity recorded in affected branches; one marriage of unaffected cousins, children normal. (Bibl. No. 82, Case 108.)

Fig. 304. *Nettleship's Case.* Horley family. Mother, I. 1, aet. 49 in 1883, sight failing 20 years, cortical cataracts, close beneath anterior capsule, in form of fine dots, and large and small spokes; all parts of surface cortex affected except anterior pole, which is quite free. Son, II. 1, under care for lamellar cataracts. No other data. (Bibl. No. 82, Case 79.)

Fig. 305. *Davidsen's Case.* Lamellar cataract seen in both eyes of two brothers, II. 1 and 2, and a sister, II. 3, aet. 31, 29, 24 years respectively. No other data. (Bibl. No. 44.)

Fig. 306. *Nolte's Case.* Becker family. I. 1, cataract formed in both eyes after whooping cough at age of 12, both eyes operated on. I. 2, her husband had good eyes. Issue, five children: II. 1, male, normal-eyed, has four normal children, III. 1. II. 2, female, cataract, said to be lamellar, not examined. II. 3, male, "lamellar" cataract diagnosed soon after birth; at about aet. 6 optical iridectomy performed in each eye; when aet. 18 (1879) patient was badly developed, severe nystagmus, corneal diameter 10 mm. (average is 11·6), in each lens a central opacity with projections into the cortex, and in right the cortex is cloudy; right needled then, and again in 1889. In 1895 right vision with + 10 D. equal fingers at 3 metres, some remains of lens. Left cortex now opaque in coloboma, vision fingers 1·25 metres, lens removed; result, vision with + 10 D. fingers at 10 m. II. 4, female, normal-eyed; has one normal child. III. 5, sex not stated, with lamellar cataract, died in early infancy. (Bibl. No. 68.)

Fig. 307. *Harman's Case.* Turner family. The opacities observed in the lenses of this family were of small size, and vary from complete spherical lamellar opacities of sharply defined contour, to small dense white opacities situated immediately anterior to the lens nucleus, and taking the shapes of Y, X or ✳.

The position and general contour of these latter markings suggest the possibility of their being allied to the smaller forms of the coralliform cataract. Cases have been seen in ages ranging from 70 years to three years; there is no evidence of increase in the density of the cataract with age. Senile cataract does not appear in the family. The teeth were good; only one case was found with ridged teeth of slight degree, IV. 24. The consorts were examined where possible; there was no case of in-marriage, and in no case was a consort affected. III. 3 married twice; by the first wife he had two cataractous and two normal children; by the second wife (a widow who had normal children by previous husband) he had one normal child. The intelligence of the family, especially in the affected branches, is only fair; one member, IV. 1, is mentally defective, and one, III. 12, is now insane. The inheritance is always through affected members. Males and females transmit equally; in one line the transmission is through male-female-male; in another it is through male-male-female. Of the affected subjects, ten are males, nine females. Of 55 persons who lived beyond infancy, 41 were investigated by the author, and 19 had cataract. This pedigree shows well the necessity for personal investigation of these histories, for in one sibship of seven members (IV. 23—29), only one bad-sighted member was acknowledged, but four were found to be cataractous. In another sibship of five, III. 1—5, cataract was acknowledged in two, but examination showed four were affected. The discrepancy in report and finding arose from ignorance of the fact, and not from any idea of deception, for several of the affected subjects had quite good vision, notwithstanding the lens opacities. I. 1, male, dead, report of cataract from two branches of his family; his wife reported to have excellent sight. II. 1, female, died recently, had cataracts removed in childhood. II. 2, female, died many years ago, said to have had good sight. II. 3, male, died a few months ago (1909), small-sized congenital cataracts; his wife has perfect eyes. II. 4, female, dead many years; three branches of family state she had cataract from childhood. II. 5, female, eyes said to be good, "reads very fine print with ease." III. 1—5, offspring of II. 1, mother cataract, father normal. III. 1, male, dead, had operations for cataract in childhood. III. 2, male, dead, hospital record found, "congenital cataract, 1863." III. 3, male, aet. 49, small congenital opacities in each lens just anterior to nucleus; vision good; teeth normal; married twice, wives unrelated to him or each other; daughter of second wife by previous husband seen, eyes normal. III. 4, female, aet. 46, lens opacities like preceding, teeth normal. III. 5, female, aet. 43, eyes and teeth normal. III. 6—12, sibship of II. 2, parents reported good sight. III. 6, female, aet. 50, eyes and teeth normal. III. 7, female, and 8, male, died in infancy. III. 9, female, died of phthisis, schoolmaster reports good vision. III. 10, male, died at birth. III. 11, male, aet. 40, eyes and teeth normal. III. 12, female, aet. 38, inmate of lunatic asylum, eyes normal. III. 13—20, sibship of II. 3, father cataractous, mother normal. III. 13, female, aet. 40, cataracts removed in childhood, teeth good. III. 14, male, aet. 38, same as 13. III. 15, female, aet. 35, well-marked, hard-edged, congenital lamellar cataracts of small size (see fig. 8, Plate L), optical iridectomies give fair vision, teeth good. III. 16 and 17, twins, III. 16, female, died aet. $2\frac{3}{4}$ years, "thought to have had good eyes"; III. 17, male, born dead. III. 18, female, lives in Madagascar, said to have perfect sight. III. 19 and 20, twin males, eyes and teeth perfect. III. 21 and 22, males, children of III. 4, cannot be traced, said to have had good sight. III. 23, male, aet. 30, perfect eyes and good teeth. IV. 1—5, first family of III. 3, father cataractous, mother dead, said to have had perfect sight. IV. 1, male, aet. 26, mentally defective, minute opacities each lens like father, teeth good. IV. 2, male, aet. 24, cataractous lenses removed in childhood, teeth good. IV. 3, female, died aet. 17 of phthisis, sight said to have been good. IV. 4, female, aet. 15, eyes and teeth good. IV. 5, male, aet. 4, normal eyes, he is child of III. 3 by second wife (whose daughter by previous husband has normal eyes). IV. 6—10, offspring of III. 5, mother normal; father dead, said to have had perfect eyes; IV. 6, male, aet. 27, in S. Africa; IV. 9, male, aet. 21, postman; IV. 10, male, aet. 19, all not seen, eyes said to be perfect; IV. 7, female, aet. 26; IV. 8, female, aet. 23, eyes and teeth excellent. IV. 11—15, offspring of III. 6, mother normal, father dead, said to be normal; IV. 11, female, aet. 20; IV. 12, male, aet. 18; IV. 13, female, aet. 15; IV. 14, male, aet. 12; IV. 15, male, aet. 8; all have perfect eyes and good teeth. IV. 16 and 17, offspring of III. 11, parents' eyes normal; IV. 16, male, aet. 12; IV. 17, female, aet. seven, eyes and teeth normal. IV. 18 and 19, offspring of III. 13, mother cataractous, father normal; IV. 18, male, died in infancy; IV. 19, male, aet. 15, both lenses removed for cataract, teeth good. IV. 20—22, offspring of III. 14, father cataractous, mother normal; IV. 20, female, aet. 13, cataracts removed, teeth good; IV. 21, male, aet. 10; IV. 22, female, aet. 5; both have normal eyes and teeth. IV. 23—29, offspring of III. 15, mother cataractous, father normal (all said to have perfect sight, save 26, but on examination four were found cataractous); IV. 23, female, aet. 13, 5 D. of hypermetropia, lenses and teeth normal; IV. 24, female, aet. 11, small opacities in front of nucleus of each lens, Fuchs' colobomata of optic discs, teeth slightly ridged; IV. 25, female, aet. nine, similar lens opacities, right Y, left X-shaped, teeth normal; IV. 26, female, aet. six, dense lamellar cataracts occupying middle three-fifths of each lens, removed with good results, teeth normal; IV. 27, female, aet. 5, 3 D. of hypermetropia, lenses normal; IV. 28, male, aet. three, both lenses have delicate lamellar defect and ✳-shaped opacities just anterior to nucleus, Plate L, fig. 14; IV. 29, female, aet. six months, lenses quite clear. IV. 30—33, offspring of III. 18, emigrated to Madagascar; IV. 30 and 32 died at birth; IV. 31 and 33, females, and said to have perfect sight. V., only one member as yet, a boy, V. 1, aet. three, a normal-eyed child of normal parents. (Bibl. No. 87.)

Fig. 308: see p. 155; Fig. 309: see p. 158; Fig. 310: see p. 156; Figs. 311—313: see p. 158; Fig. 316: see p. 152.

Fig. 314. *von Arx's Case.* In a tabulation of 189 cases of cataract the author cites this and the succeeding case; in both there is hereditary or familial cataract. Ch. Sp——, II. 1, female, aet. 37; zonular cataract $6\frac{1}{2}$ mm. diameter; right removed, left iridectomy; vision fair; suffered badly from fits. R. Sp——, II. 2, female, aet. 38; sister of foregoing; cataract in right eye $5\frac{1}{2}$ mm. diameter, left 6 mm. diameter; fits, honeycombed teeth; iridectomies gave very fair vision. (Bibl. No. 56, Cases 100 and 101.)

Fig. 315. *von Arx's Case.* H. W——, female, aet eight; zonular cataract in both eyes of extraordinary delicacy; with congenital capsular opacities of pyramidal shape, very small in left, very large and white in right eye (cf. Plate L, fig. 14); the thicker inner surface of the opacity dipped down into the anterior layer of the zonular opacity; the white spots were noticed fourteen days after birth; vision bad; father also had spots in the eyes; mother short-sighted. (Bibl. No. 56, Case 71.)

PLATE XXXII. Fig. 348. *Harman's Case.* W——t family. The information concerning generations II. and III. comes from IV. 2. Such of his statements as are capable of verification have proved correct. II. 3, the father of gen. III., the third of four siblings, died aet. 68, he was well known to his grandson who heard no suggestion of bad sight in him or his family. II. 8, his wife, was the youngest of twelve siblings, there is no recollection of her, but it is remembered that one of her four brothers, II. 5, was "always nearly blind." There was no consanguinity in this marriage; the issue was a sibship of twelve children of whom two are known to have been cataractous. III. 1—3 are in their proper order. III. 1, male, died aet. 68, practically blind from cataract; married a normal woman, and had three offspring, of whom two are cataractous; III. 2, female, died aet. 46, good sight, married a normal man and had five normal children; III. 3, male, dead, had cataract removed in childhood, sight greatly improved; married normal woman, twelve children, one cataractous. III. 4, five males; III. 5, four females, order not known, it is said "the girls were all right." IV. 1—3, offspring of cataractous father and normal mother; IV. 1, female, aet. 59, good sight, husband, IV. 1*a*, normal, one child, V. 1, normal; IV. 2, male, aet. 57, born with cataract, operations in youth, now fair vision one eye, constant nystagmus and squint, dental enamel good; married his first cousin, IV. 7, unaffected; issue nine, of whom five are known to be cataractous. IV. 3, male, aet. 55, both eyes operated on for cataract in youth; seen by Dr Rolston, of Plymouth, in 1907. He reports that right eye is nearly blind; left has clear pupil, vision with glass poor; married, had seventeen children of whom eleven died young, one operated on for cataract, three others "bad sight." IV. 4—8, sibship of five, due to normal parents; IV. 4, male, he and his progeny investigated by Dr Jameson Evans of Birmingham; IV. 4 is a widower, good sight, wife had good sight; of his seven children (V. 13) one is dead, two examined, all reported normal; of his eight grandchildren, VI. 7, all reported normal, two, a girl aet. 15 and a boy aet. 13, were examined and found normal. IV. 5, female, and her children are reported normal. IV. 7, female, married her first cousin who is cataractous and she died aet. 41 of phthisis, she had nine children, five cataractous. IV. 6, male, also died of phthisis, unmarried. IV. 8, male, normal, unmarried. IV. 9—12, sibship of eight due to cataractous father, all are reported normal except the youngest, a male, IV. 12, who had cataracts removed at Plymouth in youth; he has four normal children. V. 2—10, sibship of nine due to cataractous father and his first cousin who had good eyes. V. 2, male, aet. 30, delicate lamellar cataracts with small anterior polar opacities, 7 D. of myopia in each eye, works fairly well as "compositor," dental enamel good; wife, V. 2*a*, normal; four living children, eldest cataractous. V. 3, female, died aet. 5 months, "nothing noticed wrong with eyes." V. 4, male, aet. 24, postman, "passed eye tests," not seen. V. 5, female, aet. 22, born with cataracts, operated, vision poor, nystagmus, squint; beautiful teeth. V. 6, female, died aet. 3 months, eyes thought to be normal by parents. V. 7, female, died aet. 6 weeks, cataracts were seen by parents. V. 8, female, aet. 19, born with cataracts, operated, poor vision, teeth excellent. V. 9, female, aet. 18, same as 8. V. 10, female, aet. 15, lenses perfect, left eye myopic. V. 11—12, sibship of seventeen due to cataractous father, V. 11 was operated on for cataract at 4 years, married, no note as to children; one sister is blind of one eye (cause?), and two have "bad sight." Thus the group V. 12 in the pedigree figure should have been broken up into: three probably cataractous, two unknown, ten who died under 15 months, and one who died of bronchitis at 7 years; each of the last eleven being supposed to have good sight. VI. 1—6, sibship of four and two miscarriages due to cataractous father and normal mother. VI. 1, male, aet. 7, born with cataracts, right removed, vision very fair, left lamellar cataract of large size untouched, of permanent teeth one pair molars only cut, enamel good. VI. 2, male, aet. 5, perfect eyes. VI. 3 and 4 miscarriages. VI. 5, male, aet. 4, perfect eyes. VI. 6, girl, aet. 12 months, perfect eyes. (Unpublished.)

GROUP I *b*. *Discoid Cataract with and without lamellar and mixed forms.*

PLATE XXXII. Fig. 345ª. *Nettleship-Ogilvie-Johnson's Case.* Coppock family. Discoid cataract. In this family there was first discovered (by Doyne) a very definite and peculiar variety of stationary congenital cataract. The lens opacity is so small that it requires careful search. The patient is often unaware of the imperfection of the eyes, and shows no symptom save a liking to shade the eyes against a strong light. The opacity takes the form of a sharply defined circular disc placed deep in the lens between the nucleus and the posterior pole. In most cases the texture of the disc is uniform, in one or two a tri-radiate structure is evident. It is generally large enough to block the ordinary—say 4 mm.—pupil. It always affects both eyes and equally. It has been seen at as early an age as ten years and at as late an age as 82. Age changes in discoid cataract: Doyne at a recent meeting of the Ophthalmological Society of the U.K. stated that, so far as it was possible to judge by impression, he was inclined to think that in those individuals whose cases he had examined at the first, there showed now after twenty years a slightly increased density of the discoid opacity. There is some reason to think that the cataractous branch of the family has an unusual tendency to pre-senile lenticular change in the form of scattered dots and small smudgy striae. Such changes were noticed in at least twelve of the affected division; six of these have also the typical family cataract, whilst of the other six, who have only the scattered changes, three are the adult children of a father who himself has the typical single cataract. The disc cataract does not affect the visual acuity seriously, which seldom fell below $\frac{6}{12}$ unless there were coincident errors of refraction. Doyne saw and described the first case, IV. 6, in 1888, at the Oxford Eye Hospital; and later he saw V. 25, V. 9, and probably V. 11. The pedigree covers 288 persons, 131 male, 115 female, and 42 sex not known. The cataractous division contains 134 persons, 71 male, 57 female, and 6 sex unknown. Of these 134 persons, 33 are dead, of the remaining 101, ninety were examined by Ogilvie. Descent: the inheritance was continuous in every instance, once through four generations (IV. 3 to V. 9 to VI. 31 to VII. 8); twice through three generations (IV. 3 to V. 11 to VI. 38, etc.); twice through two generations (IV. 6 to V. 25 and IV. 10 to V. 26, etc.). Transmission was by the father four times, by the mother thrice. The four affected fathers had twenty-two children, of whom eight were affected; the three affected mothers had twelve children, of whom five were affected. Similarly the unaffected are found in continuous descent, the descendants of a normal being normal, as is shown in the descendants of IV. 4 and of V. 1. The Coppock family and their descendants form a considerable part of the population of Headington Quarry. They are bright and intelligent, somewhat over the average height and girth, and exceeding the average duration of life. Constitutional diseases are rare amongst them. Not a trace of syphilis has been observed. Rickets is unknown. Their teeth are exceptionally good. Ogilvie came to the conclusion from their appearance, high intellectual standard, and manner, that they were a different race from the local inhabitants. Their luxuriant dark hair, brilliant eyes, and white teeth suggested gipsy blood. At one time it was thought they were of Cornish stock, imported long ago, in order to quarry the Headington stone used in building the Oxford Colleges, but this hypothesis has not been proved.

The earliest known member of the stock—John Coppock (I. 1) born in 1774—came from the village of Milton in Oxfordshire, where he died in 1812; his wife was named Adkins, but nothing is known of her side. They had nine children, II. 1—9, seven males, two females. II. 2 and 3, females, and 4, 6, and 8, males, appear to have died without issue; II. 5, 7 and 9, married, having sibships of 5, 1 and 1 respectively, they had a succession of sibships through the IV., V. and VI. generations but no cataract appears in these branches of the tree. II. 1, married, one, II. 0 *a*, of two females, sole siblings; the other female, II. 0 *b*, married but had no progeny. II. 1 and 0 *a* had for offspring three males. III. (In this and succeeding generations the progeny of the unaffected branches will not be named, save where they intermarry with the affected, but their succession is shown in the tree.) III. 1 and 3, males, appear to have died without issue; III. 2 married a woman named Bateman, III. 2*a*. Since the original publication of the pedigree Ogilvie has examined various descendants of her family and all have been found normal. In the sibship of III. 2 and 2*a*, ten in number, there first appear known cases of discoid cataract. IV. (In this and subsequent generations examinations were made by Ogilvie, a midriatic was used, the fundus examined, and the vision tested whenever possible.) IV. 1, female, aet. 82, seen 1906, typical central, symmetrical cataracts of rather above average size and density, also some peripheral changes of senile type; four children, one affected. IV. 2, male, died 1894, aet. 68; saw badly in bright light, used to shade his eyes in order to see clearly, not examined; four children, eight grandchildren, one greatgrandchild, not affected (these will not be referred to in subsequent generations). IV. 3, male, died 1904, aet. 76, not examined, but living members of family believe he was affected like themselves; three children, two affected. IV. 4, male, aet. 76; not affected, lenses show a few fine peripheral striae; two children, seven grandchildren, not affected (these not referred to again). IV. 5, female, dead, no information; married twice, first had three children, six grandchildren, not affected (these not referred to again), secondly no progeny. IV. 6, male, aet. 72, the one in whom the typical cataract was first discovered by Doyne in 1888, aet. 54; he complained that he could not see well in a bright light, though he could at other times; there were commencing cortical striae; optical iridectomy done 1892 with advantage; dental enamel perfect; nine children, one affected. IV. 7, male, aet. 66; not affected, lenses

quite clear; no progeny. IV. 8, male, 9, female, died in infancy, no information. IV. 10, female, aet. 62, seen 1906, typical central symmetrical cataracts, also a rather large peripheral opacity in right, and several fine spokes in circumference of left; dental enamel perfect in remaining teeth; married her second cousin of unaffected branch, he died of epithelioma of tongue, nothing known of eyes; they had seven children, three affected. V. 1—4 sibship due to affected mother IV. 1. V. 1, female, aet. 60, not affected, lenses show a few fine peripheral striae; married her second cousin once removed of unaffected branch, nine children, six living not affected, five grandchildren not affected (these not referred to again). V. 2, male, aet. 56, seen 1906, typical central symmetrical cataracts of average size and density, and rather freely stippled, outline regular and perfectly circular; a few peripheral opacities; eleven children, one grandchild, not affected (these not referred to again). V. 3, female, dead, had one child, dead; both no information. V. 4, female, aet. 49, not affected; has two children, not affected (not referred to again). V. 5—8 offspring of affected father IV. 2. V. 5, female, died aet. 19, no information. V. 6, female, died aet. 23, no information; married her second cousin once removed of unaffected branch, had one child and one grandchild, not affected (not referred to again). V. 7, male, died aet. 27; had one child also dead, no information of either. V. 8, female, aet. 33, not affected, lenses quite clear, high myope (R. 13 and 1 D. As., L. 10 and 2 D. As.) and sees badly with glasses; large myopic crescents; the only member of the pedigree seen with considerable myopia; had six children unaffected (not referred to again). V. 9—11 offspring of affected father IV. 3. V. 9, male, aet. 55 in 1906, seen by Doyne in 1895 for an abrasion of cornea, no complaint of vision, on examination found to have "symmetrical circular and perfectly flat-looking opacities in both lenses, through which the fundus can be seen though somewhat blurred, the opacity is towards the posterior part of the lens, not at the nucleus" (Pl. M, fig. 17); vision quite good, dental enamel perfect; four children, three affected, four grandchildren, one affected. V. 10, female, no information; three children, one grandchild, not affected (not referred to again). V. 11, male, aet. 50, seen 1906; central symmetrical cataracts, finely stippled, smaller and less circular than type, circumference being slightly irregular, they are much less dense than usual, rest of lens clear, fundus normal; vision good; teeth "finest seen in man of his age and station"; aet. 14 lost every hair all over his body, bald since, cause unknown; six children, two affected. V. 17—25 sibship of affected father IV. 6. V. 17, male, aet. 41, not affected, no issue. V. 18, male, V. 19, female, died in infancy. V. 20, male, aet. 36, not affected, lenses have fine peripheral changes; is epileptic; six children, not affected (not referred to again). V. 21, female, aet. 34, not affected, lenses show few fine peripheral scattered pre-senile changes; six children, four living, not affected (not referred to again). V. 22, male, aet. 32, lenses perfect, no issue. V. 23, female, aet. 30, not affected but has pre-senile changes, no issue. V. 24, male, died in infancy. V. 25, female, aet. 25, seen first 1891, aet. ten, could not see well at lessons, "circular central posterior polar opacities," hypermetropic with some astigmatism; in 1903, ill-defined, smudgy opacities found in cortical layers of lenses independent of central opacities; teeth very good; is now a schoolmistress. V. 26—32 offspring of affected mother IV. 10 by her second cousin IV. 12 of unaffected branch. V. 26, female, seen 1906, aet. 40, "has always had perfect sight, but does not like a very bright light," central symmetrical cataracts smaller and less dense than usual, texture rather roughly stippled, outline circular but slightly irregular; fundus seen clearly quite normal; enamel of remaining teeth quite good; no children. V. 27, male, died aet. seven, no information. V. 28, male, aet. 38, typical cataracts, like 26; dental enamel good; two children not affected. V. 29, male, aet. 34, lenses perfect. V. 30, male, aet. 31, seen by Mackay in Edinburgh, vision excellent; in each eye are fine traces of persistent capsulo-pupillary membrane, but no lens opacities; dental enamel good; three children not affected. V. 31, male, aet. 28, typical central symmetrical cataracts, perfectly regular in outline and rather denser than usual so that fundus cannot be seen through opacity; cataracts resemble those of his mother IV. 10 and not those of his brother and sister (V. 26 and 28); dental enamel good; no issue. V. 32, male, died aet. 6, no information. VI., affected sibships only referred to. VI. 31 to 34 offspring of affected father V. 9. VI. 31, female, aet. 29, seen 1906, makes no complaint of sight though vision poor (R. $\frac{6}{36}$, L. $\frac{6}{24}$), hypermetropic astigmatism; central symmetrical cataracts so very faint that they might easily be overlooked; the finest details of fundus seen easily; opacity has no stippling, size and outline typical; there are a few peripheral smudges of opacity; teeth very good; one child only affected. VI. 32, male, aet. 24; symmetrical circular cataracts, somewhat granular, not very dense, i.e. intermediate in size and other characters between the large granular and large transparent forms; fundus seen fairly well through opacity; vision good; slight deficiency of enamel of incisors, enamel of molars and bicuspids good, right milk incisor remains in jaw behind permanent tooth; his firstborn, twins, VII. 9—10, examined by Ogilvie under midriatic fourteen days after birth, lenses found perfect. VI. 33, female, aet. 19; central symmetrical cataracts, full size, stippled but less dense than type, outline regular; fundus seen easily; vision, with glasses correcting mixed astigmatism, good. VI. 34, male, not affected. VI. 38—43 offspring of affected father V. 11. VI. 38, female, aet. 24; considers her sight perfect, vision good (R. $\frac{6}{9}$, L. $\frac{6}{12}$); typical central symmetrical cataracts perfectly regular in outline, denser than usual, freely stippled, and showing triradiate marking; fundus barely visible through opacity; remainder of lens clear (Pl. M, fig. 18); teeth very good. VI. 39, female, aet. 19, not affected. VI. 40, male, aet. 17, not affected. VI. 41, female, aet. 12, not affected. VI. 42, female, aet. ten; vision good; central symmetrical cataracts of the ordinary

size and perfectly regular outline, but extraordinarily faint and difficult to see; they are even fainter than VI. 31 and VII. 8; in left is a small pinpoint of opacity lying in front of inner circumference of cataract as in VII. 8; teeth good. VI. 43, female, aet. seven, not affected. VII. Nine sibships are shown. Six are of single siblings; one only of two births of which the first birth brought twins; one only with three; and one of "miscarriages" of unknown number. Of these nine sibships only one is issue of affected parent, and the sole child of that issue is affected. VII. 8, issue of affected mother VI. 13, female, aet. ten, vision poor (R. $\frac{6}{36}$, L. $\frac{6}{36}$), hypermetropic astigmatism; central, very faint, circular cataracts exactly like those in her mother; in left lens there is also a small pinpoint opacity close to inner circumference of the circular patch, but lying slightly in front of the latter, as is proved by the paralactic test. Consanguinity: "In regard to the influence of consanguinity in causing the peculiar cataract itself, a glance at the chart, which is a condensed combination of the pedigrees drawn up by Mr Ogilvie and Mr Johnson, the vicar, respectively, will show that the family cataract has been found hitherto in one branch of the stock; that although in three of the four consanguineous marriages one of the cousins was a member of that branch, we find that the cataract appeared in the offspring of only one of the cousin-marriages, and that in that case one of the parents had it; in the other cousin-marriages, where all the parents were free, their children were free also." (Bibl. No. 83.)

Fig. 342. *Nettleship's Case.* S—— family. Lamellar and discoid cataract with retinitis pigmentosa. The figure shows the connections of about 275 persons: 110 males, 116 females, and about 50 sex unrecorded. All are descended from the two brothers I. 5 and 6 and their respective wives; the brothers were "captains" of mines near St Austell in Cornwall. Nothing is known as to their sight, or of that of their wives. Examination of the chart shows that it is divisible into a large part to the right (about 180 persons), containing numerous cases of lamellar and discoid (Coppock) cataract, and a smaller part to the left (about 150, the overlapping members counted twice), containing cases of retinitis pigmentosa; whilst near the centre (III. 15—20) the two diseases overlap to a small extent. Of the lamellar and discoid cataract there are 32 cases; of retinitis pigmentosa only 15 have been found, but more may be extant for the family is difficult of access. Nettleship thinks there is no reason to suppose that the congenital cataract and the retinitis pigmentosa are causally related; their presence in the same genealogy is doubtless due to each disease having been introduced by different ancestors from independent sources. All the cases of cataract can be followed back to the brothers I. 5 and 6, and as the retinitis pigmentosa has been found only in the descendants of I. 5, we may reasonably suppose it was brought by his wife (marked I. 4), about whom nothing is known. Sex affection. The family cataract affected more males than females: 19 males, 13 females. It usually descended through the father: through affected fathers six times, normal father once; through affected mothers once or twice; through both parents once The descent was continuous except in two cases (V. 46 to 51, and IV. 100). On the other hand the retinitis pigmentosa affected far more males than females: 12 males, 3 females; the descent was always discontinuous and took place through the mother, who was herself normal. This phenomenon of discontinuous descent, though by no means the rule for retinitis pigmentosa, is not uncommon, whilst continuous descent is the rule in congenital cataract of all kinds. Sir Anderson Critchett published an account of IV. 83 in the *Trans. Oph. Soc. U. K.* in 1900 as illustrative of the good effects of optical iridectomy (removal of a small portion of the iris) in selected cases of small lamellar cataract; from this case Nettleship commenced his investigations. This pedigree shows well the collocation of ordinary lamellar cataract and the discoid cataract, not only in two sets of cousins, but in two brothers (III. 28 and 30), and in a brother and sister (IV. 60 and 66). This is of particular interest clinically and from the genetic point of view, because in the pedigree of the Coppock family (Plate XXXII., fig. 345), Nettleship and Ogilvie came to the conclusion that the changes in the eighteen affected persons of that genealogy could not be described as lamellar in actual structure, and probably not in developmental origin, although even in them several varieties of size, density and structure were observed. Nettleship concludes from the evidence of this pedigree that a graduated series will probably be found passing from the smallest and faintest disc of intransparency between the nucleus and posterior pole of the lens, to the common lamellar cataract, with its well-developed anterior and posterior layers and riders, and having a diameter considerably larger than the natural pupil (Plate M, figs. 20—26). In this pedigree of 32 cases of congenital cataract, 24 are marked "lamellar," 16 males, 8 females; and 8 as of the "discoid" type, 3 males and 5 females. Pre-senile cataract. In no less than 18 members of generations III., IV. and V., small striae or vacuoles of the lens were found in smaller or larger numbers, in three of these there were coincident lamellar cataracts, and one discoid cataract; the ages of these persons range from 43 to 12 years. Senile cataract. The presence of these cases in the chart has no special significance in relation to the congenital forms of cataract, but well-marked senile striae of the lens tend to obscure the diagnosis of pre-existing lamellar or discoid cataract; III. 36 is a case in point. The general intelligence of the members of the family was good, and in all cases where the condition of the teeth was ascertained the enamel is described as of perfect type.

I. 1, 2 and 3, no information. I. 2 had 13 siblings, I. 1, most of these lived to be old and had good sight so far as is known. I. 4, no information. I. 5, "Captain" Johnson S—— had good sight according

to report; he had six children of whom one, II. 6, is believed to have had cataracts. I. 6, "Captain" Tom S——, brother of I. 2, kept an inn; killed by kick from a horse; had 13 children of whom some ten are said to have had bad sight. I. 7, female, no information. I. 8, other siblings, number and sexes not known, but some were male. II. 2—11 sibship of six from I. 4 and 5, father presumed normal, mother unknown. II. 2, male, unknown, had 11 children of whom three are believed to have had retinitis pigmentosa. II. 4, male, migrated to Australia with his family, unknown. II. 6, male, is said by his son (III. 18) to have had cataracts for certain, but saw pretty well and was not night blind; he lived to be aet. 89; married his first cousin (12) whose condition is unknown, and had 13 children of whom one, III. 18, is known to have had retinitis pigmentosa and another, III. 20, believed to have had cataract. II. 7, male, had only two children, but died childless; no information concerning eyes of any. II. 9, male, married a Somerset woman aet. 17 (II. 1) when he was aet. 28, died aet. 70 about 1897, having founded tailoring business in London some forty years ago, now carried on by III. 28; had six children, III. 26—31, four cataractous. II. 11, male, unknown, married aet. 26 a Cornish wife aet. 22, and had nine children, III. 32—40, of whom eight had some variety of cataract. II. 12—17 sibship of about 13, father believed to be normal, mother unknown. II. 12, female, unknown, married her first cousin, *vide supra* II. 6. II. 13, male, migrated to Australia, had several children, condition unknown. II. 15, male, believed to have had good sight; had several children, believed to be normal. II. 17, about ten daughters; some of them are said by III. 44 to have been "very close-sighted and could not wear glasses," these defective ones had dark hair and dark eyes, all that had fair hair and blue eyes had good sight. Generation III. Of the 45 indicated 13 are certainly living; 12 of these have been seen (1907). III. 1—13 offspring of II. 2 and 3, parents unknown. III. 1, female, died aet. 75, good sight; husband unknown; had four children, three had senile cataract, one retinitis pigmentosa. III. 2, female, died at birth of first child; good sight. III. 3, female, died young, place in sibship unknown. III. 4, "Blind Tom," died aet. 60; driver of a stationary engine and had to give up aet. 50 from his defective sight; "he could never see when it was moonlight"; his cousin III. 18 said "he used to turn his head from side to side to see things"; no operation; doubtless retinitis pigmentosa. III. 5, female, married, no issue; good sight. III. 7, male, farm-labourer and preacher, died aet. 68; could see to do his farm work till about aet. 50, when he gave up because of defective sight; was called "moon-blind" and "could only see in certain lights"; doubtless retinitis pigmentosa; married twice; first wife was first cousin, by her had four children; by second wife had six children; only the first-born of the ten, IV. 22, traced, reported perfect sight. III. 9, female, dead, had ten children of whom one, IV. 30, had bad sight, the other nine and the mother said to have seen well. III. 10, 11, 12, females, unknown. III. 13, female, aet. about 70, has retinitis pigmentosa with some patches of choroidal atrophy, many senile opacities in lenses, more in right; married; had four children, one, IV. 3, has pre-senile cataract, others unknown; nine grandchildren, two, V. 38 and 41, have retinitis pigmentosa. III. 15—23, sibship of 13 due to cataractous father with his unknown first cousin. III. 15, female, dead, said by III. 18 and IV. 38 to have been night blind; had 13 children, eight grew up, four living and believed to have good sight; several grandchildren, some, V. 46, 49, 50, with cataract. III. 16, female, dead, had good sight; had eleven children, three living and normal, others emigrated. III. 17, female, one child, unknown. III. 18, male, aet. 71, examined by Nettleship 1907; slight striae of lower part of each lens but no polar or other axial cataract; fundus appearances quite normal, yet he said: "I cannot see at night and never have been able to all my life," that he was night blind was confirmed by wife and neighbour; no children. III. 19, male, unknown. III. 20, male, dead, operations for cataract at Plymouth and London, sight always bad, blind aet. 50; no mention of "night blindness"; married, one child only, IV. 44, lamellar cataract. III. 21, female, unknown, four children, unknown; two grandchildren, V. 52, said to have bad sight. III. 22, female, unknown. III. 23, five siblings, unknown. III. 26—31 sibship of six of normal father, unknown mother. III. 26, male, unknown; eight children, four certainly known to be normal. III. 27, female, unknown. III. 28, male, aet. 49, in each lens deeply seated behind nucleus but in front of posterior pole, a small axial and finely granular opacity of "Coppock" type, a second smaller opacity close to axial one on its temporal side and slightly up, in each lens; his wife (III. 37) who is his first cousin, also has a very small opacity in each lens apparently at the posterior pole, but less circular than the "Coppock" type, more radiating; they have had seven children of whom two have cataract. III. 29, female, aet. 47, typical small lamellar cataracts with clean well-defined edges and no spokes, but with three knobs; formerly had very good teeth; myopic; had 13 children, twice twins, five had cataract of various kinds. III. 30, male, aet. 44, both lenses needled by Brudenell Carter for lamellar cataract when aet. 11, did well; but now one lost from detached retina by injury, other eye good; six children, two, IV. 80 and 83, lamellar cataract. III. 31, female, died aet. 34, known to have cataract like 30. III. 32—40 sibship of nine, parents unknown. III. 32, male, died aet. 52, had operations for lamellar cataract; five children, two cataractous. III. 33, male, aet. 65, perfect lenses and sight; no note of marriage. III. 34, male, died aet. 52, believed to have had operations for lamellar cataract about aet. 12; four children, all cataractous, one with retinitis pigmentosa. III. 35, female, aet. 53, cataracts removed by Brudenell Carter, aet. 17, fair vision in one eye; one daughter, two grandchildren, normal. III. 36, female, aet. 50, early senile cataract, no appearance of lamellar cataract, but small "Coppock" could not be excluded; four children,

one cataractous. III. 37, female, married first cousin, see III. 28. III. 38, male, died aet. 50; some relations say he had the family cataract, others that he had perfect sight; no children. III. 39, female, aet. 43; well-marked, moderate-sized lamellar cataracts, right denser than left; also early senile lens changes; teeth good but some molars carious; no children. III. 40, male, aet. 41, small, faint lamellar cataracts alike in each lens, with early senile changes; dental enamel good. III. 42—45 offspring of presumed normal parents. III. 42, female, aet. over 62, reported good sight. III. 43, husband of III. 44, q.s. III. 44, female, aet. 62, eyes quite normal; husband, not consanguineous, aet. 68, senile cataract; they have had four children, two cataractous, and one grandchild, normal. III. 45, other siblings, unknown. IV. 2—5 offspring of normal mother, unknown father. IV. 2, female, aet. 61, good vision, peripheral striae in each lens (senile cataract); had three children, all died young. IV. 3, male, aet. 54, farm labourer, has never seen well especially at dusk, but no marked deterioration until ten years ago, since when unable to see to do farm work; counts fingers at one foot; posterior polar radiating opacities of lenses and advanced retinitis pigmentosa; unmarried. IV. 4, female, aet. 49—50, senile cataracts commencing; married, no children. IV. 5, female, aet. 50, "sight never so good as some people's, but never very bad"; vision, right $\frac{6}{24}$, convergent squint, left $\frac{6}{12}$; early senile changes in lenses; optic discs look atrophic; no retinitis pigmentosa. IV. 8—18 sibship of seven from father with retinitis pigmentosa. IV. 8, female, aet. 50, lenses normal; high myope; one daughter, normal. IV. 9, male, aet. 49; quite normal; six children, five normal, one miscarriage. IV. 11, male, aet. 42, normal; six children, four known normal, two died young. IV. 13, female, aet. 49, high myopic astigatism with macula choroiditis and vitreous opacities; lenses normal; all front teeth gone; four children, one retinitis pigmentosa, one pre-senile cataract, one myopic but otherwise normal, one dead. IV. 15, female, dead, had good sight. IV. 16, female, normal; three children, one with pre-senile cataract. IV. 18, female, seen by Rowan of Glasgow, pre-senile opacities and high myopia; eight children, two sons retinitis pigmentosa, five daughters and one son, normal. IV. 22—28, first family of four of father with retinitis pigmentosa by his presumably normal first cousin. IV. 22, female, reported perfect sight; four children, males, three retinitis pigmentosa. IV. 24, 26, 28, unknown. Second sibship of six due to some father unknown. IV. 29—30, sibship due to presumed normal parents. IV. 29, nine siblings presumed normal. IV. 30, female, dead, had bad sight, no known issue of any of the ten. IV. 31—33, family of four of mother with retinitis pigmentosa. IV. 31, female, aet. 46, high myope and fine pre-senile striae in lenses; seven children, two retinitis pigmentosa, two miscarriages; one grandson, normal. IV. 33, three siblings, unknown. IV. 34—40, sibship of 13 (once twins) due to mother with retinitis pigmentosa, eight grew up and four are living, all believed normal; several grandchildren, two lamellar cataract, one pre-senile cataract. IV. 41 and 42, sibship of eleven, three males living have good sight, others went abroad. IV. 44, sole offspring of father with lamellar cataract; male, aet. 25, typical small lamellar cataract; teeth excellent; unmarried. IV. 48—58, eight siblings of unknown father. IV. 48, female, aet. 37, normal; ten children, two pre-senile cataract. IV. 50, female, aet. 36, normal; four children, all normal. IV. 52, male, aet. 32, refused examination, sight good; four children, normal. IV. 54, female, died aet. $1\frac{1}{2}$. IV. 55, female, not seen, sight good. IV. 56, male, refused examination, sight good. IV. 57, male, died aet. two. IV. 58, female, aet. 26, not seen, believed normal; one child, normal, one miscarriage. IV. 60—66, sibship of seven due to father and his first cousin, both with discoid cataract. IV. 60, female, aet. 16, small, finely granular, post-nuclear opacity in each lens "just like the 'Coppock' cataract"; incisors very good, first molars carious (this girl was the first of the pedigree Nettleship examined). IV. 61, female, died aet. five, "meningitis" after head injury. IV. 62, male, died aet. $1\frac{8}{12}$, pneumonia. IV. 63, male, aet. 11; lenses perfect; teeth perfect. IV. 64, female, aet. $9\frac{1}{2}$; lenses perfect; teeth very good. IV. 65, male, died aet. $1\frac{1}{2}$, pneumonia. IV. 66, male, aet. five; well-marked lamellar cataracts, right two shells of opacity, left three shells, with riders and edge of outer shell not quite circular; no fits; milk teeth normal. IV. 67—79, sibship of 13 (twice twins), mother with lamellar cataract. IV. 67, male, aet. 28, sight perfect. IV. 68, male, aet. 25, sight perfect. IV. 69, miscarriage. IV. 70, female, aet. 22, cataracts of quite the "Coppock" type (Plate M, figs. 22—3); teeth very good; married three years, no issue, husband abroad. IV. 71 and 72, twin males aet. 21, elder operated for cataracts by Hartridge and did well; younger, sight perfect; 73 and 74, twin females; one died aet. nine weeks; other aet. 19, sight perfect; IV. 75, female, aet. 18, cataracts like 70. IV. 76, male, died aet. four, spasmodic croup; had no fits; sight good. IV. 77, male, aet. 16, left lens a single vacuole at or near posterior pole, right perfect. IV. 78, still-born male. IV. 79, female, aet. 13; right lens a single vacuole rather deep in lens, not at pole; left perfect; teeth perfect. IV. 70, 73, 75, 79, irides blue-grey; IV. 71, 77, and some of the other males brown or grey-brown. IV. 80—85, sibship of six, father with lamellar cataract. IV. 80, female, aet. 19, operations for lamellar cataract aet. 12 and later. IV. 81, female, died aet. three of diphtheria. IV. 82, female, aet. $16\frac{1}{2}$, eyes perfect; teeth good except first molars carious. IV. 83, male, aet. 15; had optical iridectomies by Critchett for "small lamellar cataracts, not more than 4 mm. diameter, rather dense and surrounded by faint second lamella of opacity" aet. eight; good results (*Trans. Oph. Soc. U. K.*, Vol. xx., 1900, p. 230). IV. 84, female, aet. $12\frac{1}{2}$, lenses perfect; teeth very good. IV. 85, female, aet. ten, same as 84. IV. 86—91, sibship of five, father with lamellar cataract. IV. 86, male, died aet. $4\frac{1}{2}$. IV. 87, male, died aet. four. IV. 88, miscarriage or still-birth.

IV. 89, male, aet. 37; left lens dense circular ordinary lamellar cataract with many peripheral striae, vision getting worse; right lens (?) discoid subtriangular opacity with three knobs, no cortical spokes; no note of relative sizes of cataracts; teeth good; has 11 children, two with lamellar cataract. IV. 91, female, aet. 27; a single well-defined vacuole in each lens between nucleus and periphery down and in; married her third cousin, had two children, normal. IV. 92—95, sibship of four, father with lamellar cataract. IV. 92, female, aet. 23, small, faint, nearly circular lamellar cataract in each, with three knobs; also scattered dots and minute striae, chiefly below; teeth very good; not married, IV. 93, male, in Navy, slight pre-senile lens changes. IV. 94, female, aet. 20, many vacuoles in each lens, especially lower half, and in left a few fluffy striae; teeth good except two lower first molars lost. IV. 95, male, aet. 18; right lens normal, left scattered minute dots at about the circle of undilated pupil in anterior cortex. IV. 96, sole offspring of mother with lamellar cataract; female, aet. 24, lenses perfect; married, three conceptions, two normal males, one miscarriage. IV. 98—101, sibship of four, mother with senile cataract. IV. 98, male, aet. 23, lenses perfect; teeth good. IV. 99, female, died aet. $3\frac{10}{12}$, diphtheria. IV. 100, male, aet. 18; rather small lamellar cataracts, right less dense and less defined than left, no operation. IV. 101, male, aet. 14, lenses and teeth perfect. IV. 102—110, sibship of nine, father with lamellar cataract with early senile cataract. IV. 102, female, aet. 17; in each lens faint, small, subtriangular opacity behind nucleus, quite well defined and with knobs, "Coppock" type, also one or two small striae near the periphery of the ring (Plate M, fig. 26); teeth good. IV. 103, male, died. aet. $2\frac{10}{12}$ of "fits." IV. 104, male, aet. 14, very small but typical discoid opacity in each with another rather elongated streak a little to temporal side (Plate M, fig. 21). IV. 105, male, aet. $11\frac{1}{2}$, "Coppock" cataract rather larger than usual, and many peripheral vacuoles; teeth good. IV. 106, female, aet. nine, normal, hypermetrope. IV. 107, male, aet. seven, normal. IV. 108, male, aet. six, small three-knobbed lamellar cataract in each. IV. 109, female, aet. $4\frac{1}{2}$, normal. IV. 110, female, aet. one, not seen. IV. 111—115, sibship of four, normal mother and father from another family who has senile cataract (they are third cousins of last sibship). IV. 111, male, aet. $33\frac{1}{2}$, many fine peripheral striae in each lens; has one child, not seen, sight good. IV. 113, male, aet. 32, not seen, very good sight; married his third cousin who has pre-senile cataract; have two normal children. IV. 114, female, died aet. eight, "croup"; eyes were good. IV. 115, male, aet. 20, left lens large thin typical lamellar cataract with riders, right operated; no fits, but upper and lower incisors deficient in enamel. Generation V., affected sibships only mentioned, others noted above. V. 15—18, sibship of four, normal parents, grandfather retinitis pigmentosa. V. 15, male, aet. 23, typical retinitis pigmentosa with pale, waxy optic discs, no marked night-blindness, fields full; teeth good. V. 16, female, aet. 19, fine peripheral dot opacities in lenses; teeth good. V. 17, female, aet. 15, normal save for myopic astigmatism. V. 18, female, died aet. 24 of phthisis, place in sibship not noted. V. 19—21, sibship of three, normal parents, grandfather with retinitis pigmentosa. V. 19, male, aet. 13, and V. 20, female, aet. 11, normal. V. 21, female, aet. nine, left lens normal, right large vacuole near posterior pole. V. 22—29, sibship of eight of pre-senile cataractous mother and retinitis pigmentosa grandfather; all eight living, seven seen; two of the three males well-marked retinitis pigmentosa; four daughters, normal, save one girl high myope; other daughter not seen, said to see perfectly. V. 30—33, sibship of four, due to normal parents; grandfather retinitis pigmentosa, his wife, his first cousin, normal. V. 30, male, aet. 21, myopia and astigmatism; marked retinitis pigmentosa. V. 31, male, normal. V. 32, male, aet. 18, well-marked retinitis pigmentosa. V. 33, male, aet. 18, same as 32. V. 37—44, sibship of nine, mother pre-senile cataract, grandmother retinitis pigmentosa. V. 37, male, aet. 24, some myopia, otherwise normal; has one son, VI. 1, normal. V. 38, male, aet. 22, myopia, well-marked retinitis pigmentosa. V. 39, female, aet. 19, normal. V. 40, female, aet. 17, not seen, has "splendid sight." V. 41, male, aet. 16, myopia, well-marked retinitis pigmentosa. V. 42, male, aet. nine, normal. V. 43, male, aet. seven, normal. V. 46—51, sibship of six, normal parents, grandmother retinitis pigmentosa. V. 46, female, aet. 15, typical small, dense, lamellar cataracts; dental enamel good. V. 47, male, aet. 14, and 48, female, aet. 12, both normal. V. 49, female, aet. 11, right lens normal, left a vacuole in anterior cortex. V. 50, male, aet. $7\frac{1}{2}$, lamellar cataracts, right dense filling pupil, left smaller, less dense, with many irregular projections from outer periphery; teeth normal. V. 51, female, aet. three, normal. V. 52, sibship of two, parents and grandparents unknown, great grandfather probably cataractous, he married his first cousin; the siblings cannot be seen, said to have bad sight. V. 53—62, sibship of ten of normal parents, grandparents unknown. V. 53, male, aet. 19, in Army, and V. 54, female, aet. 18, domestic, both said to see well. V. 55, miscarriage. V. 56, female, aet. 14, normal. V. 57, female, aet. 12, right lens or vacuole near posterior pole, left normal. V. 58, male, aet. ten, right lens a vacuole, left normal. V. 59, male, aet. nine, hypermetropic, normal. V. 60, miscarriage. V. 61, female, aet. five, normal. V. 62, female, died aet. $2\frac{1}{2}$, "accident." V. 73—83, sibship of ten and one miscarriage, father and grandfather, both lamellar cataract. V. 73, male, died aet. $4\frac{1}{2}$, "abscess of lung"; vision believed to be good. V. 74, male, died aet. six weeks, "wasting." V. 75, male, aet. 14, eyes and teeth normal. V. 76, female, aet. 13, typical small lamellar cataracts, left with three knobs, right less regular as to knobs; teeth normal; hypermetropic. V. 77, miscarriage. V. 78, male, died at birth. V. 79, female, aet. nine, normal. V. 80, female, died aet. $\frac{11}{12}$, "dysentery." (V. 81 account missing in MS.) V. 82 male, aet. five, well-marked lamellar cataracts of rather small size, right shows three knobs and is

more circular and denser than left, left has a long, straight, sharp, nearly clear refraction spoke projecting from each knob (Plate M, figs. 24—5). V. 83, female, aet. $1\frac{3}{12}$, normal. VI. 1, one child born to normal parents, pre-senile grandmother, retinitis pigmentosa great-grandmother, a male, aet. $\frac{10}{12}$, lenses normal. (Bibl. No. 85.)

Fig. 343. *Burton Chance's Case.* Discoid cataract. "A family in which five members are affected by a very definite and peculiar variety of congenital cataract." Part live in Germany and part in America, so the full connection could not be worked out. I. 2, a female, married twice, son by second marriage know of no disease of the eyes in parents, though it is remembered they wore glasses. II. 1, female, child of first marriage of I. 2, "had bad sight, and one of her five children, a girl, had eye troubles." II. 2, 3 and 5, males, offspring of I. 2 by second husband; II. 2 and 5 had good sight, but nothing positive known of their history. II. 3, male, a tall, muscular, railway engineer, moderately myopic and wears glasses for reading, found to have opacities in his lenses similar to those found in his sons'. II. 4, his wife, normal. III. 6, 7 males, no data. III. 8, male, aet. 20, "has good sight, but deep in his right lens was a dense, opaque clump in the nasal half of a faint circular disc, while in the left there was a disk of such extraordinary thinness as to be scarcely detected—it might have been likened to a simple condensation of the crystalline substance." III. 9, female, aet. 16, "also had discs in her lenses; that in the right, studded with tiny dots, and in the centre was a dense opacity; in the left were several clumps with fine dots about them"; myopia 4 D. III. 10, male, act. 13, myopic, "discs in his lenses, dot-like in consistence, but irregularly arranged; in each were six distinct circular vacuoles." III. 11, male, aet. 11, noticed, aet. six, not to see as well as other children; health good; not so tall or robust mentally as boys of his age; seems dazed in bright light, peers at objects; vision poor, improved by high minus cylinders; in each lens a circular disc, sharply defined, minutely granular, in right clumps of opacities like mitotic figures, in left star-shaped; fundus could not be seen through the discs. "In size all these opacities were large enough to block the usual pupillary spaces...and measured about 4 mm. in diameter. In each person they were double and without exception accurately symmetrical in the two eyes. They appeared to be stationary. Their exact position was hard to define. They were not polar, or nuclear, but lay between the nucleus and posterior pole." "Teeth II. 3, III. 9 and 10 badly decayed, though in none of the family were there signs of rachitis." The author gives drawings of the opacities of each eye of the five known cases. (Bibl. No. 84.)

Fig. 344[a]. *Harman's Case.* Familial Discoid Cataract. I. 1, male, healthy, well-developed man, aet. 42, lenses perfect, no trace of opacity, vision, refraction and fundus excellent. I. 2, female, his wife, aet. 35, healthy, lenses perfect, vision, refraction and fundus healthy; these people come from different home counties, they are not related in the remotest degree, no connection with the Coppock family can be found; they show no trace of constitutional disease; father dark hair and eyes, mother reddish hair, blue eyes. II. 1, female, aet. nine; each lens presents the characteristic discoid cataract in its posterior layers; the opacity is perfectly circular, measures 3 mm. in diameter, it is of singular delicacy, and uniform in density; the density is so slight that the details of the fundus can be seen perfectly through it, and unless the lens be examined by light projected into the eye by a plane mirror it is scarcely to be seen, then it appears as a fine disc against the red ground of the fundus reflex; child is intelligent, has excellent vision, 2 D. hypermetropia, excellent teeth. II. 2, died aet. four, no knowledge of his state. II. 3, female, aet. seven, the child was brought to hospital on account of occasional divergent squint of the left eye, she has considerable mixed astigmatism, with glasses vision is good and the squint does not occur (in examining her refraction the discoid cataract was discovered and led to examination of her family); each lens has the characteristic discoid opacity, about 2·5 mm. diameter, perfectly circular and very thin, no other marks in lenses; fundus normal; first permanent molars good. II. 4, female, aet. two, lenses quite free from opacities. All of this family were examined with fully dilated pupils. (Bibl. No. 91 with coloured plate of cataract.)

Fig. 344[b]: see p. 165; Fig. 345[a]: see p. 145; Figs. 345[b] and 346: see p. 160.

Fig. 347. *Priestley Smith's Case.* Forman family. Discoid cataract. The author's description is as follows: This pedigree, the leading name in which is Forman, bears much resemblance to the Coppock pedigree published by Nettleship and Ogilvie in 1906. A connecting link between the Coppocks and Formans has been searched for but has not been found. Formans, for the last six generations, have been born, lived, reared families, and died in the village of Stapleton near Hinckley, in Leicestershire. By means of the Parish Registers I have been able to trace back the male line as shown in the chart, but it has been impossible to learn the surnames of the women whom the earlier Formans married, for the record of a marriage where husband and wife belonged before marriage to different parishes remains in the parish to which the wife has belonged and where the wedding takes place. The eye disorder may have been introduced into the Forman family through one of these earlier marriages. This family belongs almost exclusively to the labouring class, most of the men working on farms, some being coalminers. They live, with few exceptions, in the district lying between Nuneaton, Leicester and Derby. They are, taken as a whole, a healthy, well-developed and good-looking family. Many of them

have dark hair, but this is by no means universal, some, both of the affected and unaffected persons, being of fair type. Those who were examined were examined in every case with the ophthalmoscope, but it was only possible to make a minute examination after dilating the pupil in two cases; the others were examined at their own homes with undilated pupil. The opacity is discoid in form and apparently post-nuclear in position. Its size varies in different individuals, producing various degrees of visual impairment. So far as my observation goes, it is well-marked, and more or less dense in all cases. I did not see any instance of the very slight opacity described by Ogilvie, or of lamellar opacity. The facts of the hereditary transmission are as follows: A certain John Forman (IV. 3) a well-known carrier, was troubled throughout life, according to the testimony of several descendants and others who knew him well, by a peculiar weakness of eyesight which, though it did not incapacitate him, obliged him to shade his eyes with his hand when he wanted to see his best. Of his two brothers (IV. 1 and 2) the elder had good sight, and has left many descendants among whom no defect of the kind has been known; the younger died without issue and the evidence obtained with regard to his eyesight is conflicting. The children of John Forman (IV. 3) were fourteen in number, and several of them, like their father, have had very large families. The number of his descendants, dead and living, is at the present time at least 113, probably a few more. Information has been obtained concerning the eyesight of 103, and of these 27 were or are affected with discoid cataract. Of the 27 affected persons 24 were examined, and of the 76 unaffected 49 were examined. If we exclude all the unaffected sibships and exclude from the affected sibships those concerning whom there is no information, we have 51 descendants of John Forman remaining, and of these 51 persons, 27 are affected, i.e. 53 per cent. Of the affected 15 are male, 12 female. Every affected person, with one exception, who has had offspring, has transmitted the disorder; the exception being a man who has at present only one child. To a private enquiry the author replied: "There is no evidence of consanguinity. Many of them were noticed to have good teeth." (Bibl. No. 92.)

Group II. *Coralliform Cataract.*

Plate XXIX. Fig. 316. *Nettleship's Case.* Dobson family. Character of cataracts suggested to be akin to coralliform. I. 1, female, born with cataracts; one of her daughters was similarly affected. II. 1. The latter had several children, III. 1—6, of whom five were cataractous, viz., III. 1, first-born, female, operations unsuccessful; III. 2, female, seen aet. 18, with white anterior polar and nuclear congenital cataract; and three others whose sex and exact places in the sibship not specified; no further particulars. (Bibl. No. 82.)

Plate XXX. Fig. 317. *Nettleship's Case.* P—— family. Coralliform cataract (part worked out by Marcus Gunn, Leslie Paton, and others). The pedigree shows 22 cases of cataract, 20 of these patients are living, 18 were examined. Nearly all of the non-affected of the cataractous branches were seen. (For description of coralliform cataract, see above, page 127.) In every case of cataract the condition is alike in both eyes. No other inferiority or degeneration has occurred in the stock. From examination of subjects of ages from 72 years to 13 years, it appeared that the total amount of opacity very slowly increased with age; this increase of the opacity with age, and the fact that no case of this cataract was seen at an earlier age than eight years, leads Nettleship to think that it is doubtful whether this form of cataract is present at birth. The teeth showed no abnormality of enamel. Early deaths, still-births, and miscarriages were rare. Only one consanguineous marriage (between half-first cousins) has taken place (III. 5 and 7). At the right-hand end of the pedigree are shown three cases of total blindness, probably due to glaucoma or some form of irido-cyclitis; only one of this stock, and he himself with healthy eyes (III. 14), married into the cataractous family, and no bad effects ensued.

I. 1—3, no data concerning eyes. II., 4 and 5, brother and sister of sibship of nine or ten of same mother by two different fathers, it is not known how many belonged to each father; II. 4 and 5 are stated by III. 6, 7 and 9 to have had sight defective as their own. II. 5 lived to aet. 86. II. 6, his wife, died of consumption, aet. 35, after bearing him five children. III. 6, female, aet. 72, has radio-axial opacities and nuclear haze, complicated by corneal nebulae, sight very bad. III. 7, male, aet. 66, characteristic opacities and nuclear haze, could read until aet. 40, sight now very bad; his wife, III. 5, who was his half-first cousin, is dead. III. 9, female, aet. 62, typical opacities. III. 10, female, dead, had nine children (IV. 21—24), all free from eye defect, six died of consumption about aet. 20. III. 21, aet. 53, has bad vision from old choroido-retinitis, which remains as when seen by Streatfeild 24 years ago, condition started aet. 29, three years after marriage (? tubercular or constitutional), she has no children. IV. 1—4, females, offspring of III. 5 and 7, all grown up, aged from about 35 to 25; eldest and youngest affected. IV. 1 has very good teeth; no still-births or miscarriages. IV. 5—12, females, except 11, offspring of III. 6, aet. 48 to 30, no still-births; the four affected females all have typical opacities; all four married, one has had no pregnancies. IV. 13—20, offspring of III. 9, no still-births. IV. 13, male, coarse striae in lenses. IV. 14, female, normal. IV. 15, male, normal. IV. 16, female, now

aet. 33, seen aet. 13 by the late J. E. Adams at Moorfields, "central opacities at posterior surface of each lens, considerable degree of mixed astigmatism, vision good with glasses"; she married aet. 17, has five children, one affected. IV. 17, male, refused examination, relatives agree sight as bad or worse than other affected ones. IV. 18, male, normal. IV. 19, female, same as 17. IV. 20, male, aet. 22, slight myopia, always "short-sighted," now a little worse than when at school, typical lens opacities and good teeth. V. 1—6, offspring of IV. 5. V. 1 and 2, females, both affected. V. 3 and 4, males, normal. V. 5, male, aet. 16, has a single elongated dot of opacity a little upwards and inwards of posterior pole in right lens, left lens perfect. V. 6, male, aet. ten, small axial opacities, teeth good. V. 7—9, offspring of IV. 8. V. 7, female, aet. eight, typical opacities. V. 8, still-born. V. 9, died aet. eight months. V. 12 and 13, offspring of IV. 13; V. 12, female, aet. 13, normal. V. 13, male, aet. 9, opacities like his father, but less dense. V. 14—18, normal siblings from normal parents. V. 19—23, offspring of IV. 16. V. 19, 22, 23, normal females. V. 20, male, died aet. 11 months. V. 21, male, aet. ten, typical opacities. (Bibl. No. 88.)

Fig. 318. *Nettleship's Case.* Betts family. Coralliform and lamellar cataracts. About 100 persons figure in the genealogy, of whom 30 are known to have or have had cataract, and 19 of these were seen by either Nettleship, Marcus Gunn, Walter Sinclair of Ipswich, or other surgeons, whose hospital records were obtained. Judging by the history, the cataract must have been present at birth in all cases; it is usually almost stationary, but in two cases some shrinking of the lenses occurred after middle age. With two exceptions, the cataract has presented the same features in every case that has been seen; they are depicted in Holmes Spicer's drawing of the case (IV. 2 in this pedigree), published by Gunn (*Trans. Oph. Soc. U.K.*, London, vol. xv. 1895), for which the name "Coralliform" was originally suggested. The description of the cataract appears above, p. 127: see also Plate M, fig. 27. In two of the cases of V. 2 and 5, of different sibships, Sinclair found ordinary lamellar cataracts, not the coralliform variety. There has been nothing special in the health or development of this family and no reason to suspect any constitutional disease; no history of convulsions; no deafness. In cases where the teeth were noticed, the condition of the enamel is described as good. No marriage of cousins found within the cataractous branch of the family, though it occurs in other branches not affected. Transmission continuous from affected to affected; of the affected, nineteen are males, eleven females; the cataract has no relation to the fecundity of the earlier generations. I. 1 had bad sight, his children told their children this was due to cataract; his wife, I. 2, is known to have had good sight. II. 1, male, cataract, died unmarried. II. 2, male, cataract, had five children, three eldest cataractous. II. 3, 4, males, cataracts, married, no issue. II. 5, female, cataracts, unmarried. II. 6, female, no cataracts, three normal children. II. 7, female, no cataracts, said to have had 21 children, all with good eyes. II. 8, female (second wife to 8 *a*, who by his first wife had eight normal children), was born with bad sight, died aet. 50, bore five children, of whom the two eldest, III. 10—11, females, have cataracts, the three youngest died in infancy. III. 1—5, issue of II. 2. III. 1, male, cataracts removed, had two sons, one, IV. 2, coralliform cataract (described and illustrated). III. 2, cataracts removed, had eight or nine children, four males cataractous. III. 3, female, cataracts removed aet. 51, described as densely white and appeared to be shrunken, one child born prematurely and died. III. 4 and 5, females, normal. III. 6 and 7, males, normal. III. 8, female, normal. III. 9, twenty-one siblings, issue of II. 7, normal. III. 10—12, issue of II. 8. III. 10, female, aet. 71 in 1905, cataracts, right operated aet. 27, vision still fair, left not touched, cataract is complete and shrunken, has no children. III. 11, female, aet. 64 in 1905, cataracts, right removed, left typical coralliform, has had ten children and one miscarriage, of whom seven have cataract. IV., 1 and 2, issue of III. 1. IV. 1, male, normal, has three normal children. IV. 2, male, coralliform cataracts, had three children, eldest *lamellar* cataract, second "white cataract like father." IV. 3—9, issue of III. 2. IV. 3, two or three died in infancy, state of eyes unknown. IV. 4, male, cataracts. IV. 5, male, cataracts not removed; married twice, by first wife one boy, V. 5, with *lamellar* cataracts, by second, four normal children. IV. 6, male, cataracts removed aet. 17 by Morton, opacities described as "congenital," "opaque pearl-like bodies" towards centre with clear periphery; in 1905 has three children with good eyes. IV. 7, female, normal. IV. 8, male, normal. IV. 9, male, cataracts. IV. 9 *a*, issue of III. 3, seven months child, died early. IV. 10, 11, issue of III. 4, normal. IV. 12, issue of III. 5, normal. IV. 13, 14, issue of III. 8, normal. IV. 15, issue of III. 9, number unknown, reported normal. IV. 16 to 26, issue of III. 11. IV. 16, female, normal, has one normal child. IV. 17, female, died aet. $3\frac{1}{2}$, good eyes. IV. 18, male, died aet. $1\frac{1}{2}$, cataract. IV. 19, female, no cataract, at aet. 20, had large choroidal exudation in right eye, had previously abscess over right jaw leaving depressed scar, lost all her teeth aet. 18; no evidence of hereditary syphilis (?tubercle); seen again aet. 40, nothing fresh, has had three normal-eyed children, V. 15—17. IV. 20, miscarriage. IV. 21, male, cataracts operated on aet. five or six, seen aet. 18 and 40, fair vision; has four children, eldest and possibly youngest cataractous. IV. 22, male, cataract, married, no children. IV. 23, male, died, aet. four, cataract. IV. 24, female, cataract, teeth good except that lateral incisors absent aet. 14; has two children, elder cataractous. IV. 25, female, cataract, no operation, seen aet. 20 and 32, on each occasion appearances of coralliform cataract typical, can see large print; teeth good enamel; unmarried. IV. 26, male, cataract, married, no children. V. 1, three normal offspring of IV. 1. V. 2—4, offspring of IV. 2.

V. 2, female, *lamellar* cataracts, no note of teeth, died aet. seven. V. 3, male, died early, had "white cataracts like father." V. 4, female, normal. V. 5, male, first child of IV. 5, *lamellar* cataracts operated aet. ten. V. 6—9, second family of IV. 5, all normal. V. 10—12, family of IV. 6, all normal. V. 13, issue of IV. 11, numbers unknown, reported normal. V. 14, issue of IV. 16, female, normal. V. 15—17, issue of IV. 19, normal. V. 18—21, issue of IV. 21. V. 18, male, cataract of same form as his father and grandfather, right operated aet. five, eye lost by intraocular haemorrhage ten days after operation, thought to be caused by a blow; left, not operated, useful vision aet. 15. V. 19, 20, females, normal. V. 21, female, believed to have cataract. V. 22, 23, issue of IV. 24. V. 22, female, reported to have cataract. V. 23, male, normal. (Bibl. No. 82.)

Fig. 319. *Nettleship's Case.* Tomes family. Coralliform cataract. Generation I., four members noted, of whom I. 3, female, was personally known to II. 4 (cataractous), she is reported to have died aet. 60 "with every tooth in her head perfect," is well known to have had defective "short" sight all her life, and II. 4, from his description of it, evidently thinks it was like his own; she had an only child, II. 2, now living, aet. 60 or more, known to have perfect sight. II. 4, male, seen by Treacher Collins in 1906, typical coralliform cataract, one of his daughters seen at the same time with identical condition. III. 1—8, issue of II. 4. III. 1, 3 and 5 not seen, but reported to have perfectly good sight, quite different from their affected sisters. III. 6, still-born. III. 2, female, aet. 30, typical opacities of lenses, more abundant in right than left, has one child, IV. 1, normal. III. 4, female, aet. 25, similar cataracts. III. 7, female, aet. 13, seen by Treacher Collins in 1906, defect of sight noticed by school teachers, not by her own family, vision $= \frac{6}{18}$, characteristic trumpet-shaped opacities radiating from axial part of lens, seen two years later unchanged; no history of fits or illness, good teeth. Generation IV. Three males. IV. 1, aet. three months; IV. 2, aet. $4\frac{1}{2}$ years; IV. 3, aet. $1\frac{1}{2}$; the children of two affected sisters, examined by Nettleship and found normal. There appeared to be no special constitutional diseases, defects or degeneracies in the genealogy, except the cataract, and all seen were intelligent, well-developed people. All the children and grandchildren of II. 4 have either blue or grey irides, and a brown one was not heard of in the family. There has been no consanguineous marriage. (Bibl. No. 88.)

Fig. 320. *J. H. Fisher's Case.* G—— family. Coralliform cataract. Generation I., three known to have good sight, the fourth unknown. II. 1, father Scotch, mother North of England, no consanguinity; mother, II. 2, had cataract, for which she had operation at age of 48, whether condition congenital or acquired unknown. III. 1—7, seven children, the last two only affected. III. 7, female, aet. 48, had typical coralliform cataract in left eye, the right had been operated on aet. 23, failed owing to "cholesterin filling the gap," the eye was ultimately excised. III. 6, male, had "congenital" cataract; one eye operated on was unsuccessful. III. 1—5, males and females, were normal; they are all married, and have numerous normal-eyed children; no consanguinity. (Bibl. No. 80.)

Fig. 321. *Harman's Case.* Donaldson family. Axial or coralliform cataract. The relations of generations I. and II. are only known by the report of III. 7; he has a remarkable knowledge of his kin. I. 1 and 2, no information, save "not cousins." I. 3 and 4 "died in a cholera epidemic, I. 3 was a fine workman." II. 1 was a deaf and dumb compositor, eyes believed to be normal. II. 3, female, said to have had good eyes. II. 4, male, died aet. 76, had "perfect sight." II. 5, female, wife of II. 4, died aet. 64, was deaf from aet. five "due to scarlet fever," had perfect sight. II. 6 was a naval man, evidence of good vision. II. 7, female, dead, said to have had good sight. III. 1—7, six siblings, parents II. 4 and 5, normal-eyed. III. 1, 4 and 5, males, all died in early infancy, no knowledge of eyes. III. 2, female, married, no children, good eyes. III. 6, female, unmarried, good eyes. III. 7, male, had both lenses removed for "congenital cataract" as a boy, his sight is poor, there is constant lateral nystagmus; dental enamel good. III. 8, wife of III. 7, normal. IV. 1—3, offspring of III. 7 and 8, father cataractous, mother normal. IV. 1, female, a frail child of seven years, vision bad, constant very rapid lateral nystagmus; right eye large axial cataract, scar in capsule of lens and posterior synechiae of iris confirms statement of father that operation had been attempted two years ago; left eye untouched, axial or coralliform cataract of medium size, and of very white colour; vision for general purposes was improved by removing the right lens, and iridectomy in left eye; fundus of eyes appears good; enamel of primary dentition good; no sign of hereditary syphilis. IV. 2, male, aet. five, eyes and primary dentition perfect. IV. 3, female, aet. six months, eyes perfect; no pregnancies between 2 and 3; no consanguinity. (Bibl. No. 93.)

Fig. 322. *Knies's Case.* Mixed axial and lamellar cataract (?akin to coralliform). Knies gives drawings of the conditions of the lenses of several of the cases. The form of cataract is similar to that earlier described by Müller (Pl. M, fig. 28). I. 1, male, lenses shrunken and tremulous, had been quite blind (no perception of light) for twenty-one years. II. 1, normal. II. 2, daughter of I. 1, aet. 36, incomplete lamellar cataract denser in left than in right eye. III., six children of II. 2. III. 1, male, aet. $14\frac{1}{2}$, cataract involving nucleus, both poles, and the axis of the lens, besides presenting some radiating masses. III. 2, female, aet. 13, minute left corneal nebula, no opacity in lenses. III. 3, female, right

eye damaged by leucoma adherens, left some lens defect. III. 4, female, aet. nine, minute left corneal nebula; no opacity seen in lenses, but since there was irregular astigmatism of the eyes not attributable to the cornea, author assumes it to have its seat in the lens. III. 5, male, aet. seven, right eye incomplete lamellar cataract, left minor degree of axial cataract. III. 6, male, aet. three, axial cataract very like III. 1. No consanguinity in parents; children did not suffer from fits; teeth good, except in case of III. 3, where honeycombed; intellects bright; no other defects in development. (Bibl. No. 48.)

Fig. 323. *Nettleship's Case.* Dobson family. ? Coralliform cataract. I. 1, born with cataracts; one of her daughters, II. 1, similarly affected. The latter had several children, III. 1—6, of whom five were cataractous, viz.: III. 1, girl, operated on unsuccessfully; III. 2, girl, seen aet. 18, with white anterior polar and nuclear congenital cataract, and three others; sex and exact places in sibship not specified. No further particulars. (Bibl. No. 82, Case 58 B.)

Fig. 324. *Appenzeller's Case.* A woman, III. 2, married a man, III. 1, not related by blood and had children, number not given, all of whom had good sight; her second husband was her first cousin, III. 3, who also had good eyes, and by him had three children, all of whom were born with double congenital cataract. IV. 1—3, normal children of III. 2's first marriage. IV. 4—6, cataractous children of III. 2's second marriage with her first cousin. IV. 4, sex not stated, was aet. four at date of publication, no particulars of the cataract. IV. 5, female, seen aet. three, had nuclear cataracts with projecting spicules and clear cortex; it is described as peculiar, and was presumably not lamellar and rather suggestive of "coralliform." IV. 6, sex not stated, born two years after 5, had double cataracts and died at five weeks. No known malformations in the ancestors of either of the parents III. 2 and 3. (Bibl. No. 58.)

Fig. 325. *E. Müller's Case.* Mixed lamellar and axial cataract (?akin to coralliform). Müller records in great detail the particulars of a peculiar mixed form of cataract; his diagrammatic figure is reproduced in Plate M, fig. 28. I. 1, female, appears to have been affected like her daughters, to judge by the history. II. 1—5, five females. II. 1 and 2 had good eyes. II. 3, aet. 30 at date of record. II. 4, aet. 29, and II. 5, aet. 24, each had the particular form of cataract described. (Compare Knies' case, Fig. 322, for similar form of cataract.) (Bibl. No. 43.)

Fig. 326. *Nettleship's Case.* C—— family. ? Coralliform cataract. II. 1, seen aet. 13; L. lens shows small speck just above posterior pole, squint; history of fits; teeth, defective enamel. II. 2, seen aet. 26; R. eye normal; L., dense opacity of axial part of lens passing from anterior pole back deeply into lens; there are blunt or knot-like projections from the axis; cataract believed to have been present at birth; enamel of teeth somewhat deficient at cutting edge. (Bibl. No. 82, Case 59.)

For Fig. 327, see p. 158.

GROUP III. *Stellate Cataract.*

PLATE XXIX. Fig. 308. *P. H. Adams's Case.* Stellate cataract. The opacities in these cases take the form of lines marking out the radii of the union of the ends of the lens fibres, for the most part the lines are in the posterior part of the lens, but in some cases there are Y- or X-shaped opacities of the anterior part. The opacities are situated closely beneath the capsule. The opacities become slightly denser with age, although II. 1 had very fair vision until past 80 years. I. 1; II. 2 and 4; and III. 2 are believed to have had the opacities on account of the likeness of their history to those in whom the opacities were seen. II. 1, male, was examined aet. 83, "the changes had progressed chiefly in the anterior part of the lens, the opacity taking a rather tri-lobed shape, as though it had spread from an original Y-shaped mark. One could see numerous fine spokes at the periphery of the lens, which was comparatively clear; these were situated on the posterior surface." III. 1 and 2 not seen; III. 2, female, always had defective sight; IV. 3 and 4, issue of affected mothers, believed to have good sight. IV. 1, male, "seen by Doyne, 1892, when he showed Y-shaped markings on the anterior surface of both lenses, and three primary rays, each branching into two on the posterior surface." Seen by author 1909, when he had "fairly dense opacities in front of both lenses, chiefly small circular opacities, and also numerous rays on the posterior surface." V. 1, female, aet. ten, "posterior lines very sharply marked, seven rays in the right eye and six primary ones in the left eye, with some secondary branches"; high myopia with astigmatism, vision poor. V. 2, female, normal. V. 3, male, aet. five, in addition to the posterior markings had very faint Y-shaped marks on the anterior surface; hypermetropic astigmatism. V. 4, female, aet. three and a half, "showed four primary rays in the right eye, branching at the end of two of them: and three in the left eye, almost immediately dividing into two." V. 5, female, aet. seven months, "showed an inverted Y on the posterior surface just branching into two at the end of the limbs with nothing visible on the front of the lens." "In none of them were the lenses perfectly transparent; they seemed to have a faintly dotted appearance and the fundus looked blurred, but normal." "The lines themselves were as if they had been drawn with a fine BBB pencil on rather rough paper, but were very sharply defined in

the children. In IV. 1, who is now aet. 36, the posterior lines appeared slightly broader and not so sharply marked." "The condition appears to be very slowly progressive, the opacity increasing most in the anterior part of the lens." On enquiry the author kindly supplied further data concerning the descendants of the unaffected branches shown in the right wing of the pedigree; he also stated that the teeth of V. 1—5 were perfect; and that there was no consanguinity. The author gives drawings of the anterior and posterior opacities seen in each eye of IV. 1, V. 1—5. Plate M, fig. 29, is a drawing made from the figures supplied of the right eye of V. 3. (Bibl. No. 89.)

Fig. 309: see p. 158.

Fig. 310. *Zirm and Bergmeister's Case.* Löwy family. Stellate cortical cataract. Nettleship obtained information direct from Bergmeister that the family was Jewish, but no information was to be had regarding consanguinity. Zirm describes the case of a boy (V. 8) in detail, and gives two good figures. The boy's sight was defective as long as he could remember. By focal light the anterior cortex of each lens showed five long radial striae which became enlarged (club shaped) at peripheral end; at posterior cortex a second stellate figure of three primary spokes dividing into six, all broader, shorter and somewhat denser than the anterior spokes; between the anterior striae, especially the lower ones, were delicate punctiform and linear spots. By transmitted light many finer striae came into view. All of the opacities stopped short of the equator, leaving that part quite clear. I. 1, female, became blind, cause unknown. II. 1, senile cataract; his wife had good eyes. III. 1, male, both eyes operated for senile cataract. III. 2, male, when aet. 71 found to have stellate cataract. III. 3, male, 4 and 5, females, all see well. IV. 1 to 7 issue of III. 1 (father, cataractous); IV. 1, male, aet. 57 to 58, mixed stellate and senile cataract both eyes, almost complete in left, and closely resembling the stellate cataract of V. 8 (see above) in right; a strong man with normal bones. IV. 2, male, aet. 43 to 45, sight defective from early life, now stellate cataract with numerous punctate opacities; myopic; bones and teeth normal. IV. 3 and 4, males, both dead, no details (Nettleship thinks the ages suggest that these two siblings really came between 1 and 2). IV. 5, male, aet. 42, lenses clear. IV. 6 and 7, females, good eyes. IV. 8, male, stellate cataract. IV. 9 and 10, males, one had good eyes, the other said to be "short-sighted." V. 1 to 5, issue of IV. 1. V. 1, male, aet. 34 to 35, stellate cataract: 4 D. myopia, bones and teeth normal. V. 2, female, stellate cataract. V. 3, male, aet. 23, same state. V. 4, male, aet. 21, normal eyes. V. 5, male, aet. 16, stellate cataract. V. 6—10, issue of IV. 2; V. 6, female, good sight, not "short-sighted"; V. 7, male, aet. 19 to 20, stellate cataract, 4 D. myopia; bones and teeth normal; V. 8, male, aet. 14, stellate cataract, details given above, 4 D. myopia; V. 9, female, aet. 11 to 12, lenses clear, myopic; V. 10, female, sees well, not "short-sighted"; V. 11 to 16, issue of IV. 5, 6 and 7; of these, only V. 11 was examined, he had corneal nebulae, no cataract, 4 D. myopia; V. 12 and 15, said to be "short-sighted," all the others reported to have good sight. VI. 1 to 3 (seen by Bergmeister only), issue of V. 1; VI. 1, female, aet. eight, stellate cataract; VI. 2, male, aet. five, normal; VI. 3, female, aet. four, stellate cataract; VI. 4 to 6, issue of V. 2; VI. 4, female, aet. nine, and VI. 5, female, aet. seven, both normal; VI. 6, female, aet. five, stellate cataract. Zirm concludes that the myopia was axial (due to lengthening of the eye-ball), and not lenticular. (Bibl. No. 64.)

Figs. 311—13: see p. 158; Figs. 314—315: see p. 144; Fig. 316: see p. 152.

GROUP IV. *Anterior and Posterior Polar Cataracts.*

PLATE XXXI. Fig. 339. *Harman's Case.* S———, P———, and T——— families. Microphthalmia and anterior polar cataract. The defect, which has appeared in four members, in two generations and three sibships, constitutes a serious and irremediable form of blindness. The eyeball is very small, the corneal diameter measures 7 to 7·5 mm. (average normal 11·6 mm.), the iris is poorly developed, the pupil eccentric, and on the front of the lens is a plaque of dense white tissue. Both eyes are equally affected. There is marked internal squint and constant nystagmus. Mentally the affected ones are below average; one died of paralysis in an asylum, but an unaffected sister made a similar end also. The vision of the affected is very poor, but they see enough to find their way about. III. 2 is worst having only perception of light. III. 8 can read Jaeger 12 type at 5 cm. Pedigree begins with the S——— family, two are remembered, but the state of their eyes is unknown. I. 1 had two children, II. 1 and 2, who were normal. I. 3 married twice, nothing is known of her or her husband's eyes; it would seem the initial defect, either actual or potential, lay with her, for in each of her families by her two husbands she had one affected child. I. 5 to 7 represent in proper order the sibship from which the consort of the progenitor of III. 1—8 sprang. I. 5 is a healthy old lady, aet. 73, the eldest of ten of whom eight are alive with good eyes. II. 1 and 2 normal. II. 3 and 4 first family of I. 3. II. 3, male, dead, good eyes. II. 4, female, dead, "she had tiny eyes and a terrible squint"; neither married. II. 5 to 9 second family of I. 3. II. 5, 6 and 7, females, normal eyes, never married. II. 8, female, normal eyes, died in a lunatic asylum. II. 9, male, died aet. 40, of paralysis in an asylum, "very small eyes, bad squint, sight extremely bad, like his two daughters III. 2 and 8." II. 10, wife of 9, fairly robust and healthy, normal eyes. III. 1

to 8, issue of II. 9 and 10, father affected, mother normal. III. 1, female, act. 24, normal, one normal child. III. 2, female, aet. 20, affected, sight equals perception of light only, inmate of blind asylum. III. 3, female, aet. 19, normal. III. 4, female, aet. 16, normal. III. 6, male, died aet. one year ten months, "eyes were perfect." III. 7, male, died aet. eight weeks, eyes were perfect. III. 8, female, aet. 12, affected, vision Jaeger 12 at 5 cm.; in a blind school; she has a high-arched palate and badly placed teeth, but dental enamel is normal; her head is considerably smaller than average and her intelligence is defective. (See Plate M, figs. 30—3 for drawings of cataract.) (Bibl. No. 90.)

Fig. 340. *Pflugk's Case*, reported by Loeb (xliii.). III. 2, "congenital posterior cataract and right microphthalmia; his father (II. 1) was operated on for the same condition; one of his grandparents (I. 1) was also operated on. One normal brother (or sister), one with same condition (III. 1 and 4); wife (III. 3) normal. One maternal aunt (II. 3) myopic. IV. 1 one son operated on for same condition." No other data. (Bibl. No. 86.)

Fig. 341. *Harman's Case.* W—— and T—— families. Posterior Polar Cataract. The type of cataract scarcely varies amongst eight of the ten affected members, the eyes are rather small; the lens when the pupil is undilated shows a deep sheen. The iris dilates to a midriatic only partially to 6 to 8 mm. Then the cataract is clearly seen. It affects the central half of the posterior part of the capsule and forwards to include more or less of the nucleus. In the three girls, III. 10 to 12 the gradations are easily distinguished; III. 10 has the usual type of cataract; III. 11 the opacity includes the whole nucleus; III. 12 the opacity springs from a broad base posteriorly, includes the nucleus, and a large anterior knob bulges slightly the anterior capsule so as to fill the undilated pupil; looked at with oblique focal illumination and dilated pupil the opacity looks like a white rock nearly submerged in a pool of clear water. From the similarity of these appearances to others where the persistence of the embryonic hyaloid artery has been demonstrated (Plate M, figs. 33, 34), I am inclined to think that such an artery persists in these cases. The family generally are stunted or undersized, particularly the affected members. Intelligence is below average. Dental enamel good, no case of pitted or honeycombed teeth found even when there was gross rickets of the bony skeleton. No consanguinity. The pedigree covers about 50 individuals; in the affected branches are 22, of these 10 are affected in three generations. The inheritance is continuous. Vision—all the affected have internal squint and constant nystagmus; they can see well enough for general purposes and count fingers at 1 metre. III. 3 can spell out words in a newspaper at 5 cm. with one eye. III. 11 and 12 are most nearly blind, for in them the cataract is largest. I. 3, the male progenitor, came from Hereford, he and his brothers and sisters were working tailors, all are said to have had good sight. I. 6, his wife, lived to aet. 89, had perfect sight, as also her brother and sister are said to have had. II. 1, female, died aet. 27, good eyes, married, no children. II. 2, male, aet. 67, typical cataract; wife normal, five children, three affected. II. 3, female, died young, eyes "quite right." II. 4, female, died after birth of second child, children normal. II. 5, female, aet. 50, typical cataract, husband normal, three girls all affected. II. 6, male, cannot be traced, he and his children said to have good eyes. III. 3—7, issue of II. 2, father affected, mother normal. III. 3, male, aet. 36, typical cataract; wife normal, eight children, two affected, four have other ocular defects (note this woman had previously a normal-eyed child by another man, III. 1). III. 4, female, aet. 34, normal, six normal children. III. 5, female, aet. 30, normal, married, no children. III. 6, female, aet. 33, unmarried, typical cataract in left eye, sight lost by unsuccessful operation, aet. 11. III. 7, female, aet. 30, unmarried, typical cataract. III. 10 to 12, issue of II. 5, mother affected, father normal, females, aet. 22, 19 and 14, all affected, for description, *vide supra*. IV. 1, normal child of III. 2 by normal husband. IV. 2 to 9, children of III. 2 by affected husband. IV. 2, male, aet. 14, IV. 6, female, aet. eight, IV. 7, male, aet. six, and IV. 8, female, aet. five, each have clear lenses, but considerable astigmatism and Fuchs' colobomata of the optic discs. IV. 3, female, aet. 11, normal. IV. 5, female, aet. nine, normal. IV. 4, female, died aet. four, of meningitis, had eyes like father. IV. 9, male, aet. two and a half, typical cataract. IV. 6, 7, 8 and 9 have bad rickets of the bones associated with malnutrition and poverty. (See Plate M, figs. 32, 33, for drawing of cataract.) (Bibl. No. 90.)

Fig. 335[b]. *Bronner's Case* (i). Microphthalmia and cataract. I. 1, G——, a strong, healthy man, aet. 45, had microphthalmia, cataract, and lateral nystagmus since birth; no member of his family known to be similarly affected; right eye, operation, vision still bad: his wife, I. 2, is normal; no consanguinity. II. 1—8, eight children; II. 1, still-born, eyes not affected; II. 2, male, aet. 21, normal; II. 3, female, aet. 18, microphthalmia, cataracts, nystagmus, and squint; operations done, vision bad; II. 4 and 7, males, normal; II. 5, 6 and 8, males, all microphthalmia, cataracts, and nystagmus. The eyes and corneae were small, no coloboma of iris or choroid found. Character of cataract not given in detail. No history of alcoholism or syphilis. (Bibl. No. 76.)

Fig. 338[b]. *Argyll Robertson's Case.* Microphthalmia and cataract. "A boy (II. 2), aet. six or eight, was seen on account of cataract, and there was very marked microphthalmia; the father (I. 1) of the boy was found to have the same condition; operation in early life gave vision sufficient for coarse work the boy's eyes were exact counterparts of his father's with nystagmus." (Bibl. No. 77.)

Fig. 341[b]. *Bronner's Case* (ii). Microphthalmia and cataract. II. 1, Mrs K——, had bilateral congenital microphthalmia, cataract, and nystagmus; right eye lost by operation, which failed; "the woman says she is married, but parentage of her children is dubious." III. 1—6, Mrs K——, had six children, two died very young; two girls and two boys seen by author, all had bilateral microphthalmia, cataracts, and two had nystagmus and squint in addition; irides normal; character of cataract not given in detail; operations performed, but vision bad. I. 1, "the mother of the woman is said to have had similar eyes," i.e. very small, "which were always on the move," and her vision was very bad. (Bibl. No. 76.)

GROUP V. *Congenital Cataracts. Variety not specified.*

PLATE XXVIII. Fig. 284. *Donald Gunn's Case*, continued by Lister and Parsons. Variety of cataract not specified. I. 1, female, cataract became apparent about age of 18 years; married and had issue. II. 1—7 sibship of seven. II. 3, 4, 5 and 6, all males, were "born with cataract"; of the three, II. 1, 2, 7, who escaped, II. 1 was a girl, the sex of the other two not stated. II. 3, male, had cataract operation, aet. 16. He married and his wife had issue: III. 1, female, hypermetropic astigmatism, but clear lenses; III. 2, female, seen aet. 2 with shrunken cataracts and nystagmus, at aet. 10½ operation performed and vision improved; III. 3, male, good eyes; III. 4, male, seen aet. one year with double cataract (no description), and has since had operations performed; III. 5 is a miscarriage, position in sibship unknown. (Bibl. No. 72.)

Figs. 285 etc.: see p. 139.

PLATE XXIX. Fig. 309. *J. C. Minor's Case*, reported by Loeb (xli.). "I recall a husband (I. 1) and wife (I. 2) each blind, having five children (II. 1—5), all of whom were blind (congenital cataract)." No other details. (Bibl. No. 86.)

Fig. 310: see p. 156.

Fig. 311. *Sir Wm. Adams's Case.* He saw two brothers, II. 1—2, from Dorsetshire, aet. 20 and 18 respectively, with shrunken congenital cataracts, which he needled with satisfactory results. (Bibl. No. 34.)

Fig. 312. *Maunoir's Case.* Form of cataract not stated. I. 1, II. 3, 4, 5, III. 3 and 4 were operated on for cataract, ages not stated. III. 3 and 4 were paternal first cousins to III. 2, but exact parentage not given. II. 1 and 2 are said to have had good eyes; of their children: III. 1 represents several girls, who were normal; III. 2, a female, who had cataract, aet. 30 years; of her children: IV. 1, sex not stated, was born with cataract; IV. 2, three others, sex not stated, normal-eyed. No other details. (Bibl. No. 39.)

Fig. 313. *Sir Wm. Adams's Case.* "In Dublin, I operated upon one of seven children, five of whom were born with cataracts, whose grandfather (I. 1) and father (II. 1), according to the mother's statement, were both born blind of the same disease. Of the seven, the two eldest brothers (III. 1—2) enjoyed perfect vision, whilst the five younger sisters (III. 3—7) laboured under congenital cataract." I. 1, grandfather II. 1, father of III. 1—7; III. 1, 2, males, good sight; III. 3—7, females, born with double cataract; III. 7, died in infancy: but III. 3, 4, 5 and 6 were seen by Adams at ages varying from nineteen to seven; all four had nystagmus, the three elder had had operations; III. 6, aet. seven, had shrunken cataract in the right eye and "fluid" cataract in the left; operations successful. (Bibl. Nos. 34 and 35.)

Figs. 314—315: see p. 144; Figs. 316—326: see pp. 152—5.

PLATE XXX. Fig. 327. *Westhoff's Case.* Form of cataract not stated. I. 1, male, perfect sight, died of apoplexy. I. 2, female, wife of I. 1, aet. 60, perfect sight; these had a child, II. 2, good eyes. I. 3, male, died aet. 66 of cancer of stomach. I. 4, female, wife of 3, aet. 76, good sight; these had a child II. 3, good eyes. II. 2, male, carpenter, good health, perfect eyes; married II. 3, female, healthy, slight hypermetropia; they had a family of six. III. 1, still-born, male; III. 2, male, parents noticed pupils looked grey soon after birth; seen by Westhoff aet. seven months, complete cataracts; operated aet. 1½ years; vision aet. six good; no fits; general condition very good. III. 3, female, seen soon after birth, no trace of cataract; died aet. 16 months of meningitis. III. 4, male, seen a few months old with double complete cataract and nystagmus, operation successful; no convulsions. III. 5, female, died in convulsions, aet. four days, parents and nurse reported pupils quite black and clear. III. 6, male, seen with complete cataract in one eye, the other being healthy; exact age not stated; had some fits, otherwise healthy, no operation. The six children were born between 1887 and 1895, the longest interval, 2½ years, being between 4 and 5. No note as to syphilis. (Bibl. No. 73.)

PLATE XXXI. Fig. 328. *Clarence Loeb's Case.* W—— family. Form of cataract not stated. I. 1, male, "blind." II. 1 and 2, males, issue of I. 1, "both blind." III. 1, son of II. 1, "blind, he is known to have had cataract. It is a fair presumption that the others (i.e. his cousins), their fathers and their grandfather also had cataract." III. 2—4, issue of II. 2, "all blind." IV. 2—5, issue of III. 1. IV. 2, female, congenital cataract: married, one child, affected. IV. 3, male, cataract at aet. three; no children. IV. 4, male, cataract at aet. two; no children. IV. 5, male, congenital cataract; married. IV. 6, blind, but no cataract (condition not stated); one child, affected. V. 1, male, child of IV. 2, cataractous mother, has congenital cataract. V. 2, female, child of IV. 5, cataractous father, and IV. 6, "blind" mother, has congenital cataract. No other details. (Bibl. No. 86.)

Fig. 329. *A. D. Williams's Case.* Form of cataract not stated. I. 1, male, blind from congenital cataract, had four blind and some not blind children. II. 1—5, offspring of blind father. II. 1, 2 and 3, males, II. 4, sex not stated, blind from same kind of cataract. II. 5, not blind, sex and number not stated. III. 1—4, family of seven of affected father; sex not stated, three blind of same cataract, four not affected. III. 5 and 6, issue of affected father, both have cataract, sex not stated. III. 7—12, issue of affected father. III. 7, male, III. 9, sex not stated, affected; III. 10—12, three others, sex not stated, unaffected. IV. 1—4, sibship of four, due to affected father; sex not stated; three blind with cataract and inmates of a blind asylum; one not affected. (Bibl. No. 52.)

Fig. 330. *Pisenti's Case.* Ancestry of mental disease, consanguinity, with physical defect in fourth generation, and cataract of form not stated. I. 1, male, died mad; I. 2, his brother, had religious mania; I. 1, had two female children, one normal, the other died of a nervous disease, these produce the parents of the third generation. II. 1—8, a sibship of eight, of whom two males were epileptic, two females hysterical, two females tubercular, one female a religious maniac and one male (II. 8) described as idiotic; he marries the normal daughter, II. 9, of I. 1 and has several defective children. II. 10, the other daughter of I. 1, bears to II. 11, an "eccentric" man, three children, of whom some are defective; she then dies of some nervous disease. III. 1—4, sibship of idiot father (II. 8) and normal mother (II. 9). III. 1, male, tuberculous. III. 2 and 3, females, religious maniacs. III. 4, normal male, who marries III. 5 his maternal first cousin and begets six children, of whom five are defective. III. 6, male, mentally defective. III. 7, male, alcoholic maniac. IV. 1—6, family of III. 4 and 5, maternal first cousins, nothing being said to the discredit of their own mental state. IV. 1, male, dead, shrunken congenital cataract, nystagmus, and hydrocephalus. IV. 2, female, squints. IV. 3, male, one of twins, cataract and hydrocephalus. IV. 4, female, other of twins fairly normal. IV. 5, female, died aet. three of tubercular meningitis. IV. 6, male, cataract and hydrocephalus. (Bibl. No. 75.)

Fig. 331. *Dyer's Case.* A form of congenital or infantile cataract becoming worse in later life; males only affected in four generations. I. 1, "vision bad from childhood," operated for cataract aet. 54, saw well until death some years later. II., large sibship. II. 1, vision always "misty," blind aet. 40, operations for cataract aet. 62. II. 2, quite blind, age not given. II. 3, died young. II. 4, "several" daughters not affected. III. 1 and 2, cataract noted, no details. IV., "large family," in which all the sons have partial cataract and all the daughters are free; these offspring appear to have been children at date of record (1846). (Bibl. No. 41.)

Fig. 332. *Gerok's Case* (vii.) of "congenital" or "juvenile" cataract in four generations, but with a break in the inheritance. I. 1, male, "congenital" cataract. II. 1, sex not given; good eyes. II. 2, male, "congenital" cataract. III. 1, son of II. 1, who had good eyes, "juvenile" cataract, aet. eight. IV. 1, sex not given, "juvenile" cataract aet. five. (Bibl. No. 23.)

Fig. 333. *Baudon's Case.* Form of cataract not stated. Congenital cataract in four generations. I. 1 had cataract, he had four unaffected brothers, I. 2—5, no mention of sisters. II. 1—7, sibship of seven from affected father; five affected, two not affected, sex not stated. III. 1—4, sibship of four from affected parent; three affected, one not, sex not stated; these have no children. III. 5—8, sibship of four of an affected parent; three affected, one not, sex not stated; these have many children, all save one, believed to be normal. III. 9, normal offspring of affected parent. IV. 1, female, affected, offspring of affected parent, was seen aet. two and operated upon. IV. 2, 3, 4 represent normal sibships of many siblings born to other affected and unaffected parents. No other details. (Bibl. No. 50.)

Fig. 334. *Armaignac's Case.* Form of cataract unknown. I. 1, the father had cataract; he had issue, II. 1—8, eight children, of whom the six eldest had cataract; the sex of the siblings, and which of them gave birth to the two sets of siblings in generation III., are not stated. None of the cases in I. and II. were seen. III. 1—3, one sibship of seven. III. 1, female, aet. 29, has never been able to guide herself, double congenital cataract with normal pupils, good perception of light (implying the cataracts were complete); microphthalmia, squint, nystagmus; left cataract extracted, vision scarcely improved. III. 2, cataract. III. 3, five normal siblings, no data. III. 4 and 5, sibship of four. III. 4, male, first cousin to III. 1, had cataract at birth. III. 5, three normal siblings. IV. 1, offspring of III. 1, sex not stated, cataractous, no note of operation. IV. 2 and 3, issue of III. 4; IV. 2, left eye cataract complete, and was needled aet. five with success; right cataract incomplete. IV. 3, normal child. (Bibl. No. 53.)

Fig. 335. *Paton's Case.* P—e family. Congenital cataract, form unknown. I. 1 and 2, of whom nothing is known, had an only child, II. 1, he had congenital cataracts removed as a child; he had twelve children, III. 1—3, 5—13, of whom four, III. 3, male, and III. 6, 9, 11, female, are known to have had cataract in childhood, for which they had operations. III. 1, 10, 13, males, III. 5 and 12, females, said to be normal sighted. III. 2, 8, females, and 7, male, are dead. IV. 1—3, family of three of affected father. IV. 1, male, both lenses removed for cataract. IV. 2, male, died aet. 14 days. IV. 3, male, eyes normal; no consanguinity known. (IV. 1 is a scholar in a blind school under my care; he is mentally dull; vision is very bad despite successful removal of the cataracts, so that retina is poorly developed; teeth badly honeycombed. Father refused to allow further enquiry. N. B. H.) (Not previously published.)

Fig. 335[b]: see p. 157.

Fig. 336. *Appenzeller's Case* (v.). Inheritance of cataract of form not stated, and of malformation of fingers. I. 7, male, had malformed fingers. II. 4, daughter of I. 7, had malformed fingers. Generation III., cataract appears on the side not connected with either of the foregoing malformed persons. III. 1—6, siblings. III. 1, female, born blind. III. 2, normal male. III. 3, male, normal, married III. 7, a woman with malformed fingers, and got issue three normal children, one blind with malformed fingers, three malformed fingers and good eyes. III. 4, 5, 6, males, born blind, no particulars. IV. 1, six normal male offspring of normal parent, III. 2. IV. 2—9, offspring of III. 3, normal father, and III. 7, mother with malformed fingers. IV. 2, female, blind at birth, seen aet. ten with dense white cataracts, fingers of both hands malformed[1]. IV. 3, male, 4 and 6, females, all normal. IV. 5, 7, and 8, females, good eyes, malformed fingers. No consanguinity on either side in any generation. (Bibl. No. 58.)

Fig. 337. *Gerok's Case.* Form of cataract not stated. III. 1 and 2, husband and wife, were first cousins, one of them had "congenital" cataract, but it is not stated whether husband or wife. IV. 1—3, offspring of III. 1 and 2, all had cataract. II. 2, a case of cataract had occurred in this generation of siblings from which III. 1 and 2 derived their cousinship, but whether the affected one was parent of either III. 1 or 2 is not stated. (Bibl. No. 23.)

Fig. 338. *Cahnheim's Case.* Peculiar posterior cortical cataract in the left eye of a boy aet. six, in other respects healthy, whose father had been operated on for soft cataracts also in the left eye. The affected eye of the boy was somewhat smaller than its fellow, and its iris darker in colour. (Bibl. No. 45.)

Fig. 338[b]: see p. 157; Figs. 339—341[b]: see pp. 156—8; Figs. 342—344[a]: see pp. 147—151; Fig. 344[b]: see p. 165; and Fig. 345: see p. 145.

Plate XXXII. Fig. 345[b]. *Giffard's Case.* Omaha, U.S.A., reported by Loeb. "The father (I. 1) had congenital cataract, but had been operated on and recovered enough sight to let him go about and make his living as a preacher. He married a woman who had trachoma (I. 2), but no signs of hereditary eye defect. Their only child was a boy, who had congenital cataract." No other data. (Bibl. No. 86, xxv.)

Fig. 346. *Nettleship's Case.* Kingshott family. I. 1, male, good sight: I. 2, female, good sight; had repeated floodings whilst pregnant with II. 5, but not with II. 8, died of bronchitis when II. 11 was aet. about five months; had eleven children. II. 5, male, cataracts recognised the day after birth; when aet. three to four years, operated on several times; seen by Nettleship aet. eight, again aet. 17, vision with glasses fair; no fits, intelligence good but head narrow, and boy generally undergrown. II. 8, female, seen aet. three, with dense yellowish white central cataracts and nystagmus; the opacity consisted of a much shrunken, flattened lens, with nearly clear zone of capsule between it and ciliary processes, through which plenty of light passes; opacity friable to needle and easily broken up; one eye lost by suppuration; no fits. II. 11, died aet. five months; two other of the siblings died, aet. six and at birth; their places in the sibship not stated. (Bibl. No. 82, lvii.)

Fig. 347; see p. 151; Fig. 348: see p. 144.

Fig. 349. *Frank C. Todd's Case*, reported by Loeb. "C. L. D—— (III. 1), male, aet. 23, has hereditary cataract (form not stated, but in previous paragraph writer considers 'congenital and hereditary' disease). Father (II. 2) eyes normal; mother (II. 1) had cataract; maternal grandfather (I. 1), two uncles (II. 3, 4), five aunts (II. 5—9), three brothers (III. 3—5), several cousins (III. 10) all had cataract; two sisters (III. 6, 7), two brothers (III. 8, 9), and several cousins (III. 11) normal; one child (IV. 1), normal," aet. $4\frac{1}{2}$, sex not stated. No other details. (Bibl. No. 86, lvii.)

Fig. 350. *Hirschberg's Case.* L—— family. I. 1 and 2, parents of II. 1, sound. II. 1, female, aet. four months, complete double congenital cataracts; no complications; both lenses needled, with an interval; child died of "dysentery" before absorption complete. II. 2, sex not stated; this, the next child was brought with double complete milky cataract a few days after birth; pupils dilated unusually well to atropine; at aet. nine weeks, when brought for operation, spontaneous absorption had begun, pupil having become almost clear on temporal side in left, and nasal side in right eye; both needled with good results; child died in early life. (Bibl. No. 47.)

[1] Right hand: index and middle fingers short, second phalanges very small, first phalanx middle finger deformed. Left hand: second phalanx, index and little finger short, first and second phalanges of middle finger similarly deformed.

Fig. 351. *Hirschberg's Case.* R——. I. 1 and 2, parents first cousins, but had good eyes. II. 1, first-born, male, seen aet. 1½ with both eyes shrunken after unsuccessful operations performed at aet. six months; died a few days later of diphtheria; history shows that child was born with small eyes and opacity of lenses. II. 2, female, also born with opacity of lenses, and when seen, aet. six months, eyes were small, corneal diameter 8 mm., and pupils not dilatable by atropine; iridectomy performed, and periphery of lens then found to be clear, but exact character of the central opacity not made out; visual power good. (Bibl. No. 47.)

Fig. 352. *Mooren's Case.* Mooren states that he operated for cataract on a mother, I. 1, and on her infant, II. 1, that was born blind from cataract, with good results. (Bibl. No. 55.)

Fig. 353. *Guiot's Case.* A young mother, I. 1, with double congenital cataract which had been operated on with poor result. Her first-born child, II. 1, male, seen at three months of age with "incomplete central" cataract in both eyes. (Bibl. No. 61.)

Fig. 354. *Brudenell Carter's Case*, reported by Loeb. "A family in which the five children (II. 1—5) were all the subjects of congenital cataract, which was so trifling in the first as to be discoverable only by careful examination, somewhat more pronounced in the second, still more in the third, and in the fourth and fifth reached a degree which called for operation. The father (I. 1) was a robust man with perfect vision; the mother (I. 2) was dead, but no history of defective vision or of relationship to her husband remains in my memory. The children were all strong and healthy." (Bibl. No. 86, xvi.)

Fig. 355. *Komoto's Case*; Tokio, Japan, reported by Loeb. "Parents (I. 1 and 2) normal; two daughters (II. 1—2) congenital cataract; one son (II. 3) normal." No further data. (Bibl. No. 86, lxvi.)

Fig. 356. *Schnabel's Case.* Orthaber family. Three siblings (II. 1—3), aged eight, six and three years, with double "nuclear" cataract; no evidence of inheritance, direct or indirect. (Bibl. No. 62.)

Fig. 357. *Schnabel's Case.* Lechner family. Three siblings (II. 1—3), aged 6½, three years and eight weeks, with double "nuclear" cataract; no evidence of inheritance, direct or indirect. (Bibl. No. 62.)

Fig. 358. *Bock's Case.* I. 1, mother, aet. 43, had a peculiar form of congenital cataract; no evidence of constitutional disease. II 1, her daughter, aet. eight, double nuclear cataract; mother stated the child was born with the disease, and that vision had got worse; nystagmus, small corneae, deep anterior chambers, and tremulous irides. II. 2, other siblings, number not stated, said to have been born with cataract. Teeth good in mother and children, and no signs of rickets in any of them. No other data. (Bibl. No. 59.)

Fig. 359. *Lusardi's Case.* Watticaux family. A man, I. 1, had cataract since the age of 30; his children, II. 1—5, to the number of five, were born with the same malady; operations successful. No further data. (Bibl. No. 37.)

Fig. 360. *Lusardi's Case.* Decamp family. Fourteen children, III. 1—14, six had cataract; alternation of abnormal and normal—"first-born was cataractous; 2nd, good eyes; 3rd, cataractous, and so on." Twins occurred once; the first of the twins had good eyes and died aet. 17 months, from an accident; the 2nd, cataract. No further data. (Bibl. No. 37.)

Fig. 361. *Lusardi's Case.* Lantlin family. I. 1 had congenital cataract, but sees well enough to get about alone. II. 1, 2 and 3, two daughters and one son had cataract from birth; both daughters married; one had a daughter, III. 1, born with cataract. No details of the cataract or of the other siblings. (Bibl. No. 37.)

Fig. 362. *Millikin's Case* (ii.) Congenital cataract in five females in three generations; lenses small and hard. I. 1, a Swedish woman, operated on for congenital cataract; age not stated. II. 1, 2, females, both double congenital cataract; II. 1 operated on early, married, and had two cataractous children; II. 2, operated aet. six years, died six months later. III. 1—2, the only two children, females, of II. 1. III. 1, operated on aet. nine months, and did well, lenses small and hard. III. 2, operated aet. five years, did well. All five cataractous subjects were strong and healthy. No other data. (Bibl. No. 79.)

Fig. 363. *Millikin's Case* (iii.) A German family. Congenital cataract in two generations, combined with axial myopia. I. 1, father, aet. 46, both eyes operated on for cataract about aet. 40; fundus showed myopic changes; had poor sight all his life; no positive information whether cataracts were congenital. I. 2, mother, aet. 44, examined and eyes found normal. II. 1—7, issue of I. 1 and 2: II. 1, 2 and 7 had good eyes, no note of refraction. II. 3—6 all showed "congenital central opacities of lens varying somewhat in shape and size" with vision from $\frac{3}{60}$ to counting fingers; eyes well formed and patients well grown. All four were operated upon with good results. II. 4 and 5 had only 6 D. and 7 D. hypermetropia after operation (usual 10 D.), and must therefore have been myopic before. (Bibl. No. 79.)

Fig. 364. *Appenzeller's Case* (vii.) III. 1, a boy, aet. three years, had cataracts of indeterminate form and somewhat shrunken; said to have been born with them; no microphthalmia. II. 1, his father, had been operated on for cataract in early life. No consanguinity, and no history of cataract in father, I. 1, brothers and sisters, II. 2, nor their children, III. 2. (Bibl. No. 58.)

Fig. 365. *Appenzeller's Case* (ii.) I. 1, married I. 2, a man not related by blood, and had normal-eyed children; later she married I. 3, her first cousin, who had good eyes, and bore three children, I. 2—4; all were born with double congenital cataract. II. 2 (sex?) seen aet. four, no data of cataract. II. 3, female, seen aet. three, had nuclear cataracts with projecting spicules and clear cortex, it is described as peculiar, and was presumably not lamellar. II. 4 (sex?), born two years after II. 3, had double cataract, died at five weeks. No known malformations of ancestors of either I. 1 or I. 3. (Bibl. No. 58.)

SUPPLEMENTARY PEDIGREES

(Collected too late for Classification, see p. 138)

PLATE XXXIII. Fig. 366. *Bastard's Case.* I. 1, died aged 75, in 1786. He suffered from cataract. He had 10 children, of whom three, II. 15—17, died young. Of the others II. 1, normal, married II. 2, normal. All their children, III. 1, and their descendants were normal. II. 3, normal, married II. 4, normal, their children, III. 2, and their descendants were normal. II. 5, normal, married II. 6 who had, weak sight and died amaurotic. They had three children. Of these III. 4 had cataract, was operated on unsuccessfully and became completely blind. She married III. 3, but nothing is said of their descendants if any. III. 5, normal, married and had three children, IV. 2, IV. 3 and IV. 5, all affected. IV. 2 and IV. 3 were operated on with only moderate success, they married, but nothing is said of their descendants if any. IV. 5, the son, was also operated on with poor success—it does not state whether he was married or single: III. 7 was amaurotic but had no cataract. II. 7, normal, married II. 8, their children and their descendants were all normal. II. 9, affected, was operated on with moderate success. She married and had two children, III. 9—10, both affected and both operated on successfully. III. 10 was married, and the account does not state whether III. 9 was married or not, but it says a grandson of II. 9's was affected—presumably he was a son of III. 10, so he has been entered as IV. 6, he was operated on successfully. II. 11 affected and successfully operated on, married II. 12 normal and had seven children III. 12—18, only two of whom were affected, viz. III. 12 and III. 13, both successfully operated on. II. 13, normal, married II. 14, normal, and had a son, III. 19, who was affected and operated on successfully, her grandson, IV. 7, and great-grandson, V. 3, were also affected and successfully operated on. (Bibl. No. 42, p. 15.)

Fig. 369. *H. W. Williams' Case* (i). I. 1 and I. 2 were normal, and II. 9 knew of no case of cataract in any of her ancestors or in her eight brothers and sisters, II. 1—8. In II. 9 the lens exhibited in each eye a disseminated dotted opacity, the cloudy spots being most numerous near the centre, while the margin of the lens was comparatively clear. Consequently in a bright light she was almost entirely blind, but in a moderate light saw sufficiently well, to perform in a slovenly manner the household duties of a labourer's wife. The capsule of the lens in her eyes and also in those of the children was transparent. Her hair and irides were dark, the children had light hair and a gray or blue iris. She had six children, III. 1—6, of whom four, III. 1, III. 3, III. 4 and III. 6, were affected. The eyes of the children presented nearly similar appearances, and one description may answer for the whole. Nearly the entire field of the pupil was occupied by opacities consisting of dots of various sizes and evidently occupying different planes of the lens. In a bright light reflections from crystals of cholesterine could be plainly seen. All the patients had sufficient vision to enable them to find their way in a moderate light, but in a bright sunlight they were nearly blind. Six operations were performed on III. 1 aged about 17, III. 3 aged about 12, and III. 4 aged about 10. They were successful, II. 9 was timorous of her own eyes and did not wish III. 6 to be disturbed till he was older. (Bibl. No. 94, p. 149.)

Fig. 370. *H. W. Williams' Case* (ii). Very few details are given of this case. He says nothing of I. 1 and I. 2 and merely states there were seven children affected, II. 1—7, the others being free. (Bibl. No. 94, p. 150.)

Fig. 367. *Hübsch's Case.* I. 1 and I. 2 were healthy and had no ocular trouble. Only one of their children is mentioned, II. 2, who was born with double cataract and was operated on successfully at 10, by Dr Bernard, Director of the Imperial School of Medicine at Constantinople. She was married at 17 to II. 3, who had normal eyes. There was no consanguinity, one being a Circassian, the other a native of Asia Minor. They had six children, three boys and three girls, III. 1—6. III. 1 had opacities in the lenses at birth. She lived two years, and died of convulsions. III. 2, according to his parents, had clear pupils till the age of three months, then the left began to look muddy and soon became quite white. At the age of six months cataract developed in the other eye. He died of scarlatina aged 18 months. III. 3, aged 10,

was normal. III. 4 developed cataract in the same way, first in the right eye, later in the left eye. The cataract began at the age of six months, and the opacity was complete at 14 months. She died of cerebral affection after a few days illness. III. 5, aged eight, was brought to Hübsch by her father. Since the second month after birth she had complete lenticular opacity in the left eye, incomplete in right eye. Vision was not completely gone, she could distinguish between natural and artificial light and between certain colours, and could find her way about the house. Examination showed a soft cataract in the left eye, fairly uniformly milky, and in the right eye an irregular opacity with more or less opaque patches and other parts more or less transparent, and two synechia, the one above and the other below, evident proof that there had been inflammation of this eye. She was operated on with fair success. She could not see fine or delicate objects clearly, but could go about alone and recognise those whom she met. III. 6, aged 18 months, when first seen was a healthy boy. He had had cataract in the right eye for two months. The opacity was uniform and milky, he could not see with this eye. The left eye was then clear but he became completely blind at the age of two years. Hübsch was afraid to operate on him so young. (Bibl. No. 95, p. 147.)

Fig. 368. *Mayerhausen's Case.* Microphthalmia with anterior and posterior polar cataract. No statement was made with regard to I. 1, he was presumably normal. I. 2 was examined at the age of 83. She had seen well in youth, but at 40 her sight declined, and at 42 she was operated on (discission). The operation was successful in right eye, but the left could only distinguish light and darkness. She had a slight degree of ptosis. Her eyeballs were about $\frac{3}{4}$ normal size. There was no nystagmus. II. 2 was seen when aged 53. She kept both eyes closed for several days after birth, they were opened by the doctor. According to the parents the eyes were quite clear, not inflamed and showed no sign of abnormal secretion. But they soon remarked that her sight was very bad, and her eyes were never quiet. Her playthings had to be put quite close. She never learnt to write, and could only read with the book close to her eyes. At 20 she had variola, and afterwards her sight got much worse, and she was operated on (very probably discission was done) and her sight improved. When examined she had ptosis of the upper eye-lid. Both eyeballs were $\frac{2}{3}$ the normal size, but were perfectly formed. She had nystagmus oscillatorius. The left pupil was very small and rather round. To the posterior surface of the sphincter adhered a small chalk-white round mass, which appeared about 1 mm. in diameter, the tissue of the iris at this spot was thinner, the white mass extended behind the iris. There were also some soft grayish capsular remains visible, adhering to the sphincter. The right pupil was very narrow and oval in form. Grayish white withered capsular remains adhered to both outside and inside, so that there was only a cleft about 1 mm. broad free, when the pupil was not dilated by atropine, through which a red light was reflected from the back of the eye. II. 2 also had coloboma choroidiae. She married II. 3, who had normal eyes, and they had nine children, III. 1—9. Six of these children, III. 1—6, died at ages of 18 weeks, 1, 2, $2\frac{1}{2}$, 6 and 8 years respectively, all these had normal eyes. Three children lived. III. 7 and III. 8, aged 17 and 12, were normal, III. 9, aged 10, was affected like his mother, II. 2. She said III. 9 did not open his eyes himself after birth, the father opened the lids a few days after, since: "Stern nicht recht in Ordnung sei." At school he could not learn to read or write. When examined his eyeballs were larger than the mother's $\frac{3}{4}$ normal size, the nystagmus was worse, it was both lateral and rotatory. The cornea was disproportionately small in comparison with the fairly normal size of the eyeballs. In the right eye on the anterior capsule of the lens was a small triangular chalk-white rather prominent opacity about $1\frac{1}{2}$ mm. in diameter occupying the centre of the pupil region. In the neighbourhood of the posterior pole of the lens, there was a central whitish opacity about $2\frac{1}{2}$—3 mm. in diameter, with an uneven granular-like surface which projected into the posterior layers of the otherwise clear lens. When illuminated from below this appeared to be united to the anterior opacity by a thin white cord going through the axis of the crystal body. In the left eye on the anterior capsule there were several (about six or eight) small white opacities, like points scattered over the pupillary region and appearing somewhat raised. From the posterior capsule extending into the cortical tissues was a grayish white opacity, which, in its greatest diameter, extended through the whole pupillary region, and allowed a red reflex to come through at each side. Mayerhausen says both II. 2 and III. 9 had microphthalmia on both sides, anomalies of the eyelids, cataract and nystagmus. As to the kind of cataract he could only speak with certainty with regard to III. 9. He had in both eyes distinct capsular cataract combined in the right eye with pure posterior polar cataract, and in the left eye with a peculiar form of posterior capsular opacity, which might be called "Cataracta capsularis anter. punctata." He thought it probable II. 2 had same kind of cataract. (Bibl. No. 98, p. 97.)

Fig. 372[b]. *Bell's Case.* No statement is made with regard to I. 1, who was presumably normal. I. 2 had been operated on for cataract at age of 14 with considerable benefit. She was dead. Of her children, four, 1—4, were affected, but it does not say whether she had other unaffected children or not. II. 1, aged 25, had very defective vision from cataract, but she was not examined. II. 2, aged 17, was born with defective sight, but could see tolerably well till six years of age, when the symptoms became aggravated. At 17 she could find her way about her home fairly well, but was quite incapable of following any employment. The right eye had a large anterior chamber indicating absorption of the lens,

and the pupil, which dilated freely under atropine, was occupied by the remains of a capsulo-lenticular cataract, reflecting little light and not very readily observed. The left pupil also dilated freely, and was occupied by a large soft cataract of milky appearance. She was operated on successfully. II. 3, aged 15, could see pretty well for several years after birth, but the cataracts became complete even at an earlier period than in the case of his sister, II. 2. The pupils responded to variations of light. They were occupied by cataracts, which, being very white and flat on the surface, appeared to be mainly capsular, the lenticular substance being probably in great measure absorbed. He was operated on successfully. II. 4, aged 13, had never perceived any object nor any colour, even the brightest. Both pupils which dilated readily under atropine without any improvement to his vision were occupied by white cataracts more distinctly capsular than in the case of his brother, II. 3. In the middle of the right capsule there was a round portion about the size of a small pin-head, of a yellow hue, which suggested the idea of its being cretaceous. He was operated on successfully. (Bibl. No. 96, p. 1063.)

Fig. 372. *Gjersing's Case.* This case was first described by Gjersing in 1878, and Norrie in 1896 gave a further account of the family, having examined one member of it, V. 1, as a recruit. He was able to add practically the whole of Gen. V. to the pedigree, the particulars being obtained from the doctor of the district in which they lived. I. 1 was first affected in her old age, but there is no doubt she had cataract. Her two daughters, II. 1 and II. 3, became affected at about 40 years of age. II. 1 had a son and three daughters, of these the son, III. 1, and the two younger daughters, III. 5 and III. 7, became affected at about 30, while the elder daughter, III. 3, was normal. Of their 13 children in the next generation IV., eleven had cataract without doubt, they either became affected at about seven years of age, or at least examination at school showed they had cataract at that age. Gjersing examined most of these cases and found they had cataract on both sides, there being, with few exceptions, a small white sharply defined irregular patch in the centre of the lens, from which now and then issued small rays. III. 1 died aged 55, he had two children, the elder, IV. 1, affected, married, and had seven children. V. 1—7, of whom five V. 1—5, possibly had cataract. Gjersing saw the three elder, V. 1—3, then aged respectively seven, five and two, and stated that at that time the lenses of V. 1 and V. 3 were clear, while V. 2 had little points of diffused cataract in her right eye, a central small white patch in the left eye. V. 1 was seen by Norrie as a recruit, and examined. He then had cataracta stratiformis (this probably means lamellar cataract). V. 3 died aged 10. Norrie says that there is some doubt whether she had cataract or not. V. 4 is another case of doubtful cataract. V. 5 was affected, V. 6—7 were normal, IV. 3, affected, had seven children, of whom V. 8—9 had cataract and the remaining five, V. 10, were normal. III. 3, normal, married, and had two daughters, IV. 5—6. IV. 5, stated to be normal by Gjersing, was by Norrie said to be a case of doubtful cataract, and is marked as doubtful. She was unmarried. IV. 6, normal, married, but dead, and had at least three children, V. 11—13, whose condition was unknown. III. 5, affected, married, and had two sons both affected, of whom IV. 8 was childless, and IV. 10 had several children whose condition was unknown. III. 5 herself went to Hospital at the age of 51, and the diagnosis was: In right eye cataracta matura. In left eye cataracta nondum matura. She was operated on, and the operation was fairly successful. III. 7, affected, had seven children, of whom IV. 12, affected, was dead. IV. 13, affected, married and had four children, V. 16—19. V. 16 is given by Norrie as a doubtful case of cataract. V. 17—19 were normal. IV. 15, affected, was unmarried. She came to Hospital at age of 27. Had cataracta stratiformis(?) irregularis (congenita) and excessive myopia. Sight right eye $\frac{15}{200}$ with concavity $5\frac{1}{2}$. Left eye could count fingers at 4 ft. distance. She was operated on, and at first operation appeared to be successful, but Gjersing reported later that her sight had gradually become much worse. IV. 16, affected, married, and had eight children, V. 20—27, of whom two daughters, V. 20—21, were affected, and the others, V. 22—27, were normal. IV. 18, affected, had one child, V. 28, also affected. IV. 20, affected, married, and had five children, V. 29—33. V. 29 was affected; V. 30 was also affected, and died aged 11; V. 31 was affected, and the other two, V. 32—33, were normal. IV. 22, affected, married, and had eight children, V. 34—41. Of these V. 34—36 had cataract, and V. 37—41 were normal, V. 41 was dead. II. 3 became affected about 40 years of age. Her only daughter, III. 10, was noticed to have weak sight at school in her seventh year, and was operated on in her twelfth year. She married and had a son and a daughter, IV. 24 and IV. 26. IV. 24, normal, had four children, V. 42—45. Of these V. 42, affected, died aged 9; V. 43—45 were normal, but V. 45 died aged 1 year. IV. 26, affected, had cataract soon after birth. She had cataracta stratiformis retrograda (congenita) in her right eye, she could count fingers at the distance of the length of the room. She had cataracta retrograda in the left eye, with which she could just distinguish the movements of the hands. She was operated on and afterwards could read a book with large print. She married and had two children, V. 46—47. V. 46 was affected; V. 47 was a doubtful case. Gjersing states that cataract appeared in this family in many forms. That I. 1, II. 1 and II. 3, III. 5 and III. 10 had ordinary capsular cataract, III. 1 and III. 7 central lenticular cataract (central cataract of the lens). Gen. IV. had most frequently central lenticular cataract, but there were many complicated forms, and V. 2, when a four year old child, had central lenticular cataract in one eye, and diffuse capsular cataract in the other eye. Norrie says the family is a typical example of inherited cataracta stratiformis (?lamellar cataract). (Bibl. No. 97, p. 273 and Bibl. No. 100, p. 942.)

Fig. 371. *Norrie's Case.* There are not many details given in this case. I. 1, normal, had three daughters, all unaffected. Of these daughters II. 1 had four children, III. 1—4, all daughters, no particulars are given of them in the pedigree, so presumably they were normal. Norrie counts them as normal in giving the number affected in each generation. II. 3 married a naval surgeon, II. 4, in 1797. He died from the result of an accident. They had four children. Of these III. 6, affected, married, and had four children. Of these children IV. 1, affected, had ten children, V. 1—7. V. 1—4 all had cataract, V. 5 was normal; V. 6, affected, died aged 3; V. 7, four children, who died under three years of age. IV. 3, normal, had a daughter, V. 8, affected, who married her first cousin, V. 9, normal, son of normal parents, IV. 5 and IV. 6. They had one daughter, VI. 1, who was very young when the account was written and so far normal. IV. 7 was normal. III. 7 and III. 8 were normal, married, and had normal children, IV. 8 and IV. 9. III. 9, affected, died aged 85, he had seven children, four affected, three normal. Of these IV 10, affected, died aged 4. IV. 11, affected, married, and had three children, V. 10—12, of whom two, V. 10 and V. 12 were affected, the daughter, V. 11, being unaffected. IV. 13, affected, was unmarried. IV. 14 was normal. It does not state whether he was married or not. IV. 15, affected, married and had five children, V. 13, all normal. IV. 17, normal, was childless. IV. 19 died in infancy not one year old. II. 5, normal, married II. 6, normal, and had four children. Of these III. 11, normal, married and had 17 children, of whom nine, IV. 33—41, died between one and two years of age. Of the survivors two were affected, IV. 20 and IV. 25. IV. 20 had nine children. Of these V. 14—15 were normal, V. 16 and V. 19 died young. V. 17, affected, married, and had four children, VI. 2—5, of whom three, VI. 3—5, died young, and VI. 2 had cataract. V. 20 and V. 22 were affected. V. 21 was normal, and V. 23 died aged 1½ years. IV. 22, normal, was childless. IV. 23, normal, married, and had two children, V. 24—25. V. 24 was normal. V. 25 died aged two years. IV. 25, affected, married, and had eight children, V. 26—27. Three of these children, V. 26, were normal, the other five, V. 27, died young, IV. 27, normal, married, and had six children, V. 28—33, of whom four daughters, V. 28—30 and V. 33 were affected, the two sons, V. 31—32, being normal. IV. 29, affected, was childless. IV. 30, normal, married IV. 31, normal, and had six normal children, V. 34. IV. 32, normal, was childless. III. 13 died aged six months. III. 14 died, aged eight; III. 15, affected, married, and had six children, of whom only one was certainly affected, IV. 48. IV. 42 died, aged nine months. IV. 43 died, aged four years. IV. 44 died, aged five years. IV. 45, normal, had three sons, V. 35—37, all normal. IV. 47 was normal. IV. 48, affected, married, and had four children, V. 38—41. Of these V. 38 died, aged six; V. 39 was affected; V. 40—41 both normal. Norrie states that in the first two generations no one seems to have had cataract; in the third generation 3 out of 12 persons; in the fourth 9 out of 34, in the fifth 17 out of 61, and in the sixth 1 out of 5. In all thirty affected persons. These numbers are not quite correct. Instead of 61 persons Norrie gives only 58 in Generation V. in his pedigree, and only sixteen were apparently affected. As he marks the difference of affected and non-affected by a change of type, which is not very marked, it is possible that a slip has occurred. He considers that the total affected may easily have been underestimated; many he had himself seen had cataract in such a slight degree, that their vision was quite good, and therefore it is possible that some of those really affected would not be reported. He suggests, but appears doubtful as to this, that the cataract developed when the children were between three and four years old; he realised that lamellar cataract may appear at birth, and states that it will be interesting as bearing on this point to notice whether the normal children, now under three years of age, will develope cataract—a remark hardly needful, if he knew that other members had certainly started cataract between three and four years. He draws attention to the great infantile mortality, not less than 33 members of the stock having died as children. (See Bibl. No. 100, p. 938.)

PLATE XXXII. Fig. 344[b]. *Harman's Case.* F——, W—— family. I. 1, 2; III. 11 reports that her father spoke of his parents frequently, but never said their sight was bad like his own. Of I. 3 and 4 nothing is known. II. 1, male, died aet. 87, believed to have been an only child, "he never mentioned brothers or sisters," had cataract as long as could be remembered, blind for many years before death, refused to have operations. II. 2, his wife, died aet. 75, good sight, as also her two brothers, II. 3. III. 1—11, offspring of affected father. III. 1, female, aet. 60; III. 2, male, aet. 56; III. 3, female, aet. 52; III. 4, male, aet. 51; all reported perfect sight; all married; III. 2, 3 and 4 have large families, all good sight. III. 5, male, died young of small-pox, born with cataract, had operations. III. 6, male, and 7, female, died infants; III. 8, female, aet. 45, born with cataracts, operated on; III. 9 and 10, sex unknown, died shortly after birth; III. 11, female, aet. 39, lamellar cataracts and high myopia, left cataract operated with only fair results; teeth honeycombed; her husband normal. IV. 4 and 5, sibship of two from affected mother, no other pregnancies; IV. 4, male, aet. 14, born with cataracts; right operation failed, eye removed, left fairly good result; teeth normal; IV. 15, male, aet. 12, normal. (Unpublished.)

DESCRIPTION OF ILLUSTRATIVE PLATES

(*Figs.* 1—7, *drawings and photographs of cataractous lenses removed from the eye*).

PLATE L. Fig. 1. Lawford's section of lamellar cataract removed by operation. Male, aet. 44, never had good sight. In each eye fairly dense lamellar opacity. Teeth much discoloured and irregular from loss of enamel. History of infantile fits not known. Both lenses removed by Nettleship in 1886. Vision considerably improved. The lenses were hardened in Müller's fluid and alcohol, and embedded in celloidin. Anterio-posterior section of the right is figured, sections examined stained and unstained, the latter proved the more useful. Right lens in fresh state had irregular surface from loss of capsule and notch cut by cystitome in anterior face; measures 8 × 8·25 mm. transversely. The opacity is plainly visible, very thin, with tiny spokes jutting out from it; measures 5·25 mm. transversely. Section: the central defective area is rather nearer the posterior than the anterior surface. It measures 2·325 mm. anterio-posteriorly, 5 to 5·25 mm. transversely. It contains numerous, irregularly shaped spots and patches, which vary considerably in size, and are generally larger and less closely packed in the central part than towards the boundary of this area. In unstained specimens they are bright and highly refracting. Although this area is very distinct from the cortical part there is no continuous line separating the two parts of the lens. The boundary line (*l*) between the central and cortical areas has a peculiar appearance not unlike that of a twisted rope, and its edges are slightly serrated; it varies in thickness a little, its measured diameter being from 0·0125 mm. to twice that thickness. The lens fibres have a decided tendency to split along this line, as though it formed a layer of degeneration, or of some change which reduced the adhesiveness of the adjoining layers. In the upper extremity of the figure there is an indication of a double line. The dots or granules in the cortical part reach generally quite up to this line, but do not extend beyond it. (Drawn by M. H. Lapidge. Original lithograph measures 17 cm. in length. In the plate the figure is reduced to about × 8 diameters and inverted to agree with other figures. Reproduced by kind permission from *Royal Lond. Ophthal. Hosp. Reports*, Vol. XII., 1889, p. 194.)

Fig. 2. Hess's section of lens with lamellar cataract; × 8. The layers with minute white dots indicate opacity. Complete layer around nucleus, large plate at posterior pole, smaller at anterior pole. Reproduced from print lent by Hess: see Graefe-Saemisch, *Handbuch der gesammten Augenheilkunde*, Bd. VI. Abt. 2. Leipzig, 1905, S. 134. The author describes the case thus:—"Drawing of a cataract removed from a man aet. 37, a subject of rickets in early life. The spots of degeneration are not confined to the perinuclear zone, and nucleus, but affect also the outer layer of the lens particularly at the poles."

Fig. 3. Norris' section of lamellar cataract; hereditary case. Small lamella around nucleus; fragmentary outer lamella, particularly in front. Reproduced from Norris and Oliver's *System of Diseases of the Eye*, IV., Fig. 71, p. 329. The author describes the case thus:—"Lens extracted from a man aet. 35, with zonular cataract, in whose family cataract was hereditary, and whose niece, aet. 12, was under treatment for zonular cataracts. The microphotograph shows that the nucleus of the lens exhibits small, irregularly-rounded dots of degeneration, some of these dots being filled with granular material, whilst others have bright, highly refracting contents; that minute spots filled with granular material are the cause of the perinuclear opacity; that the cortical fibres adjacent to the nucleus are fairly clear, although they exhibit slight, irregularly-rounded spots of degeneration, and that the whole mass is moderately coherent, with but little formation of cracks or cavities between the lens-fibres, those near the posterior surface being evidently artificial." The measurement of the cataract is not given, but it is evident that the opaque lamella is much smaller than in Lawford's non-hereditary case. The original figure measures 20 cm. in length, reduced to about × 8 diameters.

Fig. 4. Deutschmann's section of lens with very small perinuclear lamellar cataract, and *b* outlying secondary incomplete lamellar cataract. Reproduced from *Archiv für Opthalmologie*, 1886, S. 298. Section of left lens of eye of a suicide removed 12 hours after death. Age and sex not stated. Eyes preserved in Müller's fluid and alcohol. Figure reproduced shows enlargement about 4 diameters. Sections show under various magnification: (1) the nucleus and peripheral cortex to the normal; (2) surrounding the nucleus is a complete layer of opacity well defined by its colour, it is thicker at the poles than elsewhere; (3) a layer of clear lens substance about half as thick as (2); succeeded by (4) a second stratum (*b*) of opacity which however is interrupted between the poles and equator both anteriorly and posteriorly, and is thickest about the anterior pole. The opaque layer consists of lens-fibres containing myeline drops and granular fatty material with inter-fibrillary vacuoles and fissure; there is also some vacuolation and swelling of lens-fibres between the equatorial parts of the two opaque layers. It is evident that if this lens had been examined by focal illumination during life the equatorial portions of the outer layer would have appeared as "riders," its central part as a patch put beneath the anterior pole of the lens.

Fig. 5. Lamellar cataract photographed in water immediately after extraction. Extracted from an adult by Lang. No other data. Reproduced from block kindly lent by J. H. Parsons.

Fig. 6. Schirmer's section of lamellar cataract removed by operation; *a* clear nucleus, *b* lamellar opacity, *c* clear cortex. Lens extracted from left eye of boy aet. ten, who did not walk until aet. 2; no other particulars known about him. The lens had been needled six weeks previous to extraction with hardly any result. The lens was hardened in Müller's fluid and in alcohol. After hardening the following measurements were obtained:

Equatorial diameter 7·5 mm. (author says that a uniform layer of cortex seems to have peeled off during extraction).
Antero-posterior diameter 3·5 mm.
Equatorial diameter of opaque layer 5·75 mm.
Antero-posterior diameter of opaque layer 3·00 mm.
Thickness of diameter of opaque layer 0·25 to 0·3 mm.

Macroscopically. The opaque layer is quite sharply defined as to both its borders (in section); in front it comes quite to the surface of lens (on account of loss of cortex); the hinder part is covered by a thin layer of cortex; the outline of the nucleus is well defined.
Microscopically. The cortex surrounding the opaque layer is quite normal. The nucleus is not normal, it shows numerous scattered rather large vacuoles of same appearance as, but larger and much less close together, than those constituting the opaque layer, they are filled with granular material. The minute vacuoles of the opaque layer are densely packed and cease abruptly towards the clear cortex, but the deep aspect of the layer passes without any sharp boundary into the nucleus.

Fig. 7. Schirmer's section of multiple lamellar cataract removed by operation. b_1 minute opaque lamella, b_2 second lamella, with *a* clear interior, b_3 third lamella, with *d* clear interior, marked at *e* by fine incomplete lamella, *c* outer cortex. Both lenses were extracted from the eyes of a girl aet. 14. Seven years previously optical iridectomies had been performed on each eye; at that time only the central opaque layer was recorded in the notes, the author thinks it certain that the outer layer would have been detected if it had been present then, for iridectomies would not have been made unless the cortex were clear at that date; he considers the outer layer formed at about age eight, i.e. some little time after the iridectomies. No history of fits, or delayed walking, no note on teeth. The author figures sections of both lenses, that reproduced is the section of the left lens. Before extraction the lens appeared small, its margin well within the bounds of the cornea, and the fibres of the lens ligament were plainly seen. The double layers of opacity were easily seen where the iris had been removed. The inner layer was small and opaque; the outer layer and its border more or less notched, it transmitted very little mirror-light. There was a narrow zone of clear cortex superficial to the outer layer of opacity. There was a yellowish-white pyramidal opacity with its apex projecting above the anterior capsule. In section. The nucleus (*a*) looks clear except for a small delicate layer of opacity close around its centre (b_1). Microscopically both nucleus and the apparently clear layers (*d*) between the two opaque lamellae (b_2 and b_3) show a good many inter-fibrillary spaces. There are also numerous very small "droplets" which are the chief cause of the opacity of the opaque layers; and the lens-fibres in the opaque zones are often swollen or show knobs and projections. The two principal opaque lamellae vary from 0·3 to 0·45 mm. in thickness. Between the two principal opaque lamellae is a third called by the author a "second intermediary zone" (*e*) only 0·05 mm. thick, therefore much thinner than the principal lamellae and incomplete or less marked at the anterior and posterior surfaces of the lens. This layer, which clinically would have constituted a "rider," contained droplets, fine granular material and broken-looking ends of lens fibres. Figs. 6 and 7 are reproduced from the *Archiv für Ophthalmologie*, 1889, Taf. X. Figs. 1 and 3, S. 147.

(*Figs.* 8 *to* 34. *Drawings of lenses* in situ *showing various forms of cataract, with accompanying diagrammatic sections of such lenses.* N.B. *a cataract may be viewed two ways:* (1) *by focal illumination when a strong light is focussed on its surface, then it appears as a whitish opacity against the black background of the pupil or the unilluminated interior of the eye;* (2) *by reflected light, when light is projected into the eye by a mirror and returned as a red reflex from the illuminated fundus to the eye of the observer; in such circumstances the cataract looks dark or black against the bright red background. Compare Figs.* 9 *and* 10, *drawings of the same cataract seen under these different conditions.*)

Fig. 8. Small dense lamellar cataract, no riders or outlying opacities, focal illumination × 3·5. Drawn by Harman for this work; see his Case, Fig. 307. Turner family. Mrs H—— (III. 15), aet. 35; gets fair vision through optical iridectomies; teeth good; mother of seven of whom four cataractous, see Fig. 14.

Fig. 9. Fair sized lamellar cataract seen by focal illumination with several riders and small outlying masses, × 3·5. Drawn by Harman for this work. Male, aet. 15. Teeth irregular, small, very defective enamel. Generally undersized physique, limb bones distorted by rickets. No history of fits. Opacity including riders measured 6·5 to 7 mm.

Fig. 10. Drawing of same cataract, shown in Fig. 9, as seen against the illuminated fundus. Iris cut away at *i* to show clear cortex *c* and lens ligament *ll*, *o* = main opacity, *r* = rider. Drawn by Harman for this work (for more striking riders see Fig. 16, Plate M).

Fig. 11. Diagrammatic section of lens as in Fig. 10. *l* = lamellar opacity, *r* = rider, *c* = clear cortex, *n* = clear nucleus (Harman).

Fig. 12. Double-shelled lamellar cataract seen by focal illumination, × 3·5. Drawn by Harman for this work from sketch supplied by Professor McHardy. Case, Fig. 288. (IV. 1—6.) Vision bad after removal of cataracts. Teeth excellent.

Fig. 13. Diagrammatic section of double-shelled lamellar cataract as in Fig. 12. *c* = clear cortex, *n* = clear nucleus, *l'* = inner layer of opacity, *l''* = outer layer of opacity. Drawn by Harman for this work.

Fig. 14. Very delicate lamellar cataract with dense central star in anterior layer; focal illumination, × 3·5. Drawn by Harman for this work. Case, Fig. 307, IV. 28. Male, aet. 3, child of mother whose cataract shown in Fig. 8.

Fig. 15. Section of lens as in Fig. 14. *s* = anterior star, *l* = lamellar opacity, *c* = clear cortex, *n* = clear nucleus.

PLATE M. Fig. 16. Lamellar cataract of moderate size with exceedingly distinct rider and looped spokes. The "rider" (occupying the site of X on the clock face) straddled the main opacity, its pointed central ends nearly reaching the poles, but it was separated from the main globular opacity by a broad clear layer of lens substance. Similarly the "spokes" were looped around and well clear of the main opacity in remarkably distinct lines.

The right eye of a female aet. 33, the cataract was diagnosed by Hulke when she was aet. 10. The left eye has similar cataract, only the large rider occupies the site of III. on the clock face. Vision when pupils are dilated very fair ($\frac{6}{18}$), she used a midriatic habitually. Teeth excellent. Drawn by Harman for this work.

Fig. 17. Discoid cataract, usual simple form; opacity appears dark against illuminated fundus, × 3·5. Nettleship and Ogilvie's Case (Fig. 345, V. 9).

Fig. 18. Discoid cataract showing trilobed denser portion of disc, lighted as in Fig. 16, × 3·5. Nettleship and Ogilvie's Case (Fig. 345, VI. 38).

(Figs. 17 and 18 are reproduced by kind permission from *Trans. Opthal. Soc.*, Vol. XXVI., 1906, Plate VIII.)

Fig. 19. Diagrammatic section of lens as in Figs. 16 and 17. *d* = discoid opacity, *n* = nucleus, *c* = clear cortex. Drawn by Harman for this work.

Figs. 20—26. Showing progression of form and size of opacity from the simplest discoid cataract (Fig. 20) to lamellar cataract (Figs. 24—25). In each case the opacity appears dark against the illuminated fundus. In Fig. 25 the iris is cut away below to show refraction stria, × 1·3. Case 342. Nettleship's pedigree of S.—— family. Fig. 21, IV. 104; 22, 23 right and left of IV. 70; 24, 25 right and left of V. 82; 26, IV. 102. Redrawn by Harman for this work from portions of Nettleship's original plate.

Fig. 27. Coralliform cataract, seen by focal illumination, × 2·75. Reproduced from a drawing kindly provided by Holmes Spicer. Nettleship's Case, Betts family, No. 318, IV. 2. The case illustrated by the figure was published by Marcus Gunn, but the drawing here reproduced is one that Holmes Spicer considers more accurate than that of his originally published. For description see page 127 of text. N.B. no diagrammatic section of this cataract is given since it is not certain whether the trumpet-shaped opacities extend into the posterior part of the lens as well as forwards and into the equator.

Fig. 28. Müller's diagrammatic section of lens with *ab* axial opacity, *c* complete lamellar opacity, *e* incomplete anterior lamella, *dd* points of thickening of axial opacity about main lamella. Case No. 325. Reproduced from *Archiv für Ophthalmologie*, 1855, S. 169.

Fig. 29. Stellate cataract, Y anterior and ⚹ posterior opacities; focal illumination from left-hand side and looked at from right-hand side, × 3·5. Drawn by Harman for this work from the separate line drawings of the anterior and posterior markings published by Adams. Case Fig. 308 (V. 3).

Fig. 30. Microphthalmia and anterior polar cataract, focal illumination × 3·5. The smallness of the cornea can be judged by comparison with Figs. 16, 17 and 18, all four of which are on the same scale. Corneal diameter measured 7·5 mm. (average 11·6 mm.). Drawn by Harman for this work. Case, Fig. 339 (III. 8).

Fig. 31. Diagrammatic section of lens as in Fig. 29; the anterior polar opacity is seen to dip into the lens; the dotted radiations indicate "refraction striae," seen when the living lens was examined by reflected light. Drawn by Harman for this work.

Fig. 32. Posterior polar or "hyaloid" cataract, with small anterior polar opacity; focal illumination from left; viewed from right side, × 3·5. The posterior part of the lens is occupied by dense white opacity fringed on its anterior aspect, it is united by a fine axial stalk to a dense white button-shaped opacity situated in or just beneath the capsule. The front of this button had an elevation which bulged forwards into the contracted pupil. N.B. only one of the ten affected in this family had this anterior polar opacity, all ten had the posterior larger opacity. Drawn by Harman for this work. Case, Fig. 341 (IV. 12).

Fig. 33. Diagrammatic section of lens as in Fig. 31. *a* = anterior polar cataract, dipping down to meet *p* posterior polar mass, *h* = hyaloid vessels probably present, *c* = clear cortex. Drawn by Harman for this work.

Fig. 34. Semi-diagrammatic section of eye with ill-developed lens, posterior polar cataract and persistent hyaloid artery. Projecting from the lower margin of the pupil into the pupillary area is a tag of persistent 'pupillary membrane.' Reproduced from a block kindly lent by Treacher Collins. (Bibl. No. 21.)

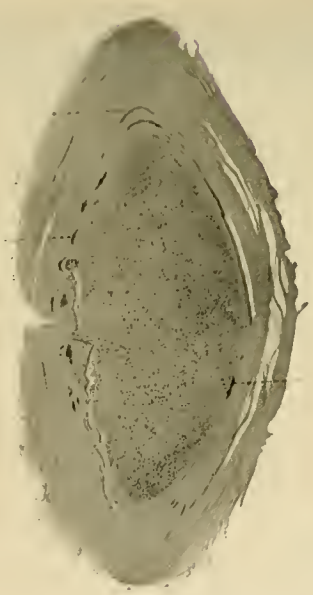


FIG. 1. Section of lamellar cataract removed by operation. ×8. (Lawford.)

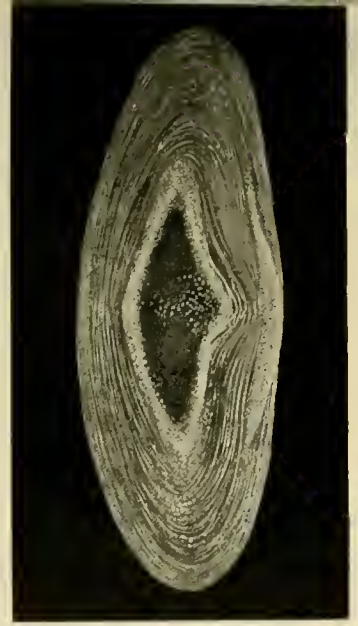


FIG. 2. Section of lens with lamellar cataract. ×8. (Hess.)

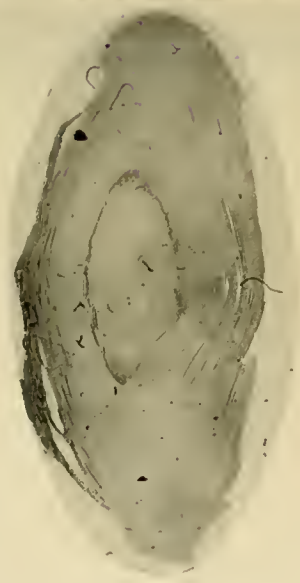


FIG. 3. Section of lamellar cataract, hereditary case. ×8. (Norris.)

FIG. 4. Section of lens with very small double perinuclear lamellar cataract. ×4. (Deutschmann.)

FIG. 5. Lamellar cataract photographed in water, immediately after extraction. (Parsons.)

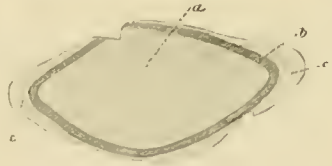


FIG. 6.

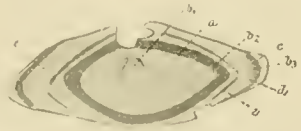


FIG. 7.

FIGS. 6, 7. Sections of single and multiple lamellar cataract. ×4. (Schirmer.)

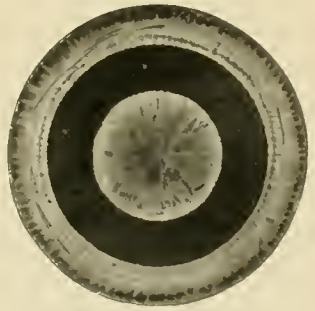


FIG. 8. Small dense lamellar cataract. ×3·5. (Harman.) Case 307.

FIG. 9. Fair sized lamellar cataract. ×3·5. (Harman.)

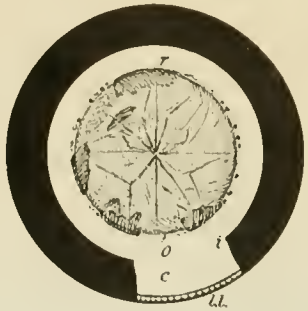


FIG. 10. Lamellar cataract, same as Fig. 9, seen against illuminated fundus. ×3·5. (Harman.)

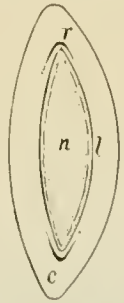


FIG. 11. Diagrammatic section of lens as in Fig. 8. ×4. (Harman.)

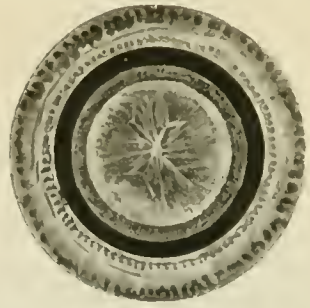


FIG. 12. Double shelled lamellar cataract. ×3·5. (Harman.) Case 288.

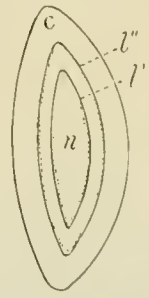


FIG. 13. Diagrammatic section of cataract as in Fig. 12. ×4. (Harman.)

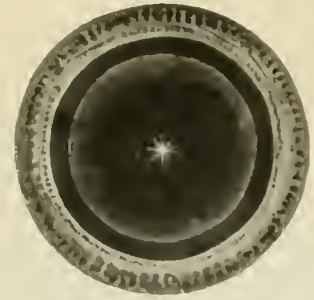


FIG. 14. Delicate lamellar cataract with dense central star. ×3·5. (Harman.) Case 307.

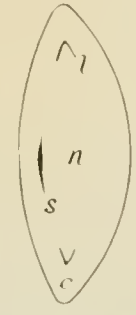


FIG. 15. Diagrammatic section of lens as in Fig. 14. ×4. (Harman.)

In sectional drawings the anterior surface is to the left (except Figs. 6, 7). It is essential to examine this plate with the fuller descriptions given in the text.

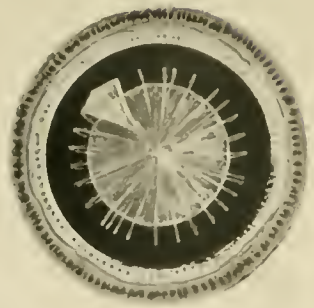


FIG. 16. Lamellar cataract with well marked rider and looped spokes. ×3·5. (Harman.)

FIG. 17. Discoid cataract, usual simple form. ×3·5. (Nettleship and Ogilvie.) Case 345.

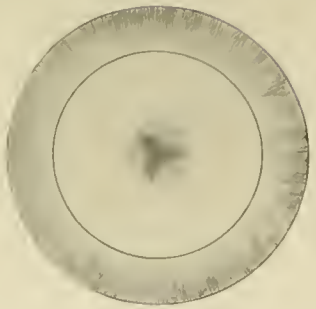


FIG. 18. Discoid cataract showing trilobed denser portion. ×3·5. (Nettleship and Ogilvie.) Case 345.

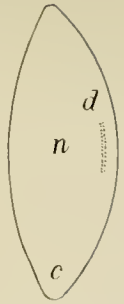
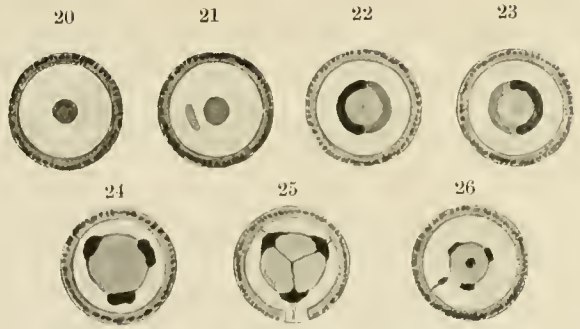


FIG. 19. Diagrammatic section of lens as in Figs. 17 and 18. ×4. (Harman.)



FIGS. 20—26. Progression of form and size of opacity from simple discoid (20) to lamellar cataract (24—25). ×1·3. (Nettleship.) Case 342.

FIG. 27. Coralliform cataract. ×2·75. (Holmes Spicer.) Case 318.

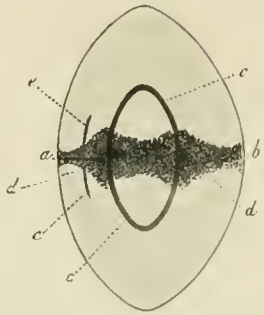


FIG. 28. Diagrammatic section of lens with lamellar and axial opacities. (Müller.) ×4·5. Case 325.

FIG. 29. Stellate cataract. ×3·5. (Harman.) Adams's Case 308.

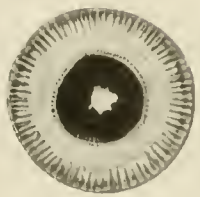


FIG. 30. Microphthalmia and anterior polar cataract. ×3·5. (Harman.) Case 339.

FIG. 31. Diagrammatic section of lens as in Fig. 30. ×4. (Harman.)

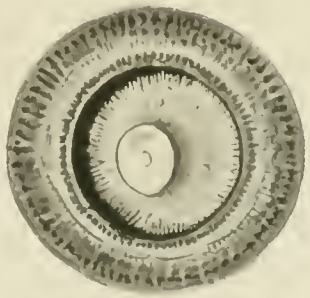


FIG. 32. Posterior polar or 'hyaloid' cataract. ×3·5. (Harman.) Case 311.

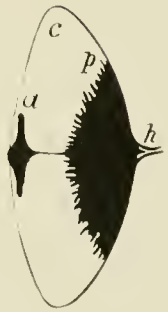


FIG. 33. Diagrammatic section of lens as in Fig. 32. ×4. (Harman.)

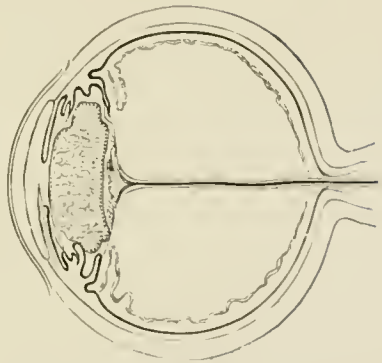
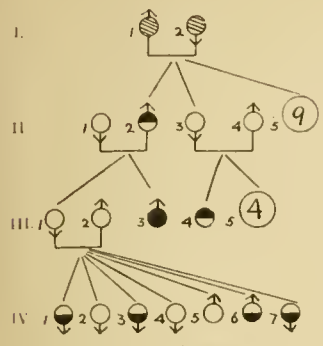


FIG. 34. Semi-diagrammatic section of eye with ill-developed lens, posterior polar cataract, and hyaloid artery. ×2. (Treacher Collins.)

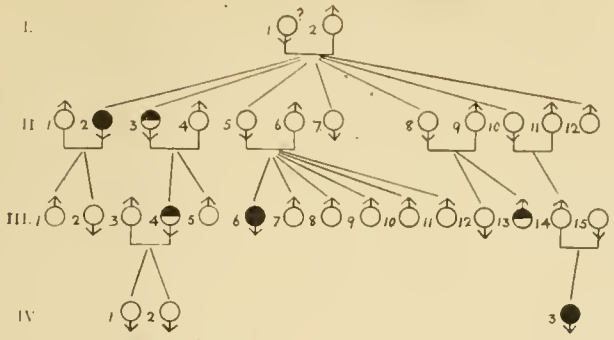
In sectional drawings the anterior surface looks to the left. It is essential to examine this plate with the fuller descriptions given in the text.

FIG. 194



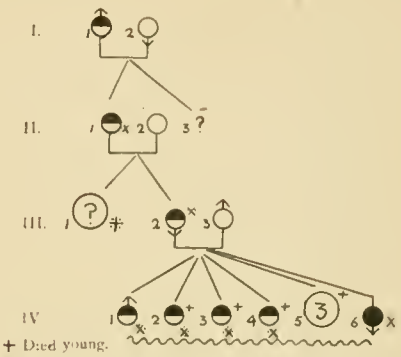
Guthrie Cayley's Case.

FIG. 195



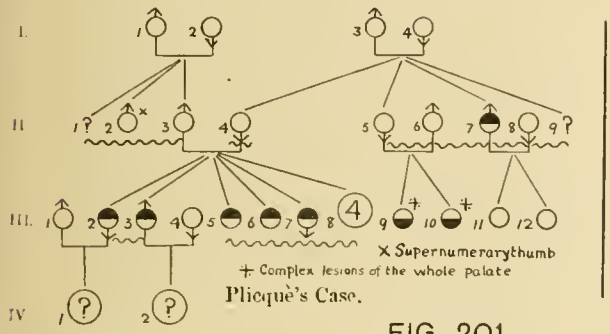
Passavant's Case.

FIG. 196



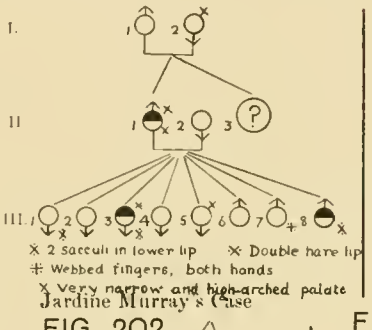
+ Died young.
‡ Many had same deformity and died young
※ Double hare lip + Sacculi
Demarquay's Case (i).

FIG. 197



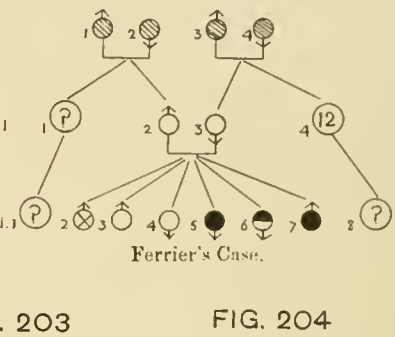
※ Supernumerarythumb
‡ Complex lesions of the whole palate
Plicque's Case.

FIG. 198



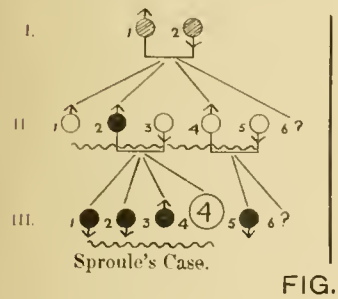
※ 2 sacculi in lower lip ※ Double hare lip
‡ Webbed fingers, both hands
× Very narrow and high-arched palate
Jardine Murray's Case

FIG. 199



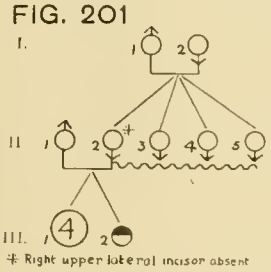
Ferrier's Case.

FIG. 200



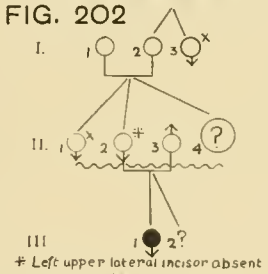
Sproule's Case.

FIG. 201



‡ Right upper lateral incisor absent
(i).

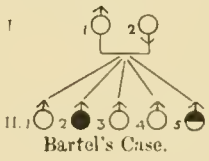
FIG. 202



‡ Left upper lateral incisor absent
× Died of Phthisis
(ii).

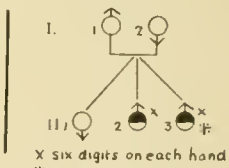
Clement Lucas' Cases.

FIG. 203



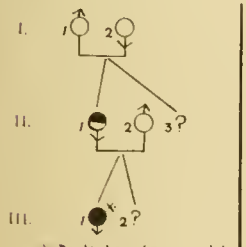
Bartel's Case.

FIG. 204



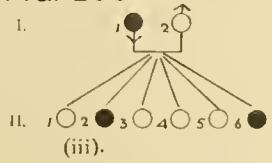
× Six digits on each hand
‡ Other anomalies
Sutherland and Jackson's Case.

FIG. 205



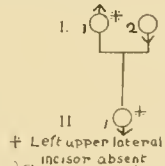
※ Double hare lip + complete division of palate
Demarquay's Case (ii)

FIG. 206



(iii).

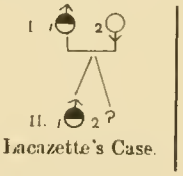
FIG. 207



‡ Left upper lateral incisor absent
(iv).

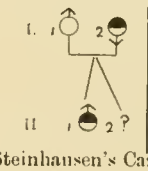
Clement Lucas' Cases.

FIG. 208



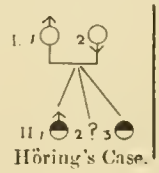
Lacazette's Case.

FIG. 209



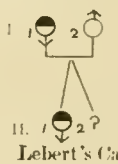
Steinhausen's Case.

FIG. 210



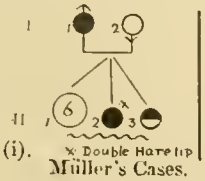
Höring's Case.

FIG. 211



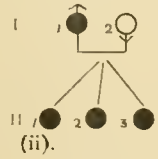
Lebert's Case.

FIG. 212



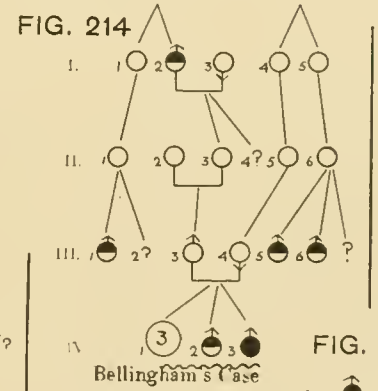
(i). ※ Double Hare lip
Müller's Cases.

FIG. 213



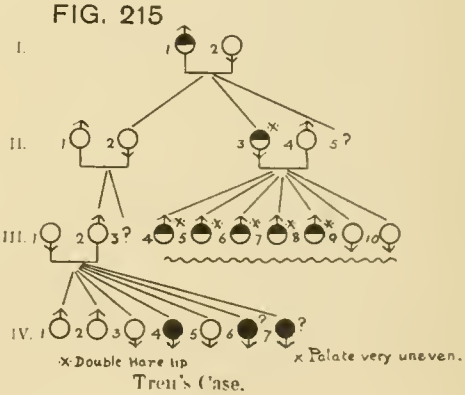
(ii).

FIG. 214



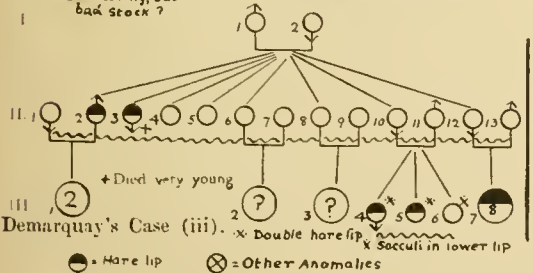
Bellingham's Case

FIG. 215



※ Double Hare lip
× Palate very uneven.
Trew's Case.

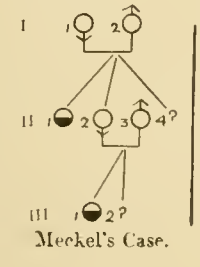
FIG. 216



‡ Healthy, but bad Stock ?
+ Died very young
※ Double hare lip
× Sacculi in lower lip
Demarquay's Case (iii).

● = Hare lip
◒ = Cleft palate
● = Harelip + Cleft palate
⊗ = Other Anomalies

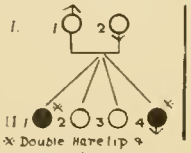
FIG. 217



Meckel's Case.

No Consanguinity recorded in the above pedigrees

FIG. 218

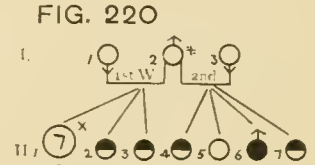


※ Double Hare lip + double cleft palate
Houston's Case.

FIG. 219

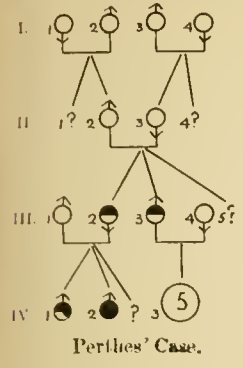
Bramann's Case.

FIG. 220



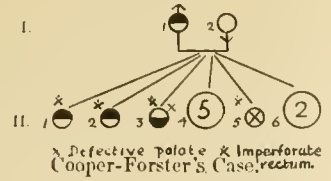
× Still born
‡ Had two relations with harelip.
Anna's Case.

FIG. 221



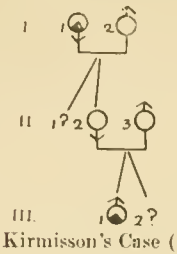
Perthes' Case.

FIG. 222



Cooper-Forster's Case.

FIG. 223

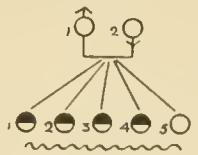


Kirmisson's Case (i).

FIG. 224

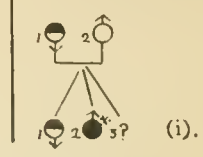
Kirmisson's Case (ii).

FIG. 225



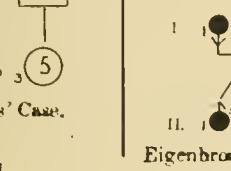
J. Lucas's Case.

FIG. 226



(i). Eigenbrodt's Case.

FIG. 227

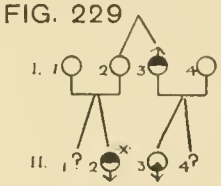


(ii). Eigenbrodt's Case.

FIG. 228

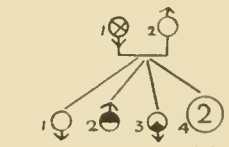
Maurel's Case.

FIG. 229



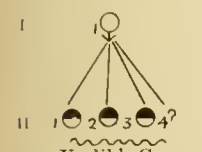
Rennert's Case (i).

FIG. 230



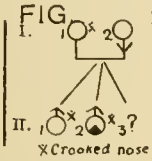
Rennert's Case (ii).

FIG. 231



Vrolik's Case.

FIG. 232



Mauclaire's Case.

Trendelenburg's Case

FIG. 233

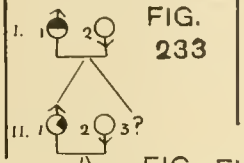
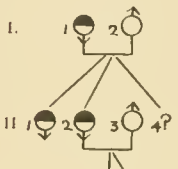
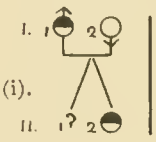


FIG. 234



Hardurcke's Case.

FIG. 235

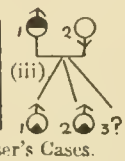


(i).

FIG. 236

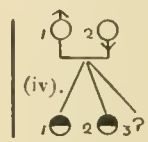
(ii).

FIG. 237



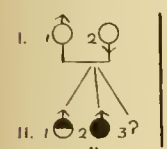
(iii).

FIG. 238



(iv). Stobwasser's Cases.

FIG. 239



(i).

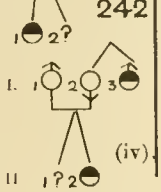
FIG. 240

(ii).

FIG. 241

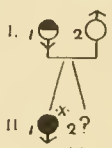
(iii). Gotthelf's Cases.

FIG. 242



(iv).

FIG. 243

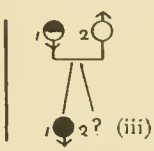


(i).

FIG. 244

(ii).

FIG. 245

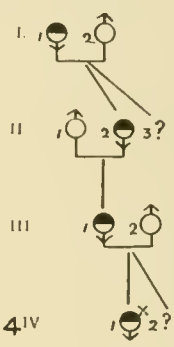


(iii). Fronhofer's Cases.

FIG. 246

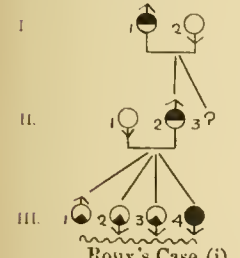
(iv).

FIG. 247



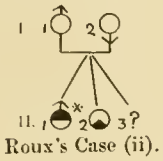
(v).

FIG. 248



Roux's Case (i).

FIG. 249

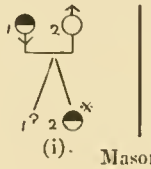


Roux's Case (ii).

FIG. 250

Roux's Case (iii).

FIG. 251



(i).

FIG. 252

(ii).

FIG. 253

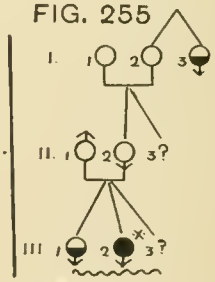
(iii). Mason's Cases.

FIG. 254

Mason's Case (iv).

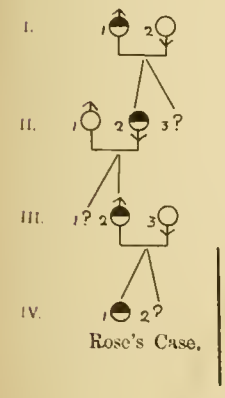
× cleft alveolus

FIG. 255



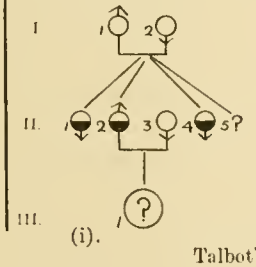
Mason's Case (v).

FIG. 256



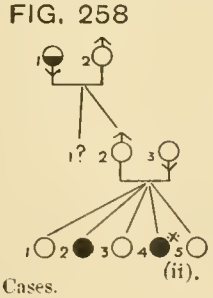
Rose's Case.

FIG. 257



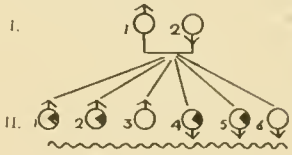
(i).

FIG. 258



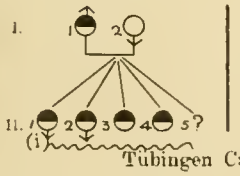
(ii). Talbot's Cases.

FIG. 259



Mason's Case (vi).

FIG. 260



(i).

FIG. 261

(ii).

FIG. 262

(iii). Tübingen Cases.

FIG. 263

Haug's Case (i).

FIG. 264

Haug's Case (ii).

FIG. 265

Tübingen Case (iv).

FIG. 266

Tübingen Case (v).

FIG. 267

Haug's Case (iii).

◓ = Hare Lip ◔ = Fissured uvula

◒ = Cleft Palate ⊗ = Other Anomalies

● = Hare Lip and Cleft Palate

◕ = Intrauterine healed hare lip

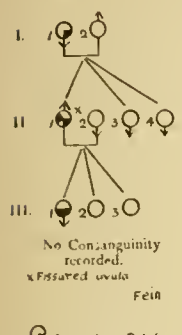
No Consanguinity recorded in the above Pedigrees

× = Double Hare Lip.

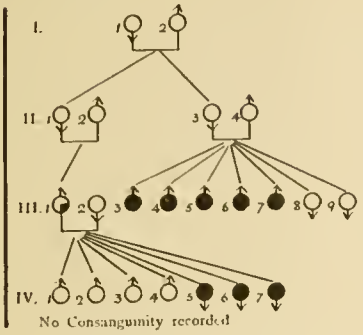
Cleft Palate and Hare Lip.

Deaf Mutism.

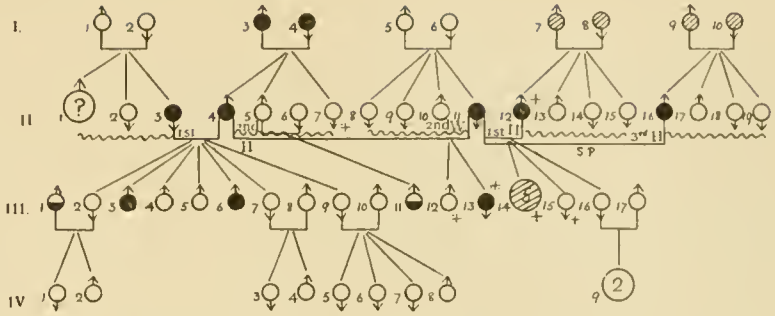
268



269

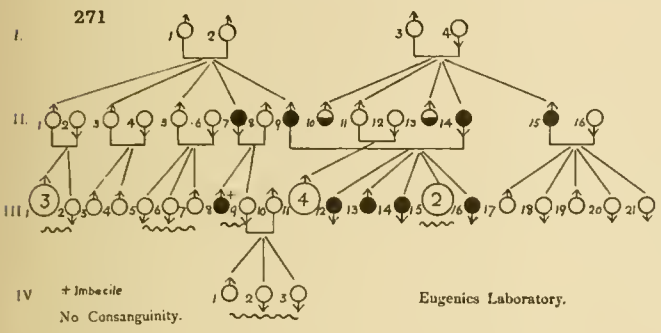


270



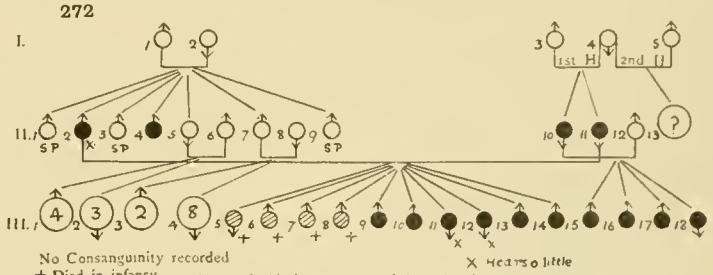
No Consanguinity
+ Died in infancy
+ Died young.
+ Said to be non-congenital
Eugenics Laboratory.

271



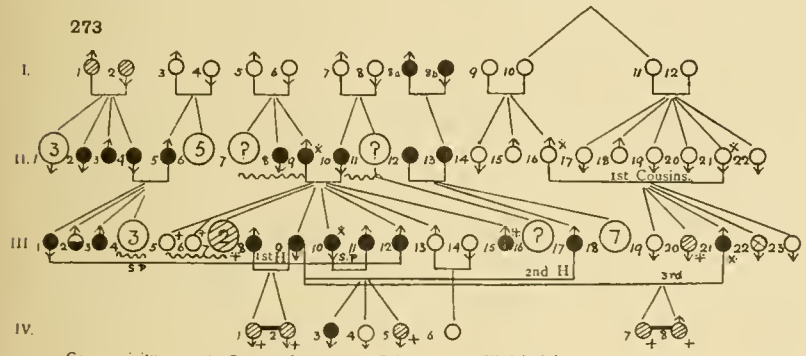
+ Imbecile
No Consanguinity.
Eugenics Laboratory.

272



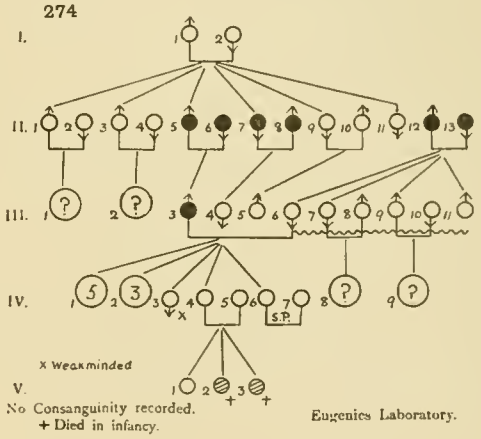
No Consanguinity recorded
+ Died in infancy
× Nearly blind some years before death
× Hears a little
Eugenics Laboratory.

273



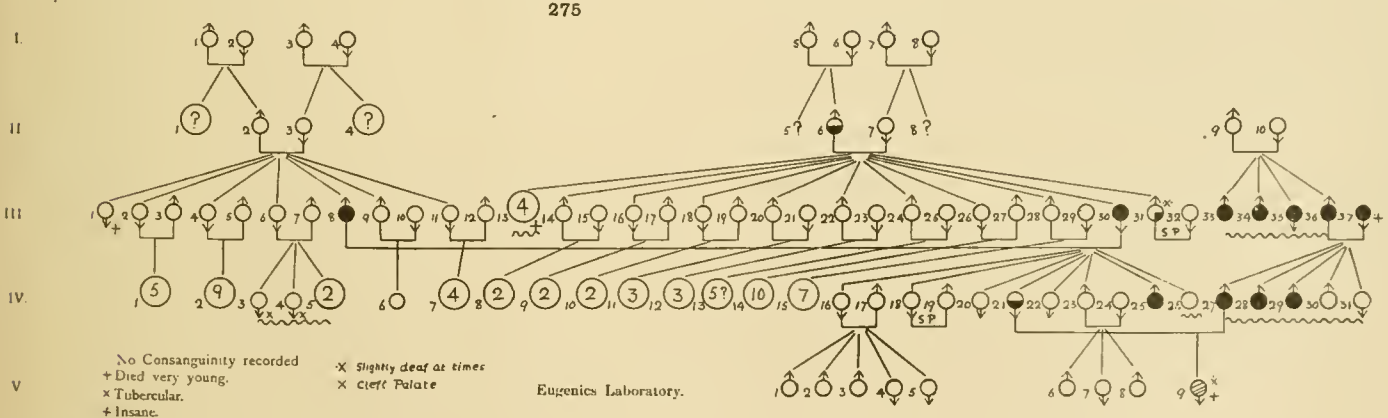
Consanguinity. and No Consanguinity. + Still-born. + Died in infancy. × a "natural"
× Alcoholic. + Imbecile + Died young.
Eugenics Laboratory

274

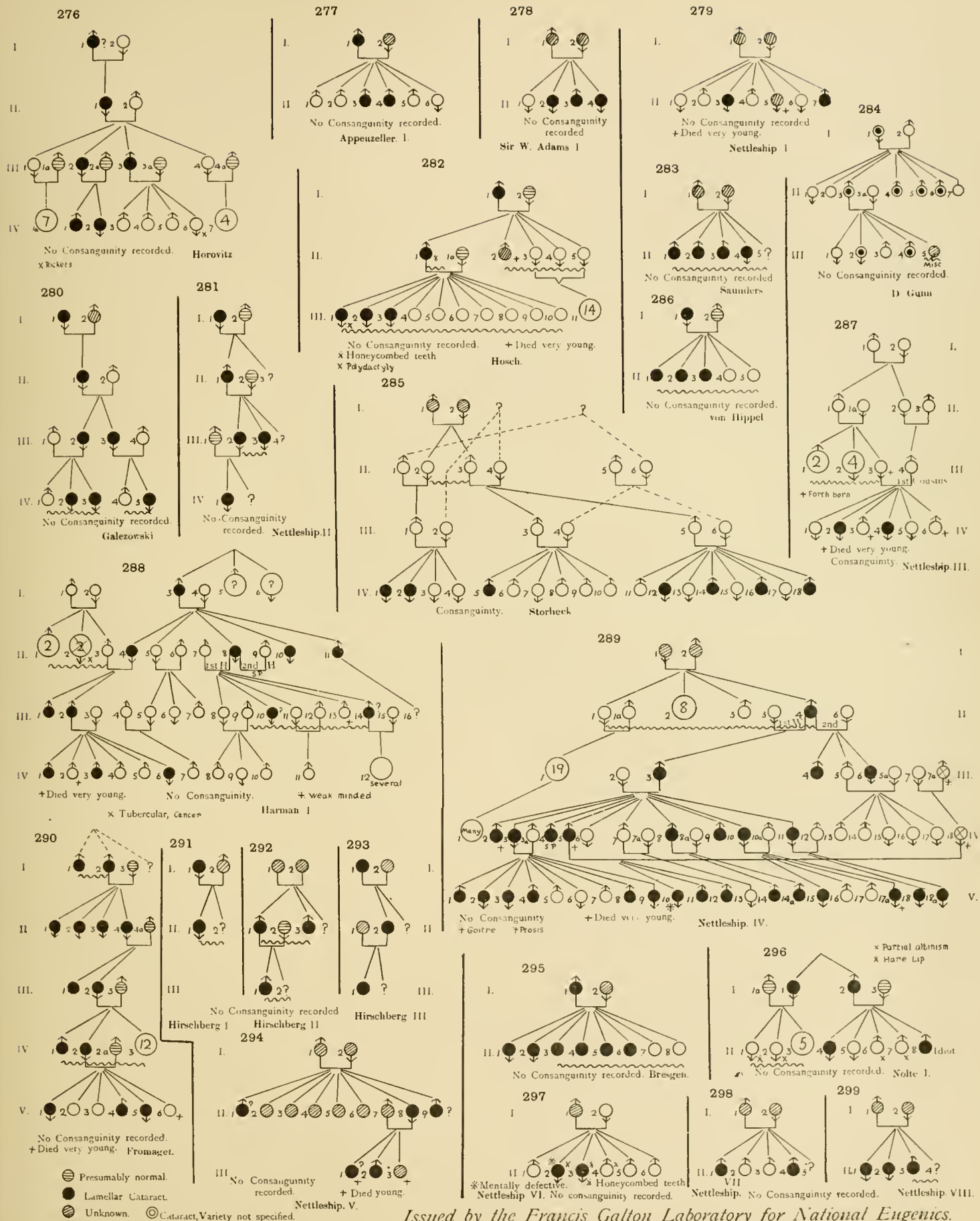


No Consanguinity recorded.
+ Died in infancy.
Eugenics Laboratory.

275

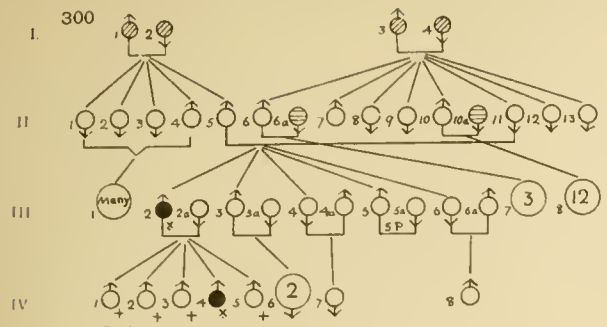


No Consanguinity recorded
+ Died very young.
× Tubercular.
+ Insane.
× Slightly deaf at times
× Cleft Palate
Eugenics Laboratory.



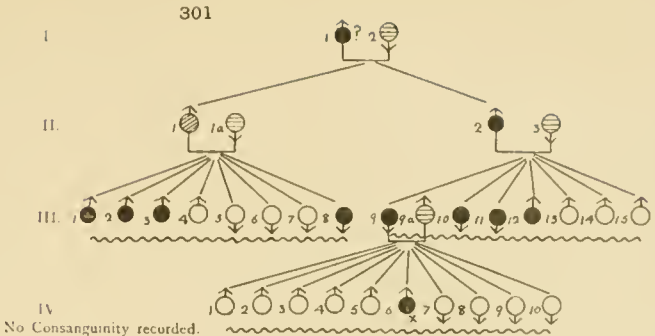
Issued by the Francis Galton Laboratory for National Eugenics.

300



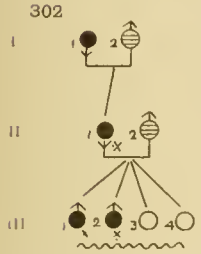
+Died very young.
No Consanguinity recorded. ⁂ Honeycombed teeth, fits.
Harman II.

301



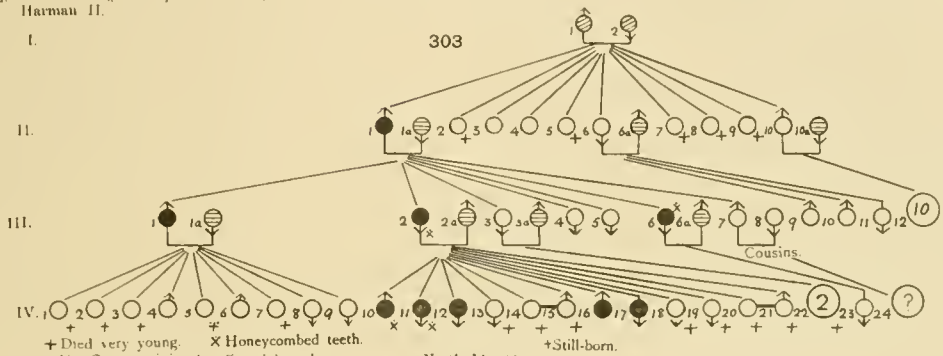
No Consanguinity recorded.
⁂Honeycombed teeth.
Grimsdale.

302



No Consanguinity recorded.
⁂Honeycombed teeth.
⁂ Rickets Kunn.

303



+Died very young. ⁂Honeycombed teeth.
No Consanguinity in affected branches
Nettleship IX.
+Still-born.

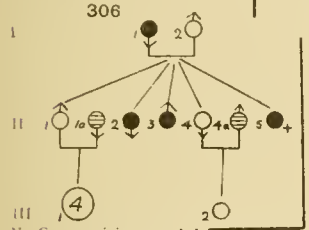
304

Nettleship X.

305

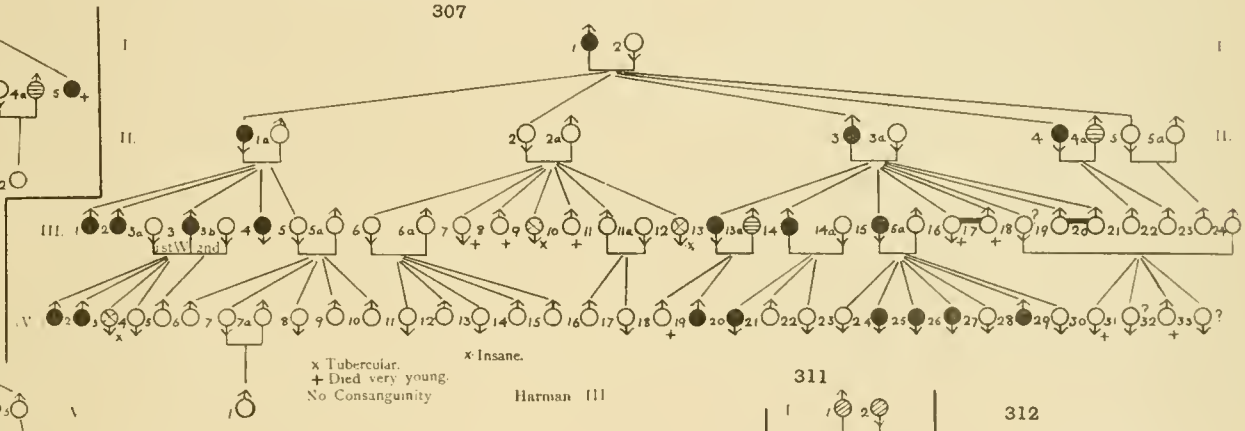
Davidsen.

306



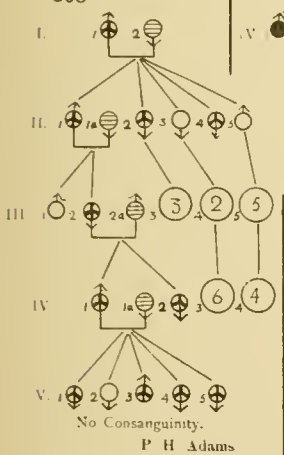
No Consanguinity recorded.
+Died very young.
Nolte II.

307



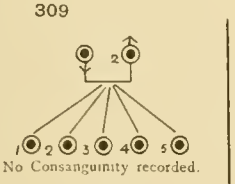
⁂ Tubercular.
+ Died very young.
No Consanguinity
⁂ Insane.
Harman III

308



No Consanguinity.
P H Adams

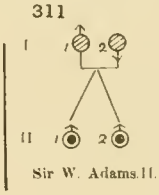
309



No Consanguinity recorded.
Minor.

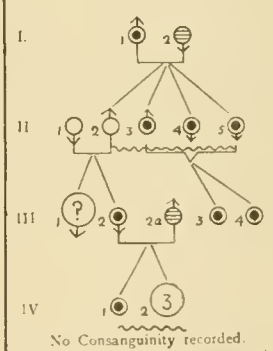
310

311



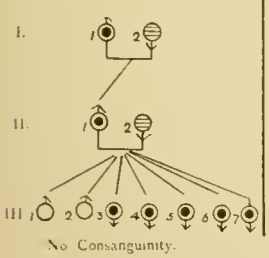
Sir W. Adams II.

312



No Consanguinity recorded.
Maunoir

313



No Consanguinity.
Sir W. Adams III

Zirm & Bergmeister.
No Consanguinity recorded.
+ Said to be "short-sighted"
† Myopic with Cataract.
⁂ Myopic
⁂ Senile Cataract.
Unknown.
Presumably normal.
Lamellar Cataract.
Coralliform Cataract.
Stellate Cataract.
Variety not specified.

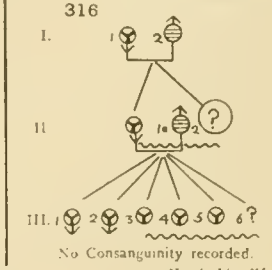
314

von Arx I

315

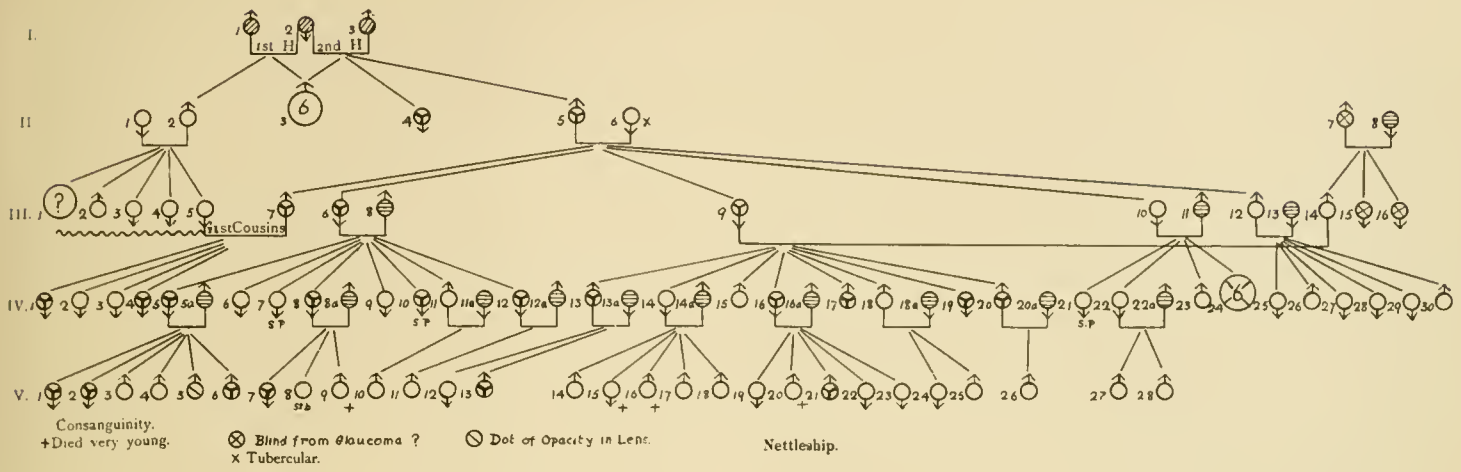
von Arx II.

316

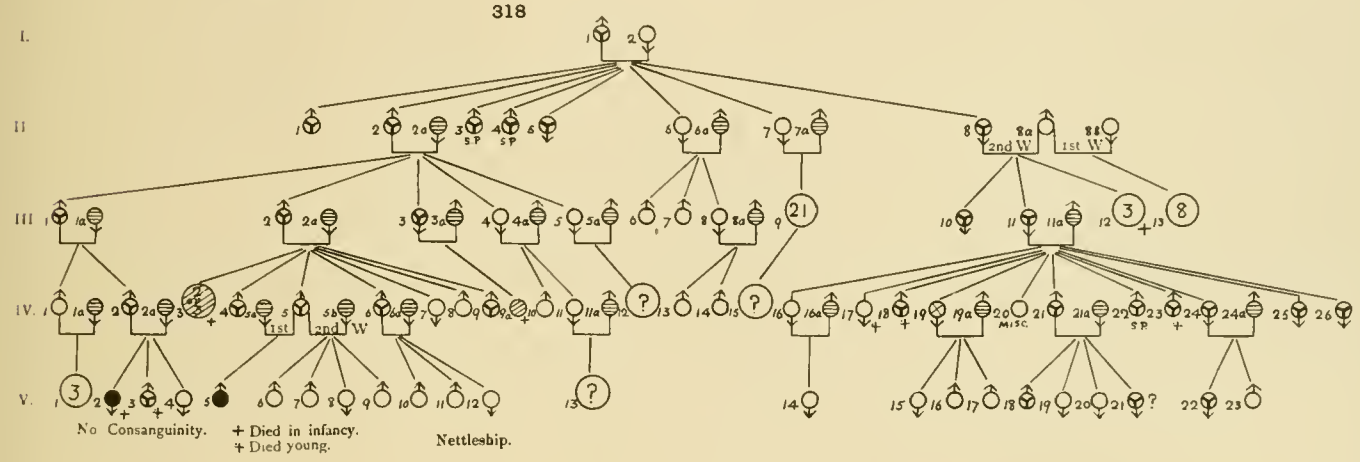


No Consanguinity recorded.
Nettleship XI.

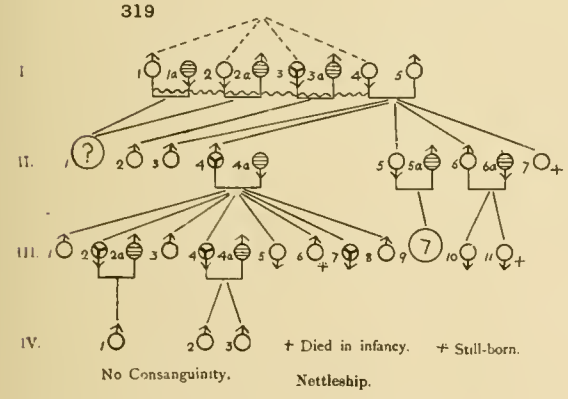
317



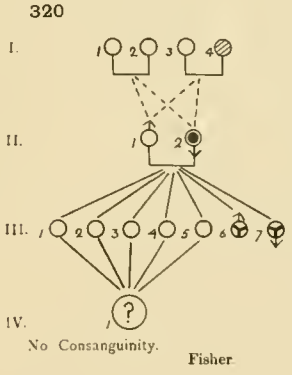
318



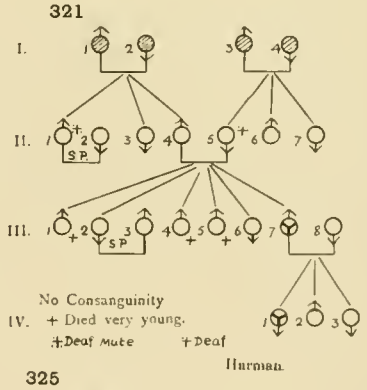
319



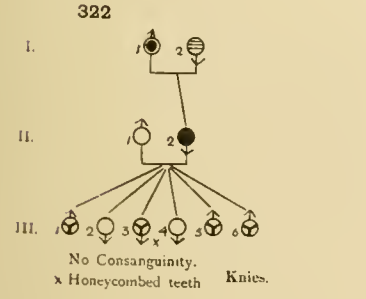
320



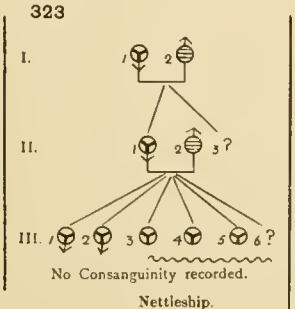
321



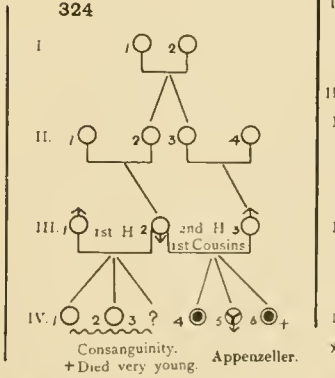
322



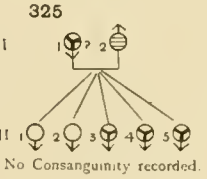
323



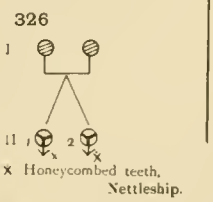
324



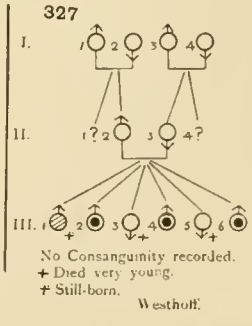
325

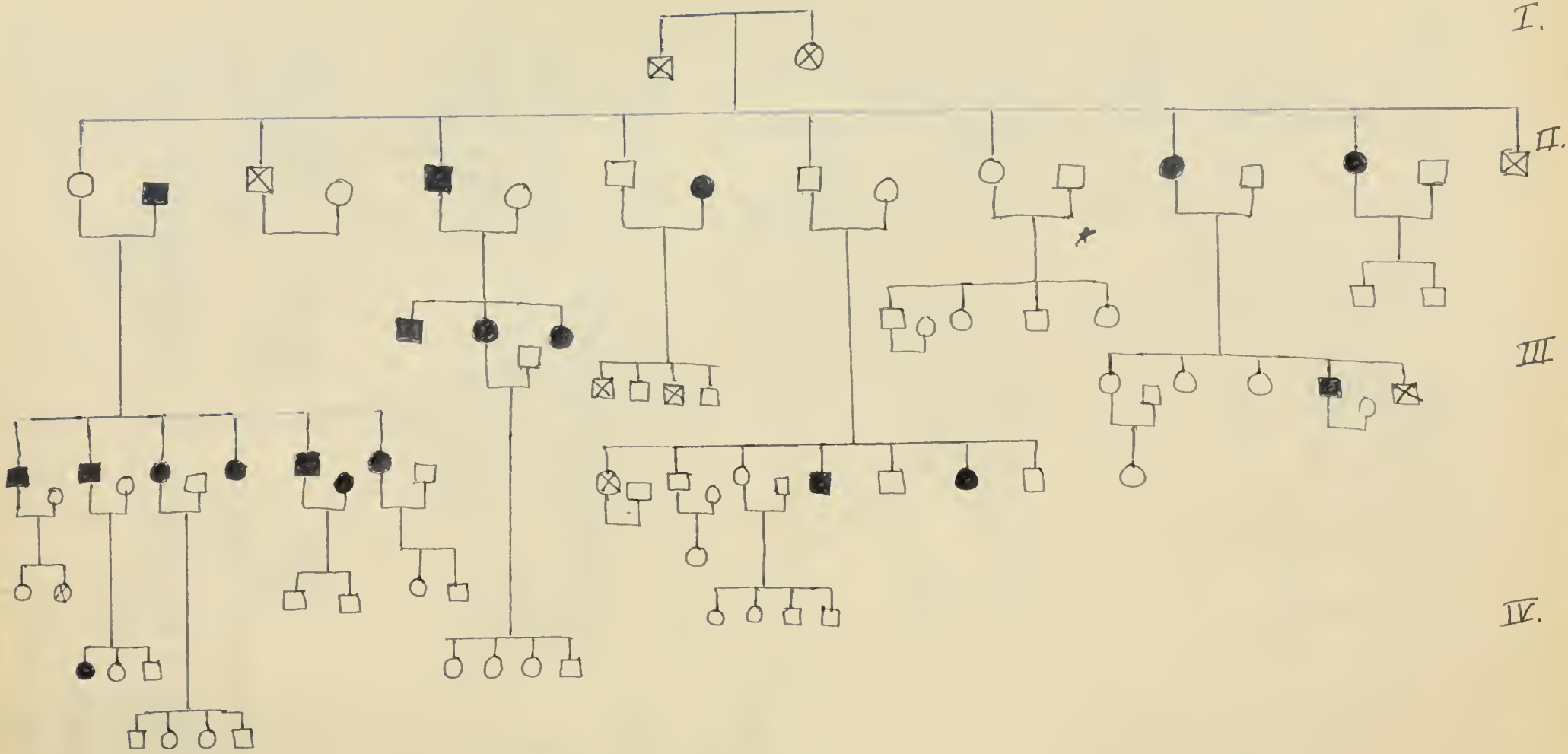


326



327



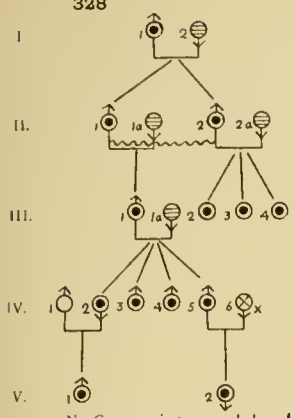


APPENDICITIS (occurances)

I. Grandparents
II. Parental generation (* my family)
III. My generation
IV. Young generation. The children in IV are still young thus no appendicitis cases have yet occured.

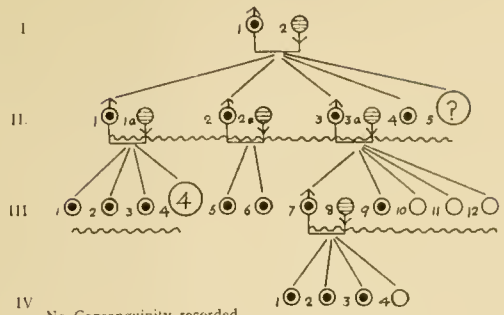
□ MALES
○ FEMALES
■ } OPERATED ON
● } FOR APPENDICITIS
⊠ } DEAD AT TIME
⊗ } CHART WAS MADE

328



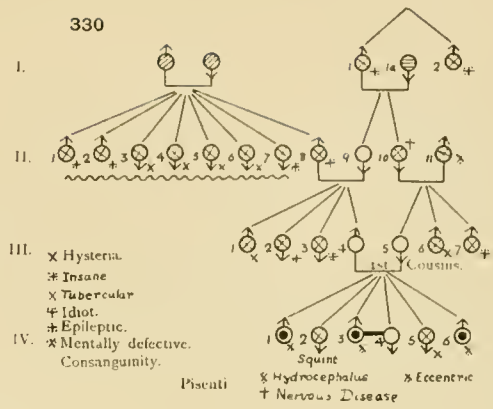
No Consanguinity recorded.
x Blind. C Loeb.

329

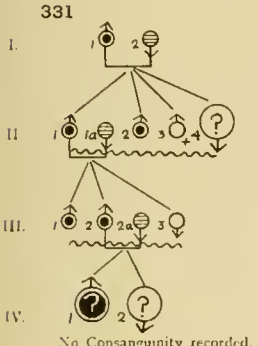


No Consanguinity recorded.
A D Williams

330

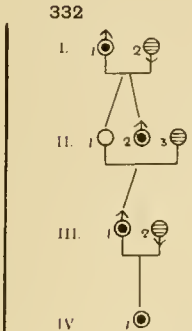


331



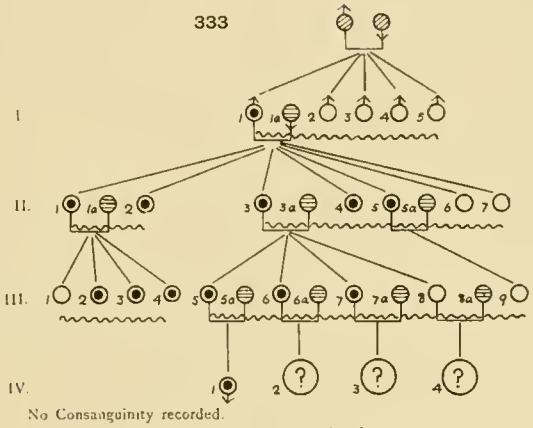
No Consanguinity recorded.
+ Died very young.
Dyer.

332



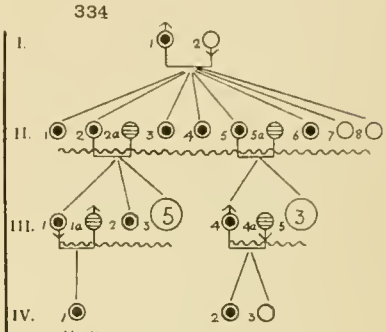
No Consanguinity recorded.
Gerok

333



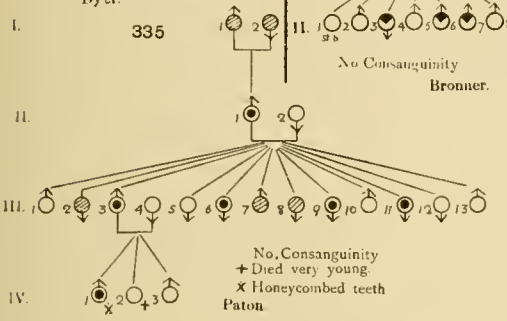
No Consanguinity recorded.
Baudon

334

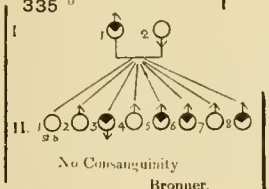


No Consanguinity recorded.
Armaignac.

335

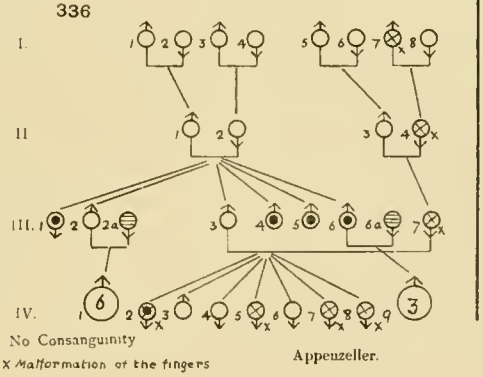


335 b



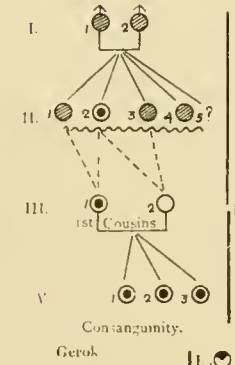
No Consanguinity
Bronner.

336



No Consanguinity
x Malformation of the fingers
Appenzeller.

337



Consanguinity.
Gerok

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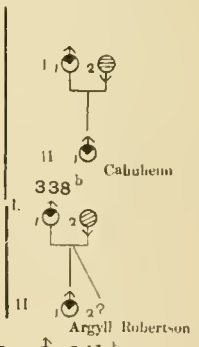

Cahuleim

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Argyll Robertson

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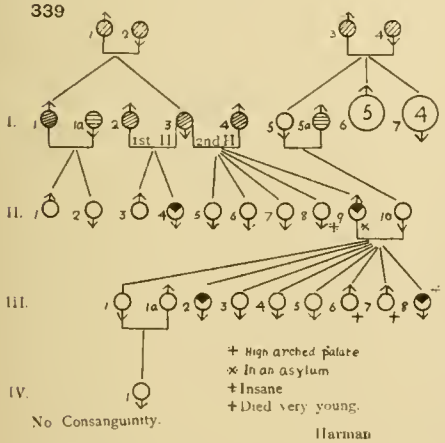

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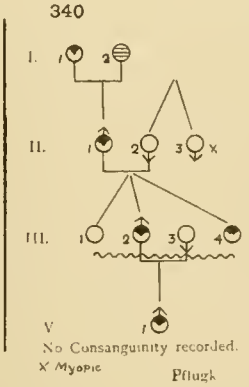

No Consanguinity recorded.
x Myopic
Pflugk

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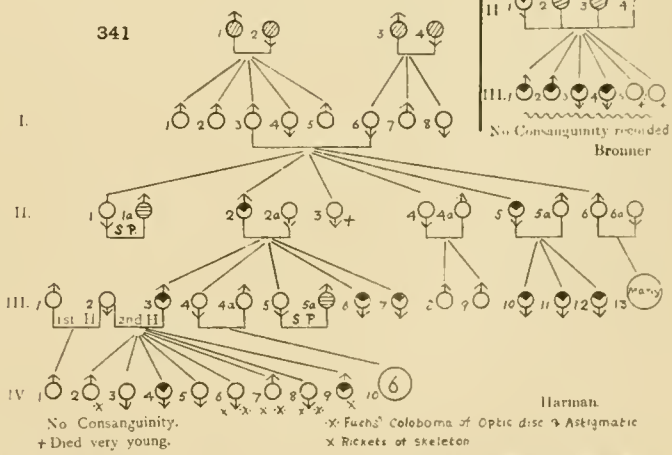

341 b

No Consanguinity recorded
Bronner

● Cataract Variety not specified.
◒ Anterior and Posterior Polar Cataract.
⊖ Presumably normal.

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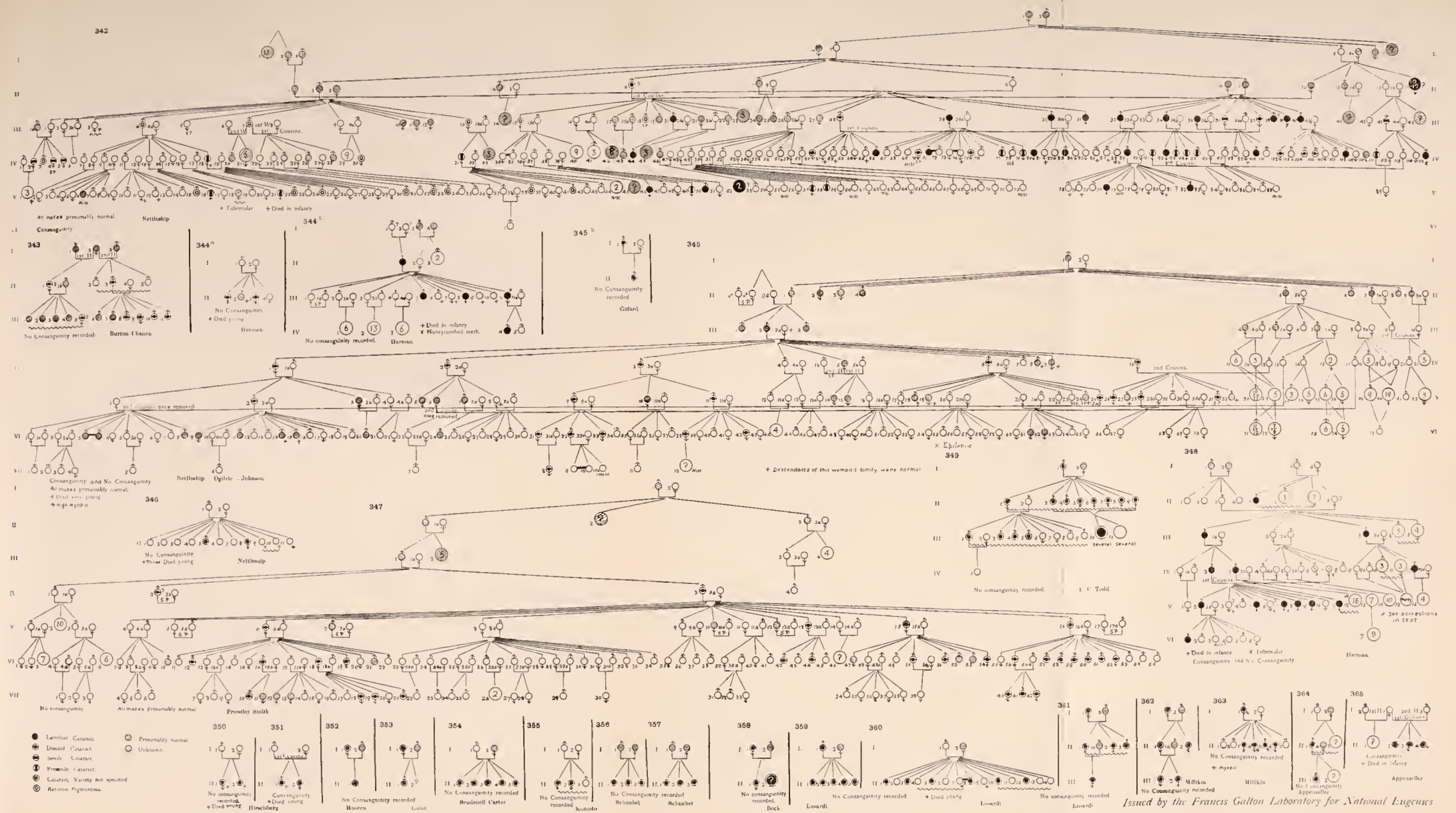
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362
363
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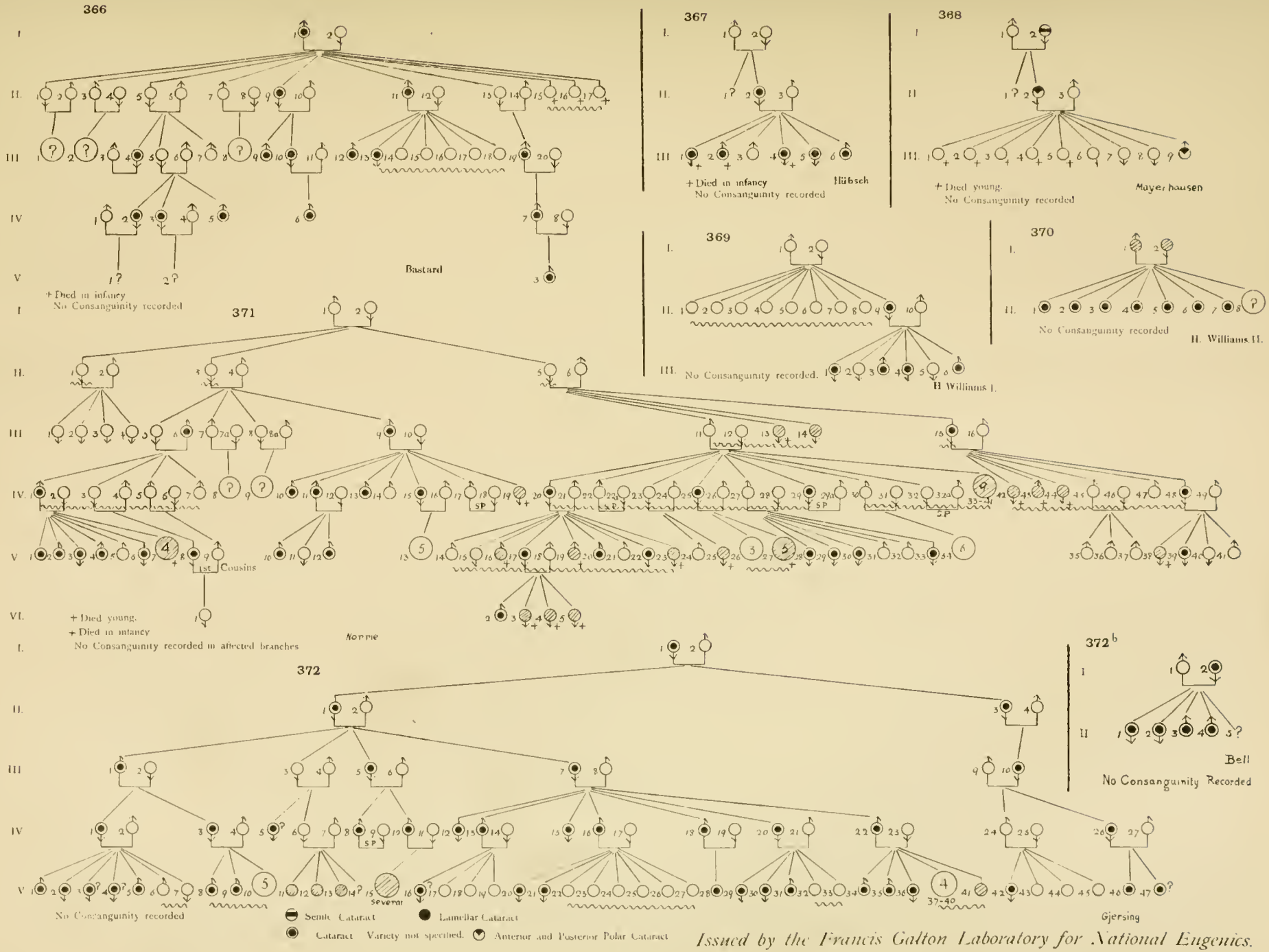
Lamellar Cataract
Discoid Cataract
Senile Cataract
Presenile Cataract
Cataract, Variety not specified
Retinitis Pigmentosa
Presumably normal
Unknown
Issued by the Francis Galton Laboratory for National Eugenics



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UNIVERSITY OF LONDON

FRANCIS GALTON LABORATORY FOR NATIONAL EUGENICS

EUGENICS LABORATORY MEMOIRS. XII.

TREASURY OF HUMAN INHERITANCE

PARTS V AND VI

SECTION XIV *a*. HAEMOPHILIA

BY

WILLIAM BULLOCH, M.D. AND PAUL FILDES, M.B., B.C.

WITH 17 PLATES OF PEDIGREES AND 1 PLATE OF ILLUSTRATION

PLATES XXXIV—L PLATE N

PEDIGREES, FIGS. 373—607, 493 *bis*

LONDON:

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1911

Fig. 5. Lamellar cataract photographed in water immediately after extraction. Extracted from an adult by Lang. No other data. Reproduced from block kindly lent by J. H. Parsons.

Fig. 6. Schirmer's section of lamellar cataract removed by operation; *a* clear nucleus, *b* lamellar opacity, *c* clear cortex. Lens extracted from left eye of boy aet. ten, who did not walk until aet. 2; no other particulars known about him. The lens had been needled six weeks previous to extraction with hardly any result. The lens was hardened in Müller's fluid and in alcohol. After hardening the following measurements were obtained:

Equatorial diameter 7·5 mm. (author says that a uniform layer of cortex seems to have peeled off during extraction).
Antero-posterior diameter 3·5 mm.
Equatorial diameter of opaque layer 5·75 mm.
Antero-posterior diameter of opaque layer 3·00 mm.
Thickness of diameter of opaque layer 0·25 to 0·3 mm.

Macroscopically. The opaque layer is quite sharply defined as to both its borders (in section); in front it comes quite to the surface of lens (on account of loss of cortex); the hinder part is covered by a thin layer of cortex; the outline of the nucleus is well defined.

Microscopically. The cortex surrounding the opaque layer is quite normal. The nucleus is not normal, it shows numerous scattered rather large vacuoles of same appearance as, but larger and much less close together, than those constituting the opaque layer, they are filled with granular material. The minute vacuoles of the opaque layer are densely packed and cease abruptly towards the clear cortex, but the deep aspect of the layer passes without any sharp boundary into the nucleus.

Fig. 7. Schirmer's section of multiple lamellar cataract removed by operation. b_1 minute opaque lamella, b_2 second lamella, with *a* clear interior, b_3 third lamella, with *d* clear interior, marked at *e* by fine incomplete lamella, *c* outer cortex. Both lenses were extracted from the eyes of a girl aet. 14. Seven years previously optical iridectomies had been performed on each eye; at that time only the central opaque layer was recorded in the notes, the author thinks it certain that the outer layer would have been detected if it had been present then, for iridectomies would not have been made unless the cortex were clear at that date; he considers the outer layer formed at about age eight, i.e. some little time after the iridectomies. No history of fits, or delayed walking, no note on teeth. The author figures sections of both lenses, that reproduced is the section of the left lens. Before extraction the lens appeared small, its margin well within the bounds of the cornea, and the fibres of the lens ligament were plainly seen. The double layers of opacity were easily seen where the iris had been removed. The inner layer was small and opaque; the outer layer and its border more or less notched, it transmitted very little mirror-light. There was a narrow zone of clear cortex superficial to the outer layer of opacity. There was a yellowish-white pyramidal opacity with its apex projecting above the anterior capsule. In section. The nucleus (*a*) looks clear except for a small delicate layer of opacity close around its centre (b_1). Microscopically both nucleus and the apparently clear layers (*d*) between the two opaque lamellae (b_2 and b_3) show a good many inter-fibrillary spaces. There are also numerous very small "droplets" which are the chief cause of the opacity of the opaque layers; and the lens-fibres in the opaque zones are often swollen or show knobs and projections. The two principal opaque lamellae vary from 0·3 to 0·45 mm. in thickness. Between the two principal opaque lamellae is a third called by the author a "second intermediary zone" (*e*) only 0·05 mm. thick, therefore much thinner than the principal lamellae and incomplete or less marked at the anterior and posterior surfaces of the lens. This layer, which clinically would have constituted a "rider," contained droplets, fine granular material and broken-looking ends of lens fibres. Figs. 6 and 7 are reproduced from the *Archiv für Ophthalmologie*, 1889, Taf. X. Figs. 1 and 3, S. 147.

(*Figs.* 8 *to* 34. *Drawings of lenses* in situ *showing various forms of cataract, with accompanying diagrammatic sections of such lenses.* N.B. *a cataract may be viewed two ways:* (1) *by focal illumination when a strong light is focussed on its surface, then it appears as a whitish opacity against the black background of the pupil or the unilluminated interior of the eye;* (2) *by reflected light, when light is projected into the eye by a mirror and returned as a red reflex from the illuminated fundus to the eye of the observer; in such circumstances the cataract looks dark or black against the bright red background. Compare Figs.* 9 *and* 10, *drawings of the same cataract seen under these different conditions.*)

Fig. 8. Small dense lamellar cataract, no riders or outlying opacities, focal illumination × 3·5. Drawn by Harman for this work; see his Case, Fig. 307. Turner family. Mrs H—— (III. 15), aet. 35; gets fair vision through optical iridectomies; teeth good; mother of seven of whom four cataractous, see Fig. 14.

Fig. 9. Fair sized lamellar cataract seen by focal illumination with several riders and small outlying masses, × 3·5. Drawn by Harman for this work. Male, aet. 15. Teeth irregular, small, very defective enamel. Generally undersized physique, limb bones distorted by rickets. No history of fits. Opacity including riders measured 6·5 to 7 mm.

Fig. 10. Drawing of same cataract, shown in Fig. 9, as seen against the illuminated fundus. Iris cut away at *i* to show clear cortex *c* and lens ligament *ll*, *o* = main opacity, *r* = rider. Drawn by Harman for this work (for more striking riders see Fig. 16, Plate M).

Fig. 11. Diagrammatic section of lens as in Fig. 10. *l* = lamellar opacity, *r* = rider, *c* = clear cortex, *n* = clear nucleus (Harman).

Fig. 12. Double-shelled lamellar cataract seen by focal illumination, × 3·5. Drawn by Harman for this work from sketch supplied by Professor M^{c}Hardy. Case, Fig. 288. (IV. 1—6.) Vision bad after removal of cataracts. Teeth excellent.

Fig. 13. Diagrammatic section of double-shelled lamellar cataract as in Fig. 12. *c* = clear cortex, *n* = clear nucleus, *l'* = inner layer of opacity, *l''* = outer layer of opacity. Drawn by Harman for this work.

Fig. 14. Very delicate lamellar cataract with dense central star in anterior layer; focal illumination, × 3·5. Drawn by Harman for this work. Case, Fig. 307, IV. 28. Male, aet. 3, child of mother whose cataract shown in Fig. 8.

Fig. 15. Section of lens as in Fig. 14. *s* = anterior star, *l* = lamellar opacity, *c* = clear cortex, *n* = clear nucleus.

PLATE M. Fig. 16. Lamellar cataract of moderate size with exceedingly distinct rider and looped spokes. The "rider" (occupying the site of X on the clock face) straddled the main opacity, its pointed central ends nearly reaching the poles, but it was separated from the main globular opacity by a broad clear layer of lens substance. Similarly the "spokes" were looped around and well clear of the main opacity in remarkably distinct lines.

The right eye of a female aet. 33, the cataract was diagnosed by Hulke when she was aet. 10. The left eye has similar cataract, only the large rider occupies the site of III. on the clock face. Vision when pupils are dilated very fair ($\frac{6}{18}$), she used a midriatic habitually. Teeth excellent. Drawn by Harman for this work.

Fig. 17. Discoid cataract, usual simple form; opacity appears dark against illuminated fundus, × 3·5. Nettleship and Ogilvie's Case (Fig. 345, V. 9).

Fig. 18. Discoid cataract showing trilobed denser portion of disc, lighted as in Fig. 16, × 3·5. Nettleship and Ogilvie's Case (Fig. 345, VI. 38).

(Figs. 17 and 18 are reproduced by kind permission from *Trans. Opthal. Soc.*, Vol. XXVI., 1906, Plate VIII.)

Fig. 19. Diagrammatic section of lens as in Figs. 16 and 17. *d* = discoid opacity, *n* = nucleus, *c* = clear cortex. Drawn by Harman for this work.

Figs. 20—26. Showing progression of form and size of opacity from the simplest discoid cataract (Fig. 20) to lamellar cataract (Figs. 24—25). In each case the opacity appears dark against the illuminated fundus. In Fig. 25 the iris is cut away below to show refraction stria, × 1·3. Case 342. Nettleship's pedigree of S.—— family. Fig. 21, IV. 104; 22, 23 right and left of IV. 70; 24, 25 right and left of V. 82; 26, IV. 102. Redrawn by Harman for this work from portions of Nettleship's original plate.

Fig. 27. Coralliform cataract, seen by focal illumination, × 2·75. Reproduced from a drawing kindly provided by Holmes Spicer. Nettleship's Case, Betts family, No. 318, IV. 2. The case illustrated by the figure was published by Marcus Gunn, but the drawing here reproduced is one that Holmes Spicer considers more accurate than the one he originally published. For description see page 127 of text. N.B. no diagrammatic section of this cataract is given since it is not certain whether the trumpet-shaped opacities extend into the posterior part of the lens as well as forwards and into the equator.

Fig. 28. Müller's diagrammatic section of lens with *ab* axial opacity, *c* complete lamellar opacity, *e* incomplete anterior lamella, *dd* points of thickening of axial opacity about main lamella. Case No. 325. Reproduced from *Archiv für Ophthalmologie*, 1855, S. 169.

Fig. 29. Stellate cataract, Y anterior and ✳ posterior opacities; focal illumination from left-hand side and looked at from right-hand side, × 3·5. Drawn by Harman for this work from the separate line drawings of the anterior and posterior markings published by Adams. Case Fig. 308 (V. 3).

Fig. 30. Microphthalmia and anterior polar cataract, focal illumination × 3·5. The smallness of the cornea can be judged by comparison with Figs. 16, 17 and 18, all four of which are on the same scale. Corneal diameter measured 7·5 mm. (average 11·6 mm.). Drawn by Harman for this work. Case, Fig. 339 (III. 8).

Fig. 31. Diagrammatic section of lens as in Fig. 29; the anterior polar opacity is seen to dip into the lens; the dotted radiations indicate "refraction striae," seen when the living lens was examined by reflected light. Drawn by Harman for this work.

Fig. 32. Posterior polar or "hyaloid" cataract, with small anterior polar opacity; focal illumination from left; viewed from right side, × 3·5. The posterior part of the lens is occupied by dense white opacity fringed on its anterior aspect, it is united by a fine axial stalk to a dense white button-shaped opacity situated in or just beneath the capsule. The front of this button had an elevation which bulged forwards into the contracted pupil. N.B. only one of the ten affected in this family had this anterior polar opacity, all ten had the posterior larger opacity. Drawn by Harman for this work. Case, Fig. 341 (IV. 12).

Fig. 33. Diagrammatic section of lens as in Fig. 31. a = anterior polar cataract, dipping down to meet p posterior polar mass, h = hyaloid vessels probably present, c = clear cortex. Drawn by Harman for this work.

Fig. 34. Semi-diagrammatic section of eye with ill-developed lens, posterior polar cataract and persistent hyaloid artery. Projecting from the lower margin of the pupil into the pupillary area is a tag of persistent 'pupillary membrane.' Reproduced from a block kindly lent by Treacher Collins. (Bibl. No. 21.)

HAEMOPHILIA.

By William Bulloch and Paul Fildes.

INTRODUCTORY AND HISTORICAL.

(Synonyms. Haematophilia, Haemorrhagophilia, Haemorrhaphilia (Schönlein), idiosyncrasia haemorrhagica (Kuhl), amychaemorrhagia (Uhde) (ἀμυχή, a small wound), Blutsucht, Bluterkrankheit, haemorrhagic diathesis, hémophilie, hémorrhagie constitutionnelle, morbus haematicus, erbliche Neigung zu tödtlichen Blutungen, Haemorrhoea.)

Haemophilia is a disease characterised by an excessive and chronic liability to immoderate haemorrhage. The liability is hereditary, and, in our opinion, based upon the published evidence, is confined to the male sex. The affected individuals are spoken of as "bleeders" in English, and by some appropriate translation of this word in other languages, e.g. "Bluter" in German, "hommes saignants" in French. It is

reasonable to suppose that, before medicine was a specialised branch of human activity, it was already well known that pressure applied to a wound would arrest the flow of blood. The sight then, of a patient slowly becoming exsanguine from a trivial injury, capable, perhaps, of being securely enveloped in bandages, must leave a lasting impression on the observer, an impression which frequently colours and adds a touch of human interest to the account subsequently written.

History. It may appear to be a matter for surprise that a disease so remarkable as haemophilia was entirely undescribed as a distinct morbid entity before the beginning of the nineteenth century, although it is now well known that numerous cases had occurred before that date, and that a few isolated examples had actually been recorded. They had, however, been overlooked or forgotten. The advent of a circulating medical literature was already bringing about a change and was producing facilities for recording the remarkable. In 1803, Dr John C. Otto[17], a physician of Philadelphia, published "An account of an haemorrhagic disposition existing in certain families," and described briefly the occurrence of this idiosyncrasy in the male descendants of a woman named Smith, who had settled some seventy or eighty years previously in the vicinity of Plymouth, New Hampshire. Otto stated that "males only are affected and all are not liable to it. Though females are free they are capable of transmitting it to their children." It would seem that Otto did not speak with personal authority on the subject of the Smith-Shepard family as he adds that the sources of his information are confirmed by the hon. Judge Livemore, and "Drs Rogers and Porter gentlemen of character living in the neighbourhood." Dr Benjamin Rush also told Otto of similarly affected families, one in York, and the other in Northampton county, U.S.A., and a Mr Boardley, through Rush, gave information of a family in Maryland (Pedigree No. 516). It would thus appear that the disease haemophilia was at that time well known to people living in the vicinity of those affected. The word "bleeder" is used in Otto's paper for the first time "for this is the name given to them." Otto's account marks the origin of our knowledge of the subject of haemophilia in so far that it was the first to arouse general interest, being reviewed in the *Sammlung auserlesener Abhandlungen zum Gebrauche praktischer Aerzte* in 1805[19]. The editor of this paper was Christian Erhard Kapp, who practised in Leipzig and Dresden. Although this account of haemophilia is constantly referred to as being by Kapp, we can find no evidence that this is so, as the article is unsigned. The writer, whoever he was, states (p. 275) that he himself knew several cases, all of the male sex, and that one or two of them had bled to death from the mouth. One may also suspect that Otto's paper was the cause of the publication of E. H. Smith's[20] case which appeared in the first volume of the *Philadelphia Medical Museum* in 1805. This case, frequently referred to as that of Coxe and Smith, consists of an extract of a letter of the late Dr E. H. Smith of New York, written in 1794 to Dr Benjamin Rush, at that time the central figure in the American medical world, and relates the history of the author's cousin, a boy who had bled to death thirty years previously, the facts being supplied to Smith by his aunt, the boy's mother. The account is not

of much value, but an addendum by Redman Coxe[18], the editor of the *Museum*, is of interest, since it calls attention to the existence of similar cases (du Gard[4], Ash[6], Clopton Havers[7]) which had been published, long before, in the *Philosophical Transactions*. Thus by 1805 haemophilia had begun to attract attention. In 1810 Brückmann[21] reported the history of a man aged 70 who bled to death from the mouth. In the same year Consbruch[22] of Bielefeld inserted in Hufeland's *Journal* an account of a family which, according to Nasse[35], is the same as that described by the anonymous author of *Medicinische Ephemeriden*[13], to be afterwards referred to. That Nasse was correct in this assumption is highly probable from the fact that he speaks of having known Consbruch personally, and also one of the bleeders described by Consbruch. Nasse's acquaintance with Consbruch came no doubt from his connection with Bielefeld, where he was born in 1778. In Hirsch's *Biographisches Lexikon der hervorragenden Aerzte aller Zeiten und Völker* it is also expressly stated in the article on Consbruch that he was the writer of *Medicinische Ephemeriden*. Consbruch (1764—1837) was a well-known physician, long in practice at Bielefeld and author of many papers in medical journals, besides being joint editor with Ebermaier and Niemann of an *Allgemeine Encyclopädie für praktische Aerzte*. It is however to the observations of physicians in North America that we are most indebted for advances. In 1813 Dr John Hay[25], of Reading, Mass., a small town near Boston, published the history of the well-known Appleton-Swain family (Pedigree No. 408), the descendants of one, Oliver Appleton, who was living at Ipswich, Mass., in the beginning of the eighteenth century. The subsequent history of this family has been traced down to modern times by Osler[499] and Pratt[869]. This family was considered so remarkable that it was cited as one of the curiosities of the district by Felt[199] in his *History of Ipswich*, published in 1834. Hay states that a person of the name of Appleton married a Smith of Haverhill, who is supposed to be the Mrs Smith mentioned by Dr Otto, and from this remark it has been stated by many writers that the Smith-Shepard and Appleton-Swain families were connected by marriage. Hay's paper was reviewed by J. F. Meckel[27] in his *Archiv* in 1816, and in this way the knowledge of haemophilia was no doubt widely spread. The next important case also comes from America, being the account in 1817 by Drs William and Samuel Buel[29] of the haemophilic descendants (Pedigree No. 398) of the Rev. Timothy Collins, the first pastor of the town of Litchfield, Conn. The same year saw the publication of Blagden's[28] case, the first contribution to the subject in this country, although Otto's paper had been reprinted in the *Medical and Physical Journal*, London, 1808. Blagden's case is not with certainty one of haemophilia, but it is of interest in so far that the haemorrhage was so great that Sir Benjamin Brodie ligated the carotid artery but without success. Blagden was followed in this country by James Wilson[32], who in 1819 described an haemophilic family (Pedigree No. 463). When, therefore, Christian Friedrich Nasse[35], Professor of Medicine at Bonn, investigated this subject in 1820, there was already a considerable mass of data. This he collected and compiled with great discrimination and skill, and introduced new material from his own observations and from those of his assistant Krimer

(Pedigree No. 440). In this way he was able to give a very complete and accurate account of haemophilia which he raised to its appointed rank in the nosological system. From the data before him he was able to assert that *males alone* are the subjects of haemophilia, and that the disease is transmitted by normal females through their marriages with normal males. "An ihnen (the women) selbst und überhaupt an einer weiblichen Person jener Familien äusserte sich eine solche Neigung (to haemorrhage) niemals." The affection of the male sex, and the transmission of the disease through normal unaffected females, is frequently described in the literature as "Nasse's law," although prior to Nasse all the classical writers on haemophilia had emphasised this mode of transmission. The publication of Nasse's paper came about the time when the teaching of J. L. Schönlein was revolutionising German medicine, and from it dates the onset of the flood of publications on haemophilia which has continued down to the present day. Among the more interesting of the publications of succeeding years may be mentioned those of Elsaesser[38] (1824), Davis[46] (1826), Chelius[48] (1827), Coates[51] (1828), Rieken[60] (1829), Hughes[65,71] (1831), Escherich[88] (1835), Vieli[164] (1846), Wachsmuth[181] (1849), and Lange[193] (1851). In addition appeared a large number of communications which, a study shows, contained nothing new or were of little value. The concept of the disease, based upon such communications, tended to become more and more obscured, and drifted farther and farther from the clear statements of the original accounts. Dr Ludwig Grandidier, a Brunnenarzt at Nenndorf and practising in Cassel, made the collection of these data the main interest of his life, and for more than forty years contributed collective accounts on the subject of haemophilia, his chief work being a monograph *Die Haemophilie oder die Bluterkrankheit*, Leipzig, 1855 (2nd edit. 1877). It cannot be denied that without Grandidier's labours a large mass of literature would have been lost in oblivion. While this, in our opinion, would have been desirable in a very large number of instances, it must be admitted that the balance of his data is of value. Nevertheless, it must be stated that his figures and statistics, which have been freely copied by all subsequent writers without criticism, can only be described as largely erroneous. A personal study of practically the whole of the haemophilic literature has led us to the belief that in many instances he had not seen the original papers, his information being derived from abstracts in journals and year-books, and suffering, no doubt, in transcription. Subsequently to the appearance of Grandidier's monograph the production of material, good and bad, continued. In 1872 Wickham Legg[331], the next notable writer, published his scholarly monograph. He appears to have been the first to attempt anything like a critical survey of the data published up to this time. His monograph was followed by Immermann's[401] exhaustive article in Ziemssen's *Cyclopaedia* in 1876, this being one of the most authoritative accounts. The second edition of Grandidier's book, published in 1877 shortly before his death, may be taken as the last of the classical works on the disease.

As has been stated above, Otto's paper (1803) was the origin of our knowledge of haemophilia. It remains however to notice the cases which have been

unearthed among the works of previous generations. The first mention of a disease resembling haemophilia in any way is to be found in the writings of Khalaf ibn Abbas, Abu-al-Kasim, commonly called Albucasis or Alsaharavius, the greatest surgical writer of the Moorish period. He is generally stated to have been born at Zahra, a summer residence of the Caliphs near Cordoba, and to have flourished in the tenth century (Hirsch). If his personality is obscure, his fame has been handed down by his work, *al-Tasrif*, a handbook of practical medicine and surgery in two parts, the first of which is known through an incomplete and barbarous Latin translation, made by Paul Ricius under the title *Liber theoricae nec non practicae Alsaharavii*, Augsburg, 1519. Albucasis[1] tells us that in a certain village there were men who when wounded or bled suffered an uncontrollable haemorrhage which caused death. The same accident happened to the boys if their gums were rubbed harshly, and they most commonly died. This account appears to have been first quoted by Testa[24], whose translator, Kurt Sprengel[26], was however unable to confirm the reference. Nasse[35], however, rediscovered a fact, known previously to Schenck[3] and Freind[11], that Alsaharavius and Albucasis were one and the same person, and identified the passage which has subsequently been quoted by almost all writers. The next very doubtful case is that of Alexander Benedictus[2] (1539), who described the case of a Venetian barber who perished miserably of epistaxis, the result of accidentally cutting his nose with a pair of scissors. To the learning of Virchow we are indebted for the case reported by Philip Hoechstetter[5] in 1674. This was a boy who at birth bled from the umbilicus. As he grew up he was troubled with epistaxis to such an extent as to cause alarm. Blood in the stools and petechiae, and suggillations in the skin, were frequent, but disappeared before his eleventh year. Very doubtful are the cases reported by du Gard[4], Ash[6], Clopton Havers[7] and Musgrave[8]. More allied to haemophilia is the case described by Banyer[9] (1743). In 1784 two instances of alleged haemophilia were published by Sir William Fordyce[12], a military surgeon long in practice in London. The one refers to a certain Laston, living at Duddington (Northampton), who suffered from epistaxis, as also did his children. The other is the history of one Hay, a drummer in the guards ("tertiae cohortis praetorianae tympanista"). Neither is, in our opinion, of importance. We believe that the reference in *Medicinische Ephemeriden*, published at Chemnitz in 1793, has not been verified in recent years on account of the great rarity of this publication. Through the kindness of Professor Th. Lochte, of Göttingen, we have obtained an exact transcript of the passage in question, which shows that the anonymous author, presumably G. W. Consbruch, was describing a classical instance of the disease, and as such is the first in the history of haemophilia. The last of the genuine cases, prior to Otto's paper, was that by Alexander Rave[14] in 1796 (Pedigree No. 510). In recent years N. Rothschild[452] (1882) has attempted to show that haemophilia must have been known to the ancient Jews. His inferences are based on certain dispensations in the Talmud with regard to the question of circumcision. Although the subject of haemorrhage is not specifically mentioned, the statements can scarcely refer to any other complication of this ancient rite. Thus in the *Tractat*

Jebamoth it is related that four sisters lived in Zipporah; the first had her child circumcised and he died, the second likewise, and likewise the third. The fourth then went to the Rabbi, Simon ben Gamaliel, who ordered that her child was not to be circumcised. As Rabbi Simon ben Gamaliel lived in the second century A.D. it is claimed that this is the oldest reference to the disease. It is also stated that a boy must not be circumcised if two sons of his maternal aunts have died from the operation. Maimonides also declared that a boy was not to be circumcised if his two brothers by the same mother, but by different fathers, had died after the operation.

Symptoms. The cardinal symptoms of haemophilia are three in number, and may be boldly summed up in the following definition—an *inherited* tendency in *males* to *bleed.*

Haemorrhage. The question of haemorrhage may be considered first. The essential feature in this respect is that the application of a trauma, which in a normal person would pass unnoticed, determines, in a bleeder, an haemorrhage, and when this haemorrhage is established there is no tendency for it to stop in the usual manner. The blood is described as trickling or oozing away from a surface in spite of all treatment, and continuing till death ensues, or, what is very much more common, till spontaneous arrest occurs after the patient is reduced to a condition of profound anaemia. A classification of the *sites* chiefly selected by the haemorrhages possesses no special significance. They may be external or internal. The blood may issue from any area of the broken skin or be suffused beneath it, constituting the typical bruise, ecchymosis, or suggillation. The blood may also escape into internal cavities or be lost to the body by way of any of the ostia. Certain special haemorrhages deserve mention. *Epistaxis* is of common occurrence in bleeders, but by itself is also frequently met with in people who present no other feature of the disease. Even when repeated and alarming, the occurrence of epistaxis alone does not in our opinion justify the diagnosis of haemophilia, although there may be no evidence of an organic cause to account for the haemorrhage. The *umbilicus* at birth is seldom a site of haemorrhage in haemophilia, and when it occurs, starts shortly after the severance of what appears to be a perfectly normal but ill-tied cord. The large number of cases of umbilical haemorrhage, quoted in the literature as haemophilia, date from a period when syphilis and other infective conditions were not recognised as sufficient causes for bleeding in this site. According to all testimony Jennerian vaccination in a bleeder is not associated with greater risk than in a perfectly normal individual. Haemorrhage from the *mouth*, following the cutting of the milk teeth and especially the eruption of the second dentition, is a constant feature of the disease. *Tooth extraction* has been responsible for a number of deaths, but it is unnecessary to assume that a haemorrhagic diathesis pre-existed. It must be recognised that in the past the dental key has been responsible for widespread intrabuccal injuries which, when inevitably infected and barbarously treated, have determined death without the aid of a tendency to bleed. *Haemarthroses* and *articular effusions* constitute one of the most typical features of the disease, and

have been noted from the earliest times. The joint commonly affected is the knee, and the lesion closely resembles tuberculosis. Error in diagnosis has led to operative interference and death on several occasions, even in the hands of the illustrious. A considerable literature exists on the subject of joint lesions in bleeders, and it will be found in the writings of Dubois[171], Reinert[309], Lossen[375], Stahel[420], Bokelmann[432], Sabrazès and Cabannes[686], Launay[701], Thébaud[691], Vannierre[846], Gocht[698], Linser[645] and Froehlich[768,769]. The principal paper is, however, that of Franz König[562], whose classical description all subsequent writers have followed. He distinguishes three stages, viz. haemarthrosis, panarthritis, and deformity. It is probable that few bleeders escape crippling from these articular lesions, although they have a tendency to become progressively rarer as the affected individual grows up.

An outstanding feature of haemophilic bleeding is that in the majority of cases it can be definitely traced to *trauma*. The haemorrhages are frequently stated to have been *spontaneous* in origin, but this much must be said, that they are usually in a position commonly liable to slight knocks or pressure. The skin lesions vary from a bruise to an haematoma, which may be of great dimensions. They are sometimes described as "purpuric" or "blue spots," but such expressions do not depict the condition usually found. The liability to haemorrhage is always *chronic*. It is noticed in early childhood, and makes the existence of the sufferer wretched, throughout youth to manhood, or perhaps to old age. It appears, however, to be established that if adult life is reached a progressive decrease in the number and degree of the haemorrhages may occur, so that in middle life the individual may be practically free from his infirmity. Variations in the intensity of the liability to bleeding are frequently well marked, and have been very clearly demonstrated over a series of years by Ripke[548]. A boy may be seen literally covered with bruises and prostrated by some particular bleeding. Pressure with the finger will determine a bruise in a few minutes and yet a week or two later the same boy may be free from bruises, react normally to pressure or a needle stab, and even sustain a cut without immoderate haemorrhage. The general condition of the patient at the time of the bleeding is, in the majority of cases, apart from the bleeding, good. Fever, malaise and constitutional disturbance are slight or absent, although they may occur particularly when the haemorrhage is internal. When the bleeding ceases, recovery is usually rapid. The boy regains his temperament of gaiety and recklessness, which outlook on life is so constantly mentioned as to constitute a minor feature of the disease. Indeed the patient presents a striking contrast to a person suffering from scurvy or purpura and other little understood conditions associated with losses of blood. Some of the older writers on haemophilia described the subjects of the disease as of fair complexion, with unusually fine skins through which the veins could be clearly seen, and whereas many have referred to this, there are as many records where it has been stated not to have been the case. There is nothing in the physiognomy or temperament by which a bleeder can be diagnosed at sight.

Sex. The question of the immunity of the female sex from haemophilia is one on which opinion is divided. Some look upon the disease as sex limited and confined

to males, whereas others repudiate this view and assert that females are attacked with comparative frequence. A superficial review of the literature, however, is sufficient to show that the latter opinion is unsupported by adequate evidence. A more weighty suggestion has been advanced, however, from analogy to other sex-limited diseases. In conditions such as colour blindness and pseudo-hypertrophic muscular palsy, which have been considered to be confined to the male sex, rare but well authenticated cases are alleged to have occurred in females. Similarly, it is suggested that haemophilia may occur in females, though rarely. This subject, being one of fundamental importance in the consideration of questions of inheritance, must be discussed in some detail.

The anonymous author, presumably G. W. Consbruch, who published the first undoubted case of haemophilia, in 1793, stated with reference to the family observed by him "all females in this family are, so far as I am aware, free from this unhappy idiosyncrasy....They menstruate normally, and are quite healthy." The family described by the American physician, Otto, in 1803, was the first to attract wide attention. He put on record that "it is a surprising circumstance that the males only are subject to this strange affection....Although females are exempt they are still capable of transmitting it....When the cases shall become more numerous it may perhaps be found that the female sex is not entirely exempt, but, as far as my knowledge extends, there has not been an instance of their being attacked." In Nasse's publication[35] the occurrence of bleeding in females appears to be noted for the first time. This author has been frequently quoted in support of the theory of haemophilia in the female, but it is clear from his account that he was opposed to such a doctrine. He knew (*l.c.* p. 425) of a family in Bonn in which the five sons "were subject to severe bleeding from adolescence onwards," while their sister, mother, grandmother, and great-grandmother suffered from menorrhagia and *post partum* haemorrhage, the two latter bleeding to death during the menopause. The father of these children also had severe bleedings in his youth. In the paragraph following this description, however, he refers to the male as "the sex to which the inherited liability to severe haemorrhage is confined entirely." The account given by Dr Theodore Davis, in 1826, is of interest in that the author was probably unacquainted with any previous writings on this disease. One of the women in the family described by him stated that "the females enjoyed good health and were entirely free from the tendency to bleed." On the other hand, Lange, reviewing the literature on haemophilia in 1849, found that the ratio of female to male bleeders was as 1 : 7, and a few years later Grandidier gave it as 1 : 14. He pointed out, however, that the ratio varied in different countries, being 1 : 11 in Germany, but much less in France, England and North America. Up to that time no female bleeder had been reported in the Swiss haemophilic families. With reference to the disease in women, Grandidier quoted, at some length, the cases described by Kuhl, Tamme Beth, and Quadrat as typical instances of the disease, and he remarked that "the less pronounced forms of haemophilia in women are characterised by various anomalies of the blood vascular system" such as disorders of menstruation, haemorrhage in connection with childbirth, and spontaneous bruising (*l.c.* p. 88).

Wickham Legg (331), in his well-known monograph published in 1872, did not follow the concept of haemophilia in women laid down by Grandidier, and adversely (p. 30) criticised the data collected by that writer. He considered that women are much less disposed than men, the attacks being milder and atypical. "They do not bleed more than is usual when wounded, and the disposition may limit itself to the occurrence of spontaneous haemorrhages, or to early, abundant and prolonged menstruation. Floodings are common both after delivery and at the time of the cessation of the catamenia." The whole of this subject is indeed dismissed in a few lines in this otherwise exhaustive and critical monograph. An entire chapter, however, is devoted to "certain haemorrhagic diatheses in women" (*l.c.* p. 129), the subject being introduced by the remark "A chronic haemorrhagic diathesis is not unfrequently seen in women." He regarded these diatheses as entirely distinct from haemophilia. In 1876 F. A. Kehrer (363), a gynaecologist, extensively reviewed this question, and judging from the original cases recorded in his paper, it would appear that he regarded severe uterine haemorrhage as synonymous with haemophilia. The view of Immermann was based largely on the writings of Grandidier. He stated that "well-marked haemophilia" was very much more common in males than in females, but that there was a large number of rudimentary or anomalous cases which were far commoner in females than in males. Further, though he was not forgetful of the fact, as he himself put it, "that the congenital and habitual character enters into the definition of the bleeder disease as an essential element," he called attention to the possibility that a single isolated haemorrhage might be the manifestation of a congenital, habitual but *latent* defect. A girl temporarily affected by a severe haemorrhage and belonging to a bleeder family, in his opinion, should be regarded as a bleeder. Wright, whose personal experience of haemophilia has been extensive, defined haemophilia as a disease of males. The latest paper on the subject, by Böhm, treats it from the point of view of the gynaecologist. He also appears to use the term haemophilia as implying merely haemorrhage from the sexual organs, especially at the menarchy or the menopause.

In endeavouring to reconcile these divergent opinions the suspicion arises that the above quoted authors are not using the word haemophilia in the same sense. It is to be presumed that the histories of the great haemophilic families have been well known to all writers on haemophilia, but if so, it is difficult to realise on what grounds many have selected "haemophilia" as a fitting title for the pathological conditions described by them. It appears to us that they have applied this term to instances of "idiopathic" and other haemorrhages from the female genitals, which have nothing in common with haemophilia except an excessive flux of blood.

Haemophilia has been described as occurring in the female in any of the following types:

I. "Well-marked haemophilia," as presumably supposed by Immermann. This concept implies that females are subject to the disease, as witnessed in the male, and as described in the beginning of this paper.

II. An abnormal tendency, in females, to bleed, without there being any obvious

connection between such females and bleeder families. It often occurs, however, that haemorrhages in other members of the family are adduced as evidence that the particular affection is inherited. To this class belong the cases of "haemorrhagic diathesis," isolated cases due to various causes, and the cases described chiefly by gynaecologists.

III. An abnormal tendency to bleed, in female members of admitted haemophilic families, such tendency being slight or atypical as compared with "well-marked haemophilia."

Wickham Legg, Grandidier, and Immermann may be said to have held this latter opinion.

With reference to Type I, that females are liable to "well-marked haemophilia," the evidence on which Immermann based this statement is not given. It probably rested on general remarks derived from Grandidier, such as "There is no lack of examples of haemophilia in females in its most pronounced form leading to death. In these cases the symptoms are only modified by the anatomical differences between the two sexes." So far as we can find, after studying the contents of some 900 papers on haemophilia, no case has yet been described in a female which bears more than a superficial resemblance to the disease as found in the male. As an illustration of this type of case may be mentioned that described by Kolster. (See Bibl. No. 630.) This case differs from well-marked haemophilia in the following ways. The external haemorrhages were entirely visceral or from the mucous membranes. There is no mention of haemorrhage following trauma. Spots appeared in crops in places not particularly liable to injury. There was no history of bruising. The swelling of the members and the face does not occur in haemophilia, except in one particular vascular area from pressure upon the blood vessels by an haematoma. The affection of the joints in haemophilia is not multiple *unless more than one joint has been subjected to injury.* On the contrary, cases of the type described by Kolster are comparatively common and are usually referred to as purpura with visceral lesions, a chronicity extending over years being a well recognised characteristic. The occurrence of *intermittent fever* and purpura in the mother is suggestive of malaria as a cause for the daughter's condition. The attempt to introduce the inherited element in Kolster's cases seems to us unconvincing. Statements that an individual suffered very much from bleeding from small wounds cannot be accepted as evidence of haemophilia.

It must again be affirmed that all statements alleging the occurrence of well-marked haemophilia in females are unsupported by adequate evidence.

With regard to Type II, the following are the cases quoted by Grandidier as typical of haemophilia in females : *Lafargue's Case* (Bibl. No. 89, Pedigree No. 546), *Kuhl's Case* (Bibl. No. 93), *Tamme Beth's Case* (Bibl. No. 56, Pedigree No. 572), and *Quadrat's Case* (Bibl. No. 124). It is perhaps unnecessary to review these cases in detail. They belong to a period long past, and few would suggest haemophilia as a diagnosis at the present day. The latter remark should also apply to those instances which have been recorded in gynaecological practice from time to time under the

name haemophilia, but we find that a recent thesis on this subject by Böhm was favourably reviewed in the English press. He collected 64 cases of "certain haemophilia in females," which have been published in the literature, and concluded "that the occurrence of haemophilia may be accepted without further question." The majority of the cases referred to by Böhm are reviewed in the bibliography appended to this paper, and freed from certain inaccuracies which we have noticed in his account. Of his seven original cases attention need only be called to the second (p. 35), the account of a girl aged 18. This appears to us to suggest morbus maculosus. Among others this subject has been discussed by Kehrer and Börner.

It is, however, impossible to dismiss the subject of haemophilia in women without reference to that rare and obscure condition, sometimes referred to as haematidrosis or sweating of blood. This disease, which is probably a complex of pathological states, constitutes a chronic "haemorrhagic diathesis." The onset of symptoms is frequently at or shortly before puberty, though cases are on record as early as six. The duration of the attack is seldom stated, the accounts being penned in the height of the condition. The symptoms consist of haemorrhage or the exudation of bloodstained fluid under the skin or from any area of the body surface. Of these haemorrhages, the most remarkable, and that which was considered a marvel, is sweating of blood from the pores, hair follicles or sweat glands. The attack is often preceded by some violent emotion or sometimes trauma. In any case the subjects are usually of a neurotic temperament. The eyelids or conjunctivae have also been known to exude blood. Cuts or similar injuries are not attended by unusual haemorrhage. Menstruation is usually abnormal in some way, sometimes reaching a degree of menorrhagia. In one case it is alleged that a girl bled 472 days, losing 3000 ounces of blood by the vagina. There is no tendency for this condition to be inherited. However remarkable may be the haemorrhages in cases of this nature, they appear to differ from haemophilia clinically, and in all probability pathologically.

With reference to Type III, that women in admitted bleeder families differ from normal women in displaying some slight tendency to immoderate haemorrhage, comparatively little has been written. The necessity for collecting evidence in support of this concept is not so much of clinical, as of scientific importance. The majority of such cases quoted as haemophilia are admitted to be slight or atypical, but it is necessary to establish the fact of the actual occurrence in the female in order to obtain reliable data bearing on the inheritance of human diseases. For the purpose of this enquiry we have confined ourselves entirely to the list of cases which we consider to be sufficiently well described to be of conclusive value. Of these 44 families only 10 *claim* to contain female bleeders. The following table supplies the precise and complete published data on which the diagnosis of haemophilia was made by the respective authors.

It will be seen that of the 19 alleged female bleeders, four, viz. the cases of Gocht, Cantani (2), Oliver II., are merely *stated* to have been affected without any evidence being adduced. They may therefore be dismissed. The other cases may be dealt with briefly. (1) *Rieken's Case*, IV. 8. The fate of this infant was the natural result

TABLE I. *Evidence of haemophilia, in females, as contained in the records of all haemophilic families which have been sufficiently reported for statistical purposes.*

Name of author and no. of pedigree	Total no. of females in pedigree	No. of alleged female bleeders in pedigree	Evidence[1]
Gocht, 386.	10	1	III. 4, statement only; no data.
Rieken, 390.	9	1	IV. 8, b. 1778, a weakling, unable to suck properly on account of tongue tie. When four days old a nurse relieved the tongue tie with some instrument. Furious bleeding from the fraenum set in and she died of convulsions and bleeding on the following day.
Eve and Bidwell, 399.	4	2	III. 9, aged 36, bruises easily, alive, unmarried. III. 10, aged 30, suffered from flooding after two confinements.
Burger, 401.	16	1	III. 3, large and stout; between the ages of 30 and 50 she used to vomit blood or pass it by the bowel in spring and autumn; livid spots throughout life. No evidence of bleeding in youth; died at the age of 72.
Wachsmuth, 409.	4	2	IV. 1, and IV. 2, "two daughters of a cousin (paternal side)" of Wachsmuth's father-in-law. The statement is made that they showed suggillations and dangerous bleedings after injuries, although they never had spontaneous haemorrhages. The one (which?) aged 20 died in the bridal night of bleeding from the ruptured hymen, the other died of bleeding at about the same age. Site of bleeding not stated. Wachsmuth was unable to give any information regarding the menstruation of either.
H. Fischer, 416.	2++	1	III. 2, "repeatedly suffered from epistaxis and bleeding from the gums."
Max Fischer, 426.	24	4	III. 10, aged 63; bled from the nose while a spinster; lost a great quantity of blood during confinements; never bled much after injuries. In later life epistaxis ceased. III. 15, aged 58; "often had epistaxis but as age advanced this disappeared." IV. 47, aged 29; as a spinster often had epistaxis "but otherwise had never bled violently." IV. 50, a "severe bleeder," aged 25. Five or seven years ago almost bled to death after tooth extraction and at menstrual periods loses extraordinary quantities of blood whereas during confinements the loss is not excessive.
Wright I., 495.	12	4	II. 13, "died of haemorrhage at climacteric." III. 16, aged 36; suffers from floodings after confinements. III. 18, "has had floodings after confinements." III. 20, "has menorrhagia and flooding."
Cantani, 374.	3	2	II. 4, and II. 5, "haemophilic."
Oliver II., 536.	2	1	III. 2, "said to have a tendency to bleed."

[1] The key numbers in this column refer to the generations and individuals in the respective pedigrees.

of the procedure described. (2) *Eve and Bidwell's Cases.* We consider that neither bruising nor *post partum* haemorrhage, in the absence of other details, is sufficient to establish the diagnosis of haemophilia. (3) *Burger's Case,* III. 3. The great frequence of gastric ulcer in the female suggests a more likely diagnosis than the rare disease under consideration. (4) *Wachsmuth's Cases.* Severe and fatal haemorrhage from laceration during the first coitus is a well recognised accident, apart from any abnormal tendency to bleed. Beyond this the evidence rests on the general statement as to the occurrence of suggillations and dangerous bleedings after injuries. While we confess that such a condition must be considered abnormal, we submit that such vague evidence in a monograph otherwise replete with so many minute details is not to be relied on. It is not even stated which of the two girls was the one which died of rupture of the hymen. Wachsmuth was unable to give any data regarding the menstrual peculiarities of either. (5) *H. Fischer's Case.* Bleeding from the nose is a very common condition, and bleeding from the gums might be due to many causes not differentiated in Fischer's paper. (6) *Max Fischer's Cases.* The preceding remarks apply to much of the evidence of haemophilia in these cases. The occurrence of haemorrhage after tooth extraction (IV. 50) is not of itself evidence of haemophilia even when associated with menstrual disorders. (7) *Wright's Cases.* The first alleged female bleeder died of haemorrhage from a site not stated. This was probably from the uterus, and in the absence of details may have been due, at the age stated, to a carcinoma of that organ.

Thus in none of the families of bleeders which have been comparatively well described and recorded in some detail do we find any unequivocal evidence of abnormality in the women, that is to say, any abnormality beyond what might be expected in any collection of females taken at random. That the females in bleeder families are abnormal in some unknown particular must readily be admitted. They are the active propagators of the disease. But if their abnormality is so slight as to escape notice, or entirely latent, as suggested by Immermann, further discussion of the question will be without profit until some method of diagnosis has been devised which does not depend on outward symptoms.

Inheritance. It has always been considered one of the most remarkable features of haemophilia that it runs in families, and the earliest writers on the disease were so definite on this point that, even in 1820, Nasse had sufficient material at his command to assert that haemophilia is propagated entirely by the unaffected females to their sons. Subsequent experience led Grandidier to state that it was the most heritable of all heritable diseases, a view upon which there has been general concurrence. We are, however, unacquainted with any work in which the data concerning the manner of transmission have been dealt with systematically. This we attempted to do, and on examining the material which has been published it became manifest, at an early stage, that in order to arrive at trustworthy results it was necessary to free a relatively small residuum from the great mass of inadequately reported cases, many of which did not bear the stamp of haemophilia at all. Any selection may appear to the reader to raise the question of bias, but in defence of our position it is

TABLE II.

Pedigree No.	No. of bleeder sibships in pedigree	No. of male bleeders	No. of doubtful male bleeders	No. of normal males	No. of "sex not stated"	No. of unaffected females	No. of alleged female bleeders	No. of males who were fathers	No. of females who were mothers	No. of instances of transmission through unaffected females	No. of instances of transmission through affected males		No. of instances of transmission through normal male	Remarks
											certain	doubtful		
"Tenna," 373.	19	31	0	13	7	39	0	9	14	19	2*	—	1†	* II. 6, and V. 38, † III. 25.
Ripke, 377.	3	5	0	5	1	13	0	0	2	2	—	—	—	
Sahli I., 378.	8	18	0	8	0	25	0	2	12	6	—	—	—	
Sahli III., 380.	3	9	0	2	1	8	0	0	3	2	—	—	—	
Woelky, 383.	2	5	0	0	0	3	0	0	0	2	—	—	—	
Gocht, 386.	5*	8	0	2	0	5	1*	0	1	5*	—	—	—	* III. 4, not counted in sibships.
Sahli II., 388.	5	9	0	6	0	16	0	1	8	5	—	—	—	
"Mampel," 389.	13	37	0	28	1	52	0	12	18	12	1*	—	—	* V. 25, married his cousin V. 19, a conductor.
Rieken, 390.	2	6	0	3	0	8	1	0	3	1	—	—	—	
Sadler, 391.	6	16	1*	12	7	10	0	3	5	5	—	—	—	* II. 6, not counted.
Buel, 398.	3	9	0	0	?	4	0	0	3	2	—	—	—	
Eve and Bidwell, 399.	2	5	0	7	0	2	2	0	3	1*	—	—	—	* II. 10, alleged to be affected.
Schneider-Cramer, 400.	2	4	0	1	0	6	0	0	1	1	—	—	—	
Burger, 401.	5	9	0	6	0	15	1	2	6	7	—	—	—	* III. 3, alleged to be affected.
Stahel I., 407.	13	24	0	0	0	22	0	6	12	12	1*	—	—	* III. 19.
Wachsmuth, 409.	3*	4	0	3	1	2	0*	1	1	1	—	—	1†	* IV. 1, and IV. 2, not in sibships. † IV. 6.
Brook, 410.	3	4	0	4	0	6	0	0	6	2	—	—	—	
Fischer, H., 416.	3	4*	0	1	0	1++	0†	0	0	3‡	—	—	—	* II. 3, not included. † III. 2, not in sibships. ‡ III. 2, alleged female bleeder.
Albers, 418.	2	4	0	8	0	5	0	0	2	1	—	—	—	
Assmann, 423.	4	8	0	2	9	6	0	0	3	3	—	—	—	
Fischer, M., 426.	6	11	1*	13	0	20	2†	4	5	6‡	—	—§	—	* III. 11, and II. 2, not in sibships. See text of pedigree for II. 2. † III. 10, and III. 15, not in sibships. ‡ III. 10, and III. 15, alleged bleeders. § II. 2, not in sibship.
Fildes, 427.	3	4	0	7	0	13	0	1	5	2	—	—	—	
Dunn I., 433.	1	7*	0	1	0	5*	0	1	1	1	—	—		* IV. 1, and IV. 2, not in sibship.
Dunn IV., 436.	5	11	0	7	7	2	0	3	1	3	1*	—	2†	* II. 10. † IV. 12, and V. 2.
Kriner, 440.	3	11	0	1	0	8	0	1	3	2	—	—	—	

Weil II., 444.	2	6	0	5	6	11	0	0	1	1	—	—	—
„ III., 445.	5	11*	0	4	0	4	0	3	1	5	—	1*	—
McCormac II., 450.	2	5	0	3	0	9	0	0	1	1	—	—	—
Stochr, 454.	2	5	0	1	1	7	0	0	5	1	—	—	—
Thompson, 460.	2	5	0	1	0	0	0	0	0	2	—	—	—
Murray, 461.	1	4*	0	2	0	4	0	1	1	0	—	—	—
King II., 475.	5	17	0	6	0	16	0	1	6	4	—	—	—
Wright I., 495.	5	11	0	0	0	8	4*	1	7	4	—	—	—
„ II., 497.	6	12*	0	12	0	22	0	1	6	6	—	—	—
Groves I., 500.	4	6	0	2	0	5	0	1	3	3	—	—	—
„ II., 502.	3	6	0	5	0	14	0	0	1	3	—	—	—
Grusche, 508.	4	7	0	4	5	7	0	3	4	3	—	—	—
Wright III., 521.	2	3	0	11	3	11	0	0	1	1	—	—	—
Rachford, 542.	2	3	0	6	0	3	0	0	1	1	—	—	—
Wilmot, 552.	2	3	0	1	0	2	0	0	0	2	—	—	—
Waterhouse, 560.	2	4	0	1	0	4	0	0	1	1	—	—	—
Nettleship, 561.	2	6	0	5	0	5	0	3	3	1	—	—	—
Muir I., 581.	12	26*	0*	22 +	7	22	0	1 +	10	11†	1‡	—*	—
Hirsch, 586.	2	3	0	5	?	3	0	0	1	1	—	—	—
Totals.....	189	406	2	236	56	453	11	61	171	160	6	1	4
Cantani, 374.	2	4	0	0	6?	1	2	1	1	1	—	—	—
Vieli I., 375.	1	2	0	1	0	1	0	0	0	0	—	—	—
„ II., 376.	1	4	0	0	?	0	0	0	0	0	—	—	—
Hamilton, 397.	1	4	1	1	1	0	0	0	0	0	—	—	—
Benedict, 402.	2	4	1*	0	?	1	0	0	1	1	—	—	—
Bowlby I., 403.	1	3	0	1	0	4	0	0	0	0	—	—	—
Du Bois, 405.	1	4	0	1	0	1	0	0	0	0	—	—	—
Davis, 406.	2	3	0	0	0	1 +	0	0	1	1	—	—	—
Hay, 408.	10	20*	—*	2	0	8	0	3	5	11	—	3*	—
Stahel II., 413.	2	3	0	1	0	3	0	0	1	2	—	—	—
„ III., 414.	1	1	0	0	0	0	0	0	0	0	—	—	—
„ IV., 415.	1	1	0	1	0	2	0	0	1	0	—	—	—
Brigstocke, 439.	1	3	0	2	0	4	0	0	0	0	—	—	—
Weil I., 443.	2	2	0	1	?	2	0	0	1	1	—	—	—
Milne, 449.	1	3	0	3	0	3	0	0	0	0	—	—	—
Wilson, Lane, 463.	1	5	0	0	0	+	0	0	0	0	—	—	—
Hopff, 476.	1	4	0	0	0	2	0	0	0	0	—	—	—
Klein, 479.	3	3	0	1++	?	0	0	0	0	3	—	—	—
Grandidier V., 484.	4	4	0	0	?	2	0	0	2	4	—	—	—
„ VI., 485.	1	3	0	0	0	3	0	0	1	0	—	—	—
Kinnicutt, 494.	1	1	0	2	0	1	0	0	0	0	—	—	—
E. B. Miles, 496.	1	3	0	1	0	4	0	3	0	0	—	—	—
Milner, 523.	2	3	0	0	?	1	0	0	1	1	—	—	—
Oliver II., 536.	3	3	0	0	?	1	1	0	2	2*	—	—	—
V. d. Scheer I., 513.	1	3	0	0	0	2	0	0	0	0	—	—	—
Greig Smith I., 574.	1	3	0	2	0	3	0	0	0	0	—	—	—
Holloway, 576.	1	3	0	0	0	3	0	0	0	0	—	—	—
Muir II., 582.	1	5	0	3	0	2	0	0	0	0	—	—	—
Consbruch, 583.	2	3	0	+	0	1 +	0	0	1	1	—	—	—
Jacob, 585.	1	3	0	2	4 or 5	1	0	0	0	0	—	—	—
Gettings, 603.	1	5	0	1	0	5	0	0	0	0	—	—	—

* II. 2, see text.

* III. 4, not in sibship.

* IV. 4, not in sibship.

* I. 3, „ „

* I. 1, not in sibship.
† II. 6, not counted.
‡ III. 6,

* I. 3, II. 4, II. 5, not included.

* See account of pedigree. I. 1, not in sibships.

* III. 2, alleged female bleeder.

necessary to point out that, into work apparently so simple as compiling a family record, grave error may enter. This has been brought home to us in the cases of Pedigrees Nos. 474 and 512, in which one and the same family history has been reported by two different observers within a few years of each other, but with very diverging results. It therefore appears to us to be essential that only those cases which bear the stamp of accurate observation and detail should be included in any attempt to estimate the characteristics of this or any other disease.

The method on which we proceeded was first of all to make accurate abstracts of all known writings on the subject. These abstracts were thereupon divided into two groups, viz. (1) those containing data illustrating heredity in this disease, and (2) those which did not, or did not refer to haemophilia at all. Group I constitutes all the pedigrees appended to this paper. To the most casual observer, however, it will at once be apparent that these pedigrees are not of equal value. In order to study the hereditary principles at work it became necessary to make a further selection, and for this purpose we reconsidered all the data in order to select the cases which showed evidence of having been most carefully studied and recorded, apart altogether from the size of the affected families or the method of transmission therein. In this way, Group I underwent a subdivision into two subgroups, the first containing pedigrees which in our opinion referred to genuine haemophilia, and the second, pedigrees in which the diagnosis of haemophilia was doubtful, improbable, or inadmissible. Thus of the 235 drawn pedigrees 75 only are included in Subgroup I, as referring to haemophilia, and even among these there are 31 which we have considered it necessary to separate from the remaining 44 on the grounds that their inclusion would introduce a high percentage of error into any calculations based upon them, since the accounts given of them rest too much on the statements of the authors rather than upon evidence which can be independently gauged.

We have therefore at hand 44 cases from which it is probable that definite conclusions can be drawn, and our subsequent remarks refer entirely to these 44 cases, although the complete list of 75 cases is retained in Table II.

Modes of Transmission. A glance at the table shows that, out of a total of 171 recorded instances of transmission, 160 conform to the so-called "law of Nasse," that the disease is transmitted by the unaffected female—the "conductor." The remaining 11 apparent exceptions to this rule require some comment. It will be seen that in 7 of these 11 instances transmission was apparently through the alleged *affected* male, the remainder being through unaffected males. The alleged case in the Mampel family, Pedigree No. 389, V. 25, is not a valid exception in so far that this man was married to his cousin, V. 19, who was a conductor. In connection with the remaining instances we think it necessary to point out that the following considerations may deprive them of some importance as exceptions to the rule. First, a number of haemophilic families occur among people socially and geographically isolated. This state of affairs leads to intermarriage, so that a woman presumably normal, but in reality a "conductor," may marry a man in a bleeder family and be responsible for his bleeder sons. In some cases such intermarriages are admitted, but

there is reason to believe that they may be much more common than is supposed. The change of name in the females makes investigations of this nature extremely difficult. Secondly, males in the earlier generations of a pedigree are wrongly diagnosed as bleeders with the result that bleeder descendants are presumed to have inherited the disease from such males, while in reality the line of inheritance passed out of the family through the maternal side. Cases of this kind are possibly not uncommon. It may readily be allowed that when haemophilia occurs in a family and is found to be passed from generation to generation, the subject becomes a matter for comment in that family and the origin of the disease is fully discussed. It is then recollected by the older members of the family that a grandfather or father died of haemorrhage from the bowels or similar condition, and he is included in the list of bleeders and presumed to be the cause of the family liability. It must be pointed out that these instances of propagation through the male belong to the past. In our series of 44 cases only one instance, viz. Stahel I., Pedigree No. 407, III. 20, was living at the time of the author's publication, all the others having been dead generations before and known only by repute. Transmission in any way other than through unaffected females has not been recorded in any recent publication.

Returning to the specific exceptions in our list it is conceivable that the alleged transmission through males in the Tenna family, in Stahel I., in Max Fischer's, and in Muir I. pedigrees is explicable on the assumption that the respective wives were conductors, all these families being large and more or less geographically isolated. In the Tenna and Stahel's families in particular, intermarriage may have been more frequent than stated, as it will be noted the same names constantly recur from generation to generation. In Muir's family the author expressly affirmed that intermarriage was common, a statement which we have personally confirmed. The instance alleged in Wachsmuth's family cannot be accepted with certainty, as the boy V. 1, was not in our opinion an undoubted bleeder. In Dunn's fourth family (Pedigree No. 436) it is alleged that propagation took place through one affected male II. 10, and two normal males IV. 12, and V. 2. Dunn's account of IV. 12, and V. 2, is extremely brief, the only evidence that V. 2, was not a bleeder being that he had operated upon him for necrosis of the sternum which was not attended by unusual haemorrhage. Such families cannot be compared in point of accuracy with those recorded by Lossen, Sadler, Stahel, and others which the reader may notice for himself. Finally, in connection with Weil III., Pedigree No. 445, in which propagation is alleged through II. 2, there is no evidence that either he or his two brothers were bleeders except the mere statement.

From a very careful consideration of all the facts we do not feel convinced that we are justified in concluding that haemophilia is capable of being propagated through the male. It has frequently been stated that haemophilia can "skip" one generation and appear in the next, being propagated by "conductors." We take this to mean that a sister of a bleeder may have a family, which *includes males*, although among them there are no bleeders, and that the daughters of this family may have bleeders in their families in turn. It cannot be said, in regard to haemophilia, that a

generation is "skipped" unless that generation includes *unaffected* males. "Skipping" as defined above is to be found among our 44 selected pedigrees only in the Tenna (Pedigree No. 373), Burger (Pedigree No. 401), Sahli III. (Pedigree No. 414), and Dunn IV. (Pedigree No. 436) families. It is however doubtful whether these instances are of much value and they must be studied in conjunction with the remarks made in the text and under the respective pedigrees.

Fertility in Bleeder Families. Wachsmuth (*l.c.* p. 35) was the first to note the unusual fertility of the women in bleeder families, and, according to his figures, 114 children were born in 12 families, giving an average of 9·5 per family. Grandidier (*l.c.* p. 99) also dealt with this question and placed the average in 17 families at 9·6. In the series of 44 cases collected above there are 189 bleeder sibships averaging 6·1 children per family, a considerable reduction on the figures of Wachsmuth and Grandidier, but still above the average. With regard to the frequence with which a female in a haemophilic family will transmit the disease it is perhaps not yet possible to speak with certainty. Some rough approximation may however be obtained. If, from our collection of 44 families, those are examined which bear upon this subject it will be found that of 142 females who had children, 125 had sons. Of these 125 mothers, 37 (29 °/₀) escaped having bleeders. Thus it might be said in a general way that one girl, in every three or four, who has sons at all will have them free from haemophilia. Similarly, it will be found that 39 (31 °/₀) of the 125 females had no normal sons. In other words, what sons there were were bleeders, i.e. the boys of one out of every three or four women of bleeder families, are all bleeders. In the remaining 40 °/₀ the families will contain both bleeders and non-bleeders. It must however be pointed out that such figures are only approximate inasmuch as several of the families were, in all probability, not completed at the time of the authors' publications.

A study of the figures in Table II. also shows that the sex ratio is the subject of great abnormalities in haemophilic stocks. Thus in our 44 families it will be seen that there are 644 males, 464 females and 56 individuals whose sex is not stated. If we assume that *none* of the 56 individuals were females the ratio of males to females is 1387 : 1000. If we assume that *all* the 56 individuals, whose sex is not stated, were females we still have an unheard-of ratio of males to females, viz. 1246 : 1000. Between the years 1838—1908 the Registrar General for England and Wales gives the ratio of males to females as 1041 : 1000 in these countries. On the basis of this normal sex ratio there should be 611 females to the 644 males in our pedigrees, but as there are only 464 females there are at least 99 females unaccounted for even if we assume that all the 56 individuals, whose sex is unstated, were females. If we keep to the actual number of females recorded, viz. 464, the loss in females is in fact 155. The explanation of this abnormity is not apparent. It is due either to the fact that the conductors in bleeder families tend to produce an excess of males, or that the pedigrees which we have selected on account of their clinical excellence are seriously defective in so far that a large number of females have been omitted. This question will be cleared up only when new data have been collected. Professor

Karl Pearson has also pointed out to us that the marriage rate of *male* bleeders in bleeder families is very low as compared with similar rates in the females, being 9·6 °/₀ : 36·8 °/₀.

Geographical Distribution and Racial Disposition. In the earlier literature on haemophilia the question of the geographical distribution of the cases was discussed at length and was considered to be a matter of great moment. We are, however, in agreement with Virchow and with Dr Wickham Legg[331], who pointed out long ago (1872) "that the number of recorded cases depends not upon the frequency of haemophilia in a given country, but upon the previous education of the medical men, and the interest they take in the disease." Hirsch, in his monumental *Handbuch der historisch-geographischen Pathologie*, made no reference to the disease. The innumerable records of bleeders and alleged bleeders from Germany, first in the Rhine country and subsequently in Prussia, can be traced especially to the influences of Nasse and Schönlein. Nasse was director of the medical clinics, first in Halle and later in Bonn, and had a large number of students under him. His attention had been particularly directed to the disease from his acquaintance with Consbruch at Bielefeld, where he was born in 1778. Similarly J. F. Schönlein, the first clinician of his day, drew very large audiences round him, first in Würzburg (1817—1833) and later in Berlin (1840—1859), and through his influence many cases came to be reported either in inaugural dissertations or in medical journals. In this way the knowledge of haemophilia became widely disseminated in Germany, and medical men were on the outlook for cases which in due time were published. The same remarks apply to other countries. Cases are reported; they create a general interest and for a time there is a flux of publications both good and bad. The point need not be laboured. It goes on at the present time with every new advance in medicine. There would, however, appear to be something in the view that haemophilia is largely confined to the Teutonic race, the vast bulk of genuine cases having been reported from Germany, Switzerland, England and the United States. Although an extensive literature on haemophilia exists in French, a careful study of the publications has convinced us that a very large number of these cases have little or no claim to the title of haemophilia. In this we are supported by so learned an observer as Hayem[542], who says "Cette maladie singulière est extremement rare en France et pour ma part je n'ai pas encore eu l'occasion d'en observer un seul exemple parfaitement authentique, c'est à dire se rattachant à la forme constitutionnelle héréditaire" (p. 999). Strange to say, however, Hayem himself, two years later[569], recorded a case of epistaxis and menorrhagia after coitus, although he adds "cette observation presente une lacune; l'examen de l'uterus n'a pas été fait" (p. 392). The most typical French case, recorded by Delmas[298] and Simon[350], refers to a French-American family from Louisiana, temporarily resident in Bordeaux. The cases described as haemophilia in the Italian, Spanish and Greek languages are not suggestive of accurate diagnosis. The Russian literature which has been examined for us by Professor Sirotinin of St Petersburg and Dr Blumenthal of Moscow presents, according to them, little evidence of true haemophilia. On the authority of Sirotinin we have

admitted only the case of Nesterovski. The Hungarian literature, which was searched for us by Professor Otto Pertik of Budapest, possesses no striking instances of the disease. From Finland the reported cases do not, in the majority of cases, appear typical. In the Scandinavian countries, Professors Karl Petrén, of Lund, and Faber, of Copenhagen, inform us that the disease in typical form is little known. Of foreign countries in the Orient only one example has been recorded, viz. that by Heymann[242] in a native family living in Palembang in Sumatra. Heymann's posthumous publication is, however, so fragmentary as to be almost devoid of value, as it is very uncertain whether he was writing with first-hand knowledge or not. In Japan it appears to be as yet unrecorded. Writing to us in 1909, Professor Aoyama, the distinguished clinician of Tokyo, says, "ich habe bis jetzt in Japan Hämophilie mit Heredität nicht beobachtet." The existence of a long strain of bleeders in South Africa among the Cape Dutch, as recorded by Muir, is traceable without doubt to their European descent. In North America all the classical cases were reported in the families of European settlers. The existence of the disease among negroes or negro crosses has been affirmed on three occasions, viz. by W. R. Steiner[733], Buck[713], and Hadlock[344]. In our opinion it is doubtful whether any of these writers were dealing with genuine cases. Thus Buck's publication, one of fifteen lines, refers to two instances of epistaxis in a male and a female, in whom no genetic relationship is stated. Hadlock's case was that of a boy, aged seven, who died after tooth extraction. An uncle had cut himself with a scythe and died in consequence, and the boy's father received a slight scratch from a briar and bled to death. It was stated, without actual data, that many of the members of the family had died from haemorrhage from slight wounds. The case of W. R. Steiner is given in Pedigree No. 504. Grandidier, Rothschild and others have referred to the excessive frequence of haemophilia in the Jewish race, the latter writer, on the basis of uncertain data and unusual methods of calculation, asserting that haemophilia in German Jews is eight times as frequent as among the other races in Germany[1]. We can find no support for this statement and indeed consider it to be incorrect. Of modern well-reported and typical instances of the disease the records in Jews are few (*M^c^Lane-Hamilton-Fry's Case*, Pedigree No. 397).

The question may be asked, whether the sparsity of haemophilic records is due to the rarity of the disease or to other causes. There would appear to be little doubt that the disease is rare. In the great clinic at the London Hospital years pass without the admission of a single case, in spite of the fact that over 10 per cent. of the total admissions are Jews, in whom the disease is alleged to be more frequent than in other races. Thus from 1900—1909 the total number of persons admitted into the hospital was 137,676, of which 15,600 were strict Jews. In this period, however, we can only find two cases of haemophilia in brothers (Pedigree No. 427).

It is probable that little importance can be attached to the records published

[1] P. Stanley Blaker's Case (*Brit. Med. Journal*, 1904, Vol. i. p. 189) was that of a female foreign Jewish child; it was said to have had two paternal aunts and a grandmother haemophilics, but the reporter states that the notes were not sufficient to make the case of real value.

by the Registrar General, as, prior to 1901, no distinction was made between purpura and haemophilia, the latter term including haemorrhagic diathesis. The figures published by the Registrar General for England and Wales since 1901 are as follows:

	1901	1902	1903	1904	1905	1906	1907	1908
Total no. of deaths from haemophilia at all ages	79	88	80	86	87	84	103	81
Total no. of deaths from haemophilia in males at all ages ...	49	59	49	59	47	46	57	57
,, ,, ,, females ,, ...	30	29	31	27	40	38	46	24
Deaths in males, from haemophilia, per million living.....	3	4	3	4	3	3	3	3
,, females ,, ,, ,, ...	2	2	2	2	2	3	3	1

If these figures are correct they represent a much higher percentage of haemophilic cases in females than has ever been alleged by any writer on this disease. The probability is that many different types of fatal haemorrhage are included in the death returns, and that the above table does not at all represent the degree of haemophilia, which is much rarer than these figures would suggest. Nevertheless it would appear that instances of the disease must be more frequent than hospital records would lead one to suppose. The disease runs in families, and in the course of time an attitude of fatalism is developed towards the resources of medicine, so that in all probability many cases do not come to the notice of the medical man at all. The barbarous treatment to which many bleeders have been subjected in the well-meant efforts of the physician or surgeon to stanch the haemorrhages must have a terrifying effect and leave an ineffaceable memory.

Another point which conduces to concealment is the fact that the women being conductors of the disease may have their chances of matrimony considerably reduced if their anomaly were widely known. This has been strikingly shown in the cases which we ourselves have investigated. Again, cases occur in families of high social standing or even of royal lineage, so that the histories are known only to the few and cannot be published. We seem justified in asserting therefore, that although a rare disease, haemophilia is probably more common than one would be led to infer from the number of published records, and we have reason to believe that there is more genetic connection between different families than is generally supposed. The fact that the females, who are the conductors, change their names on marriage, seriously complicates the efforts of investigators to trace out the ascent of individual cases of haemophilia.

Diagnosis. The diagnosis of haemophilia is of importance, particularly from the point of view of prognosis, and it should present little difficulty. The question is not likely to arise except in a case of immoderate and uncontrollable haemorrhage, or in the case of swelling of a joint. In connection with all types of haemorrhage, especially epistaxis, haematuria and haematemesis, it is necessary that every local cause must be looked for and if possible excluded. These forms of bleeding are so common that it is easy to construct a pedigree showing an inherited "haemorrhagic

diathesis," and such families are extremely common in the literature, see Pedigree No. 607. Further, it is essential to establish the fact that throughout life the individual has been more or less subject to haemorrhages from various parts of the body. No solitary haemorrhage, however inexplicable, should in our opinion be regarded as haemophilia: it is necessary to show that the individual has been repeatedly attacked, if not from birth, from infancy. C. Ray[119] emphasised this point long ago, at a time when there was a tendency to label all vehement isolated haemorrhages as haemophilia. He affirmed that "great haemorrhage most generally depends upon some local accidental cause entirely unconnected with any haemorrhagic diathesis. It is essential," he added, "that corresponding circumstances in the biography of the individual should be advanced, such as the result of the occasional incised and other wounds that are common to most in ordinary vocations." In more recent times Verneuil[486] (1884) has again insisted on the thorough examination of the various organs before the diagnosis of haemophilia is made. Thus he quotes the case of a man who had repeated and grave haemorrhages, explicable only on the assumption of haemophilia, but where careful examination revealed a splenic new growth to which the haemorrhages were no doubt due. In another case, a female, aged 40, a number of doctors could find nothing to account for the perpetual uterine haemorrhages, until an osteosarcoma of the pelvis became of such a size as to lead to a diagnosis. Again it is not enough to find the bleeding constantly recurring in one position, as for example epistaxis, even though this may be inherited, for such haemorrhage may be due to some local inherited cause, as in the case of multiple family telangiectases (Chauffard[639], Osler, Hanes). It has been held by Wilhelm Koch[546] that haemophilia is merely a manifestation of scurvy, but his arguments appear singularly inconclusive, being based chiefly on the occurrence of an haemorrhage into the calf muscles in a bleeder. In fact, on comparison, the two diseases appear to have little in common.

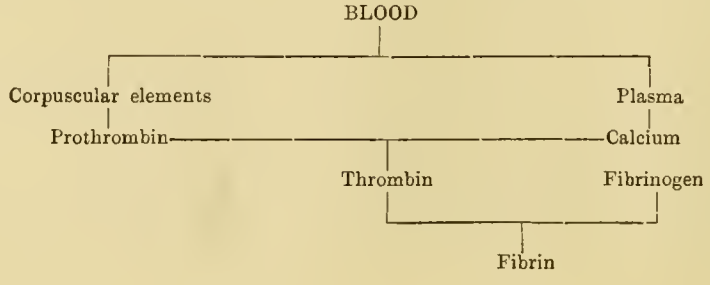
In the case of children, when as yet there has been no opportunity to observe a chronicity in the disease, a diagnosis may be more difficult. Umbilical bleeding, *unless at birth* and in the presence of *inheritance*, should receive no consideration. In children within the first week of life, and rarely after the second, a haemorrhagic condition—the haemorrhagic disease of new-born children—has been described and frequently leads to death. The attack is usually sudden, acute, and characterised by profuse and manifold haemorrhages. The pathology is unknown, but syphilis or other infection probably lies at its basis. Scurvy rickets would hardly be mistaken for haemophilia. The history of the case, the condition of the patient, the sub-periosteal haemorrhages, the spongy gums, and orbital extravasations, would be distinctive.

In connection with the diagnosis of haemophilia little mention has been made of the question of inheritance. If this is an inherited disease, is the demonstration of inheritance essential to support the diagnosis or may it be considered to have arisen *de novo* in a particular case? Apart from the suggestion of Wright that the origin of the disease may be the accidental conjunction of two persons whose blood in each case is in the condition described by him as occurring in haemophilia, the

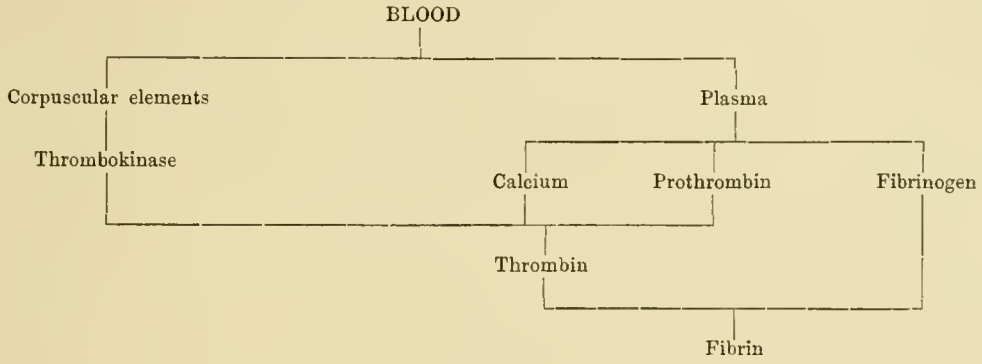
introduction of the "de novo" concept is based upon the inability of writers to demonstrate the line of inheritance in their cases. The instances of probable haemophilia without demonstrated inheritance are comparatively few, and would undoubtedly be still fewer if the authors had had sufficient time, interest or perseverance to investigate beneath what might be immediately apparent on the surface. For it must be remembered that the other bleeders necessary to establish the inheritance of the condition may not be members of the same family. Indeed it is not unlikely that they are collaterals. However, in the one "de novo' case (Pedigree No. 603) hitherto unpublished, in which this question has been particularly investigated, Dr Gettings was unable to find any instances of the disease among the collaterals although these were widely enquired into over a number of generations.

Pathology. A number of cases of haemophilia, or alleged haemophilia, have been subjected to *post mortem* examination (Grandidier, Elsaesser, Durham (Moxon), Assmann, Resal, Wickham Legg), and have revealed nothing of fundamental importance, the chief findings being an unnatural thinness of the walls of the vessels and general hypoplasia of the vascular system (Blagden[20] (p. 227), James Wilson[32] (p. 412), Burnes[116] (p. 405), Lemp[236] (*p. m.* by Virchow, p. 17), Gavoy[251], P. Kidd[390]). This led to the supposition that haemophilia is due to some abnormality in the development of the cardio-vascular system leading to excessive delicacy or brittleness of the vessels. Such reports are, however, few and belong to a period which is now somewhat remote. They cannot be accepted as of importance at the present time. Immermann[401] has enunciated an hypothesis in which four factors are alleged to account for the disease, viz. (1) hypoplasia of the vessels leading to a diminution of their capacity, (2) hypertrophy of the heart with consequent increase in the blood pressure, (3) an increase in the total quantity of the circulating blood, and (4) an abnormal action of the vaso-motor system. These four factors together or separately produce a condition of plethora and consequent bleeding. The assumptions on which Immermann's hypothesis is founded are not supported by adequate evidence, while numerous observations are opposed to them. In recent years attempts have been made to explain the nature of haemophilia on the supposition that there is some defect in the coagulability in the blood. In the earliest accounts it was noted that the shed blood did not clot, or only imperfectly, after an abnormally long interval, but it was not till A. E. Wright devised his instrument for measuring the time taken during coagulation that the question was placed on a firmer basis. He noted[579, 606, 649] a constant increase in the coagulation time of haemophilic as compared with normal blood, but numerous observers dissociated themselves from this view. The difference of opinion arose owing to the fact, pointed out by Sahli[782], that it is essential to measure the coagulation time during a normal *inter-haemorrhagic* period, and not during or immediately after a severe haemorrhage. Wright's and Sahli's observations have been extensively confirmed, and might be definitely accepted as correct, were it not for the fact that the methods in general use for measuring the coagulation time have been subjected to considerable criticism. Even assuming that the coagulability of the blood is defective, unanimity has not been reached with regard to the precise

nature of the defect. Indeed, until the process of blood coagulation under normal circumstances has been clearly elucidated, it will be impossible to speak with accuracy on the exact nature of the defect in the blood of haemophilics. Before dealing with the views at present held, it is necessary to consider briefly the present state of our knowledge in regard to the coagulation of the blood. When a wound is inflicted, the blood channels, which everywhere abound for the nutrition of the tissues, must necessarily be divided and give rise to an escape of blood. Were the blood an inert fluid there would be nothing to prevent a continuous flow, and the wounded individual would inevitably bleed to death. Blood, however, is endowed with the power of resolving itself into a solid felted mass, and this mass, the clot, effectually seals up the wound and thus prevents a fatal issue. The broad outlines of the chemistry of blood clotting may now be said to be established as follows. Dissolved in the blood fluid or plasma is a protein substance, fibrinogen, which is capable of being acted upon by another substance, thrombin, or fibrin-ferment, with the result that it is converted into fibrin, an insoluble protein substance which forms the meshwork of the clot. Thrombin is not found in the blood while still in the vessels; it only becomes apparent when the blood is shed. The essence of coagulation, therefore, is the conversion of dissolved fibrinogen by thrombin into an insoluble stringy material, fibrin. This is not a vital action, as it can readily be demonstrated *in vitro*. Pure solutions of fibrinogen can be made to clot typically by the addition of pure solutions of thrombin (Rettger[902]). The question next arises as to the origin of thrombin, and at this point opinions differ. It has long been known that the presence of soluble calcium salts in the blood is in some way essential for the act of coagulation (Arthus and Pagès[550]). If these salts are removed from the solution by the addition of a soluble oxalate the blood will no longer clot. If however calcium salts are then added to the oxalated blood, clotting will immediately proceed. The *rôle* of the calcium salts was shown by Hammarsten to be connected with the production of thrombin, and not with the formation of fibrin from fibrinogen by thrombin. Since therefore thrombin is not present as such in the circulating blood, and is not produced in the absence of calcium, it is assumed that an antecedent substance, designated prothrombin or thrombogen, exists, and that this antecedent substance combines with calcium to form thrombin. Prothrombin is considered by some to be present preformed in the blood, but this is not accepted by Howell, who asserts that the leucocytes or platelets give rise to it after the blood is shed. According to him (*A Text-book of Physiology*, 1909, p. 447) the scheme of the coagulation of the blood may be represented thus:



This concept of blood coagulation has not however been allowed to remain in such a simple form. The knowledge is old that extracts of various tissues of the body contain substances which induce or facilitate clotting of blood or pure solutions of fibrinogen. Such substances were called by Alexander Schmidt "zymoplastic," and subsequent writers have given them other names, such as "cytozym" (Fuld-Spiro), "coagulin" (Loeb), "thrombokinase" (Morawitz), and attempts have been made to determine their exact *rôle* in the phenomenon of clotting. Thus Morawitz considers that thrombokinase is the essential connecting link between the calcium salts and the prothrombin in the formation of thrombin. He supposes that the kinase is not present in the circulating blood, but is derived from the leucocytes or blood platelets after the blood is shed, while the prothrombin is normally present dissolved in the blood plasma. According to his view, and this is the one generally accepted at present, the phenomenon of blood coagulation proceeds as follows:



Whatever view is taken of the nature of the assistance to coagulation conferred by these zymoplastic substances, it cannot be accepted as proved that such assistance is essential. Although the prevailing view is probably represented by the theory of Morawitz, the recent work of Rettger would seem to throw doubt on the necessity for introducing a "kinase" to explain the phenomenon of coagulation. With regard to haemophilia, the majority of recent workers following Morawitz and Sahli appear to attribute the deficiency in blood coagulation to absence or inadequate production of zymoplastic substances, either by the tissues (Sahli), or the corpuscles (Morawitz), or both. Wright showed that a slight admixture of tissue fluids with the escaping blood increased its clotting power, and Sahli presumed that in haemophilia this property of the tissue fluids was lacking. Émile Weil also claimed that the clotting of haemophilic blood could be hastened by the addition of fresh serum, a method already suggested by Rieken[60] so long ago as 1829. Morawitz, being of the opinion that the zymoplastic substance thrombokinase was derived from the corpuscular elements, presumed some defect in these structures. In this connection the observations of Wright on the number of the white blood corpuscles in haemophilia are of great interest. He showed, and his statements have been verified by Sahli, that as a rule bleeders tend to exhibit a leucopenia, especially with regard to the neutrophilic varieties, and, further, that in other conditions associated with leucopenia there

is a tendency for the coagulation time to be prolonged. These observations therefore give some support for the theory of Morawitz. Wright, however, holds himself aloof from any theory of blood coagulation, but presents the results of his numerous experiments as a working hypothesis. According to him the coagulability of the blood in haemophilia is decreased (1) owing to some defect in calcium metabolism, which may be overcome by the therapeutic administration of calcium salts, substances for which bleeders have frequently been observed to have had an abnormal or even imperious craving, (2) owing to diminution in the number of leucocytes as in other leucopenic conditions, (3) owing to a lack of fibrinoplastic substances in the tissue fluids which mix with the escaping blood, and, lastly, (4) owing to a decreased content of CO_2 in the blood, an increased content being shown to be associated with increased coagulability. While therefore the ultimate pathology of haemophilia is involved in obscurity there appears to be sufficient reason to assume that there is some inherent defect in the process of coagulation. From the numerous histories of cases it would appear that this inherent defect is not of constant magnitude, as we repeatedly read of the *periodic* tendency to bleeding. After a haemorrhage has taken place, the bleeder may not behave differently from a normal individual, even while wounded. This might be due to the insufficient production of some factor necessary for coagulation or the factor might be produced only to be inhibited by some other substance after it has been formed. In future investigations the possible production of some anti-substance should be borne in mind. In any case advance can be looked for only when *genuine* cases of haemophilia have been studied, as in the instances observed by Wright and Sahli and a few others. The introduction of "sporadic" cases into the question, as has been done especially by French observers, complicates and retards, in our opinion, the solution of the real nature of the disease.

Literature. To anyone who has attempted to examine, at first hand, the literature of haemophilia it must at once be apparent that a no inconsiderable part of it is buried away in ancient journals or inaugural dissertations no longer procurable even in the great medical libraries. Indeed, some of the earlier publications would seem to be known only through abstracts, and being so, have been reported incompletely and inaccurately. It has been our endeavour to subject the literature on this disease to a searching and critical enquiry. That we have obtained practically the whole of the literature is shown by the fact that there remain only some half-dozen papers which have eluded our search. For the earlier references we are indebted to the monographs of Wachsmuth, Grandidier and Wickham Legg, although incorrect citations in each of these works have been the cause of considerable labour. From the publication of Grandidier's monograph down to the present time we have followed the exhaustive *Index Medicus* and the two series of the *Catalogue of the Surgeon General's Library* in Washington. Our experience shows that these are the only accurate works of reference on the subject of haemophilia. As far as possible we first exhausted the resources of the great libraries in England, particularly those of the Royal College of Surgeons and the British Museum. In Paris, extensive use was made of the Bibliothèque de la Faculté de Médecine, the Bibliothèque de l'Académie

de Médecine, and the Bibliothèque Nationale. For the remaining papers, unobtainable in these libraries, we have had the abundant cooperation of distinguished colleagues in foreign countries, and it is a most pleasing duty gratefully to acknowledge the unusual generosity manifested towards us in the course of our enquiries. Time and again they have, at a great expenditure of time, made very complete analyses of haemophilic papers, and in not a few instances have supplied us with new and unpublished data which are incorporated in this paper. In one instance only have our enquiries failed to elicit a courteous reply. It is difficult to thank every one who has assisted us in our work, but we should particularly like to record the help we have received from the following: Professors Muchin (Warsaw), Kraus, and Bartel (Vienna), Maixner (Prag), Ehrlich (Frankfort), Lochte (Göttingen), Baumgarten (Tübingen), Morgenroth (Berlin), Moritz (Strasburg), Borst (Munich), Minkowski (Breslau), Ribbert (Bonn), Kossel (Giessen), C. Fränkel (Halle), Sahli (Bern), and the late Professor Hermann Lossen, in Heidelberg, who lent us a number of important papers from his private library. In addition to these Dr Seiffert (Leipzig) and Dr Koeniger (Erlangen) have made valuable abstracts for us. The Russian literature was analysed for us by Professor V. N. Sirotinin, of St Petersburg, and Dr Blumenthal, of Moscow. Dr W. Tallquist forwarded us the entire Finnish literature on the disease, while Professors Karl Petrén (Upsala), K. Faber and Salomonsen (Copenhagen), and Harbitz (Christiania) searched the literature of Scandinavia. The Dutch literature was carefully abstracted by Professors Wenckebach (Groningen) and Tendeloo (Leyden), the Hungarian by Professor Pertik (Budapest), the Spanish by Dr Cervera (Madrid), the Italian by Dr Fano, of Milan, the Portuguese by Professor Bettencourt. Inaccessible French papers were ultimately found for us by Professor Sabrazès and Dr Eckenstein of Bordeaux, by Professor Roque of Lyons, and Dr Warden (Paris).

The modern medical literature of North America and Canada is to a large extent inaccessible in this country, many of the publications of smaller medical centres having, it would appear, a fleeting existence and a small circulation. A large part of this literature would have remained untouched but for the trouble taken by Prof. James Ewing and Dr W. H. Park (New York), Drs Homer Wright and Pratt (Boston), Professors W. Moore (St Louis), W. G. MacCallum (Baltimore), Hektoen (Chicago), Rachford (Cincinnati), J. J. R. MacLeod (Cleveland), and Adami (Montreal). Where these failed to obtain in local libraries the publications sought for we had recourse to the great medical libraries of the United States, and in this connection we desire especially to thank Dr F. P. Henry, of the College of Physicians' Library, Philadelphia, and Dr Clement W. Andrews, of the John Crerar Library in Chicago. Lastly, we cannot conclude without a special word of thanks to Dr Robert Fletcher, the principal assistant-librarian in the Surgeon General's Library in Washington, as it was through him that we were ultimately able to gather the remnants of the haemophilic literature unattainable in other places. We are thus able to offer, for the first time, a complete, and we trust accurate, account, of the known data of haemophilia. The new cases which here appear we owe to Professor

Maixner of Prague, Drs J. L. Masters of Indianapolis, Theodore Thompson (London Hospital), J. A. Milne (London), E. Blomart Miles (London), John Thomson (Edinburgh), H. S. Gettings (Walsall, Stafford), F. H. Jacob (Nottingham), and Mr E. Nettleship. Where material alterations or additions to already published records have been made these are noted in the proper place. Having made an extensive enquiry into all the published data, we deemed it advisable to append to each reference a short account of its contents, thus saving future investigators the trouble of searching for material which in the end shows itself to possess no permanent value. It only too often happens that a reference—usually incorrect—is handed on from one writer to another, and particularly so if the original source is inaccessible. There is a crying need for reform and simplification of the ever-expanding bounds of current medical literature, and particularly for the abolition of unimportant dissertations of students and of the transactions of small local medical societies. This question has already roused considerable discussion, and we find ourselves entirely at one with Oppenheimer in his recent demand "Weg mit Dissertationen und Festschriften!"

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sanguis fere quotidie per nares erupit. Eo nati omnes similiter dolebant; filiaque ejus a majore proxima eodem affectu extenuata diem suum obiit. Hujusmodi raro dantur exempla." The case of the drummer "Hay tertiae cohortis praetorianae tympanista" has also been regarded as haemophilia. He bled easily and profusely from trivial wounds and styptics were powerless to effect the arrest. He once wounded his gastrocnemius and had an interstitial haemorrhage which was difficult to stop in spite of the combined knowledge of the surgeons and physicians of St George's Hospital.]

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30. Channing, W.: A case of alarming haemorrhage from the extraction of a tooth. *The New England Journ. of Med. and Surgery.* Boston, 1817, Vol. vi. p. 235. [Male aged 40.]

31. Seeliger: Einige merkwürdige Fälle aus dem Gebiete der praktischen Heilkunde. *Beobachtungen und Abhandlungen aus dem Gebiete der ges. prakt. Heilkunde von Oesterreichische Aerzten.* Wien, 1819, Bd. i. S. 323. [Male aged 70 "aus eines sogennanten blutreichen Familie." During his life lost 1000 pounds of blood from venesection.]

32. Wilson, James: Lectures on the blood and on the anatomy, physiology and surgical pathology of the vascular system. London, 1819, p. 410. [Pedigree No. 463.]

33. Krimer: in Nasse, *vide* Bibl. No. 35, p. 409. [Pedigree No. 440.]

34. Metzger, Johann Daniel: System der gerichtlichen Arzneiwissenschaft, erweitert und berichtigt von W. H. G. Remer. Königsberg und Leipzig, 1820, 5te Auflage, p. 116. [M. says he knew three people affected with haemophilia, two of whom nearly bled to death from the gums.]

35. Nasse: Von einer erblichen Neigung zu tödtlichen Blutungen. *Archiv für medizinische Erfahrung im Gebiete der praktischen Medizin und Staatsarzneikunde, hrsg. von Horn, Nasse und Henke.* Berlin, 1820, Mai-Juni, S. 385. [This is the first complete account of the haemophilic syndrome based on an intimate acquaintance with all the literature published up to 1820. Although the facts of hereditary transmission of haemophilia were well known they were here systematised by Nasse and his statements are frequently referred to in the literature as "Nasse's law." The account of Krimer's Case (q.v.) is to be found on p. 409. Reference is also made (p. 425) to families in which copious haemorrhages may occur in both sexes, and Nasse records the history of a family living in Bonn and affected in this way. The five sons in the family all had copious bleedings from the time of adolescence onwards, while their sister, mother, grandmother and great-grandmother suffered from profuse menstruation and *post partum* haemorrhage, the two latter bleeding to death at the menopause. At this time the mother was also in danger of her life from bleeding but recovered after a visit to the country where secret remedies were given at the suggestion of a local pastor. That Nasse did not consider such cases as coming into the category of haemophilia is shown by his remark that the male is the sex "dem jene zuvor betrachtete erbliche Neigung zu starken Blutungen ausschliesslich zukommt."]

36. Vogel, Samuel Gottlieb: Handbuch der practischen Arzneywissenschaft zum Gebrauche für angehende Aerzte. Stendal, 1820, Theil v. Cap. i. S. 14. [Ancient reference to haemophilia, no original cases.]

37. Hazeltine, Richard: Case of haemorrhage from extracting a tooth. *The New England Journal of Medicine and Surgery and collateral branches of science.* Boston, 1821, new series, Vol. v. p. 240. [Considerable haemorrhage by a male patient following the extraction of a loose bicuspid. Bleeding arrested by pressure of a cork. Patient had bled severely some years previously and a sister "had once suffered the same unpleasant effects from the same cause."]

38. Elsaesser: Geschichte einer Familie von Blutern in Würtemberg. *Journal der practischen Arzneykunde und Wundarzneykunst (Journal der practischen Heilkunde) hrsg. von C. W. Hufeland und E. Osann.* Berlin, 1824, Bd. lviii. Stück 2, S. 89 und 1824, Bd. lix. Stück 3, S. 109. [Pedigree No. 393.]

39. Humphrey, Gideon: An account of an extraordinary predisposition to haemorrhage in a family of children, *The Medical Review and Analectic Journal.* Philadelphia, 1824, Vol. i. p. 278. [Pedigree No. 436, *vide* also T. D. Dunn, Bibl. Nos. 459, 599. We are indebted for an abstract of this very inaccessible paper to Dr F. P. Henry, Librarian of the College of Physicians, Philadelphia.]

40. Keller, Johan: Von der erblichen Anlage zu tödtlichen Blutungen, *Inaug. Abhand.* Würzburg, 1824. [General account of older literature—no original cases.]

41. Nasse: Bluter. *Archiv für Medizinische Erfahrung hrsg. v. Horn, Nasse, Henke.* Berlin, 1824, Bd. ii. S. 120. [Account of case communicated to Nasse by Theinhardt of Wald. See Bibl. No. 42 and Pedigree No. 430.]

42. Theinhardt: in Nasse. Bibl. No. 41. [Pedigree No. 430.]

43. Ulrich: Bluter. *General-Bericht des königl. rheinischen Medizinal-Kollegiums.* Koblenz, 1825, S. 55. [Pedigree No. 498.]

44. Clutterbuck: Haemorrhage. *The Lancet.* London, 1826, Vol. x. p. 97. [Reference to a case in which death from haemorrhage followed amputation of the leg for "tumor albus" of knee. At first, puncture caused great bleeding. It is stated that all the man's children and several other relatives were affected with a tendency to bleed.]

45. Craig, James: Cases of haemorrhoea with hereditary tendency. *The Edinb. Journ. of Med. Sciences.* Edinb., 1826, Vol. II. p. 64. [Male, aged 54, haemorrhage following operation for fistula in ano; his son, aged 19, bled severely after tooth extraction and after wounds with a hoe.]

46. Davis, Theodore: Case of hereditary haemorrhoea. *The Edinburgh Med. and Surg. Journ.* Edinb., 1826, Vol. xxv. p. 291. [Pedigree No. 406.]

47. Murray, Alexander: Cases of haemorrhoea petechialis in individuals related to one another. *The Edinburgh Med. and Surg. Journal.* Edinburgh, 1826, Vol. xxvi. p. 33. [Pedigree No. 461.]

48. [Chelius, M. J.]: Beobachtung einer Bluterfamilie. *Heidelberger klinische Annalen, hrsg. von Proff. Puchelt, Chelius, Nägele.* Heidelberg, 1827, Bd. III. S. 344. [Pedigree No. 389.]

49.* Prechtl: Diss. de haemorrhagiis hereditariis. Wirceb., 1827. [Not found. This reference appears for the first time in Wachsmuth's paper 1849, and in Grandidier's monograph where it is stated that it contains no new observations. Enquiry at the University Library in Würzburg failed to throw any light on this publication which possibly never existed. It is certainly significant that Schliemann in his inaugural dissertation published at Würzburg in 1831 makes no reference to Prechtl's publication, although he gives a fairly extensive bibliography up to that date.]

50. [Andral]: Cited by Burrows in his article "Haemorrhage" in Tweedie's System of practical medicine. London, 1840, Vol. v. p. 6. [Burrows gives the *locus* of publication as *Bull. des sciences méd.* Avril, 1828. We have identified this as *Bulletin des sciences médicales rédigé par M. le doct. De Fermon, troisième section du Bulletin universel, publié sous la direction de M. le baron de Ferussac.* Paris, 1828, Tome III. p. 353, and the so-called "Andral's Case" turns out to be merely a review of the family published by Schreyer (Bibl. No. 54), and we fail to see any grounds for supposing that even the review was written by Andral at all.]

51. Coates, Reynell: Observations on hereditary haemorrhage. *The North American Medical and Surgical Journal.* Philadelphia, 1828, Vol. VI. p. 37. [The original account of T. D. Dunn's fifth family. Pedigree No. 436.]

52. Elsaesser: Geschichte einer Familie von Blutern in Württemberg. *Journal der practischen Arzneykunde und Wundarzneykunst, hrsg. v. C. W. Hufeland und E. Osann.* Berlin, 1828, Bd. LXVII. Stück 5, S. 122. [Pedigree No. 393.]

53. Hopff, F.: Ueber die Haemophilie oder die erbliche Anlage zu tödtlichen Blutungen. *Inaug. Diss.* Würzburg, 1828. [Pedigree No. 476.]

54. Schreyer: Nachricht von zwei Blutern. *Ztschrift für Natur- und Heilkunde.* Dresden und Leipzig, 1828, Bd. v. S. 333. [Pedigree No. 591.]

55. Steinmetz, C.: Beobachtungen einer bedeutenden Hämorrhagie nach dem Ausziehen eines Zahnes, bei erblicher Neigung zu tödtlichen Blutungen. *Magazin für die gesammte Heilkunde hrsg. von Johann Nep. Rust.* Berlin, 1828, Bd. xxvii. S. 375. [Aged male Jew, haemorrhage, from finger pricks, stopped by application of cold, died of some acute pulmonary trouble. His son, aged 65, in youth had haemorrhage after slight injury. His son, again, aged 25, had haemorrhage after tooth extraction and also on other occasions. A sister of the last mentioned had a son who had slight haemorrhage after circumcision.]

56. Beth, Tamme: Specimen medicum inaugurale exhibiens historiam haemorrhagiae insolitae e digitis dextrae manus et simult. haereditariae. Groningae, 1829, 67 pp. [Pedigree No. 572.]

57. Conradi: De morbo maculoso haemorrhagico Werlhofii. *Diss. inaug. medica.* Göttingen, 1829. [Boy aged 12. At the age of 2 he bled from the gums for three days, and at 4 for nine days. At 5 his ankle and foot swelled. Twice his knees became enlarged and painful. Great bruises also occurred on different parts of his body. On one occasion six leeches were applied to his thigh, when the haemorrhage could not be arrested for 11 days. Shortly afterwards he bled for some days from injury to the mucous membrane of the mouth from a crust of bread. His subsequent history could not be ascertained.]

58. Guillemot, P.: Mémoire sur les hémorrhagies utérines après la délivrance. *Archives générales de Médecine.* Paris, 1829, Tome xx. p. 43. [Title explains itself.]

59. Lobstein, J. F.: Traité d'anatomie pathologique. Paris, 1829—33, Tome I. p. 210. [Good general account of haemophilia.]

60. Rieken, Heinrich Christoph: Neue Untersuchungen in Betreff der erblichen Neigung zu tödtlichen Blutungen, hauptsächlich in ätiologischer und therapeutischer Hinsicht mit besonderer Beziehung auf eine Familie von Blutern im Grossherzoglich Oldenburgischen Fürstenthum Birkenfeld. 12°, Frankfort-am-Main, 1829, 136 pp. [Pedigree No. 390.]

61. Schmidtmüller, Jos. Jul.: De haemorrhoea. *Diss. inaug.* Erlangae, 1829. [General account of haemophilia with history of a male aged 24. At 14 bled from cut on finger. Great haemorrhage at the age of 20 from a considerable cut on chin. Six months later profuse bleeding after extraction of molar. A venesection in 1828 bled for five days. No family history of haemophilia.]

62. Thal, R. S.: Observatio haereditariae ad haemorrhagiam a vulneribus levibus dispositionis. *Acta Regiae Soc. med. Havniensis*, 1829, Bd. VII. S. 46. [Male, aged 33, extravasation of urine, circumcision, secondary haemorrhage. Two brothers and one daughter had a tendency to haemorrhage.]

63. Kendrick, John: Haemorrhage from the extraction of a tooth. *The London Med. Gazette.* London, 1830, Vol. v. p. 788. [Same case as Liston, Bibl. No. 107, and Pedigree No. 535.]

64. Schneider (zu Aschersleben): Beitrag zur Geschichte der sogennanten Bluter. *Magazin für die gesammte Heilkunde hrsg. von Johann Nep. Rust.* Berlin, 1830, Bd. xxx. S. 463. [See Cramer, Bibl. No. 87, and Pedigree No. 400.]

65. Hughes, James N.: Case of hereditary haemorrhagic tendency, with remarks. *The Transylvania Journal of Medicine and the associate sciencces.* Lexington, Ky., 1831, Vol. IV. p. 518. [Pedigree No. 532.]

66. Jameson, Horatio: no title. *The Maryland Medical Recorder.* Baltimore, 1831, Vol. II. p. 252. [Secondary haemorrhage after perineal lithotomy. Both patient and a sister had bled after tooth extraction. The old and serious mother alleged that she had a bleeding family to such an extent that a large flat stone at her back door had been worn away by the bleeders who sat upon it during attacks of epistaxis! The nature of the stone which was thus so easily worn away is not stated.]

67. Richard: Beobachtung einer beynahe tödtlichen Blutung in Folge von Blutegelstichen bey Behandlung einer Exostosis in Jahre 1830. *Verhandlungen der vereinigten ärztlichen Gesellschafter der Schweiz.* Zürich, Jahrg. 1831, Erste Hälfte, S. 64. [Male aged 32, very scrofulous and rheumatic in childhood. Paraplegia and total deafness in one ear before 4, frequent epistaxis and purpura, twice almost died from bleeding after tooth extraction; on one occasion great haemorrhage from lower lip, but after a venesection there was no haemorrhage. At 15 kicked on knee by horse, great swelling followed, but disappeared in eight months, reappearing on two subsequent occasions. Condition continued to the age of 30, when nine leeches were placed on the knee with result as in title. His father and mother and his two daughters all had epistaxis.]

68. Schliemann, Ernst: De dispositione ad haemorrhagias perniciosas haereditaria. *Dissertatio inaug. med.* Wirceburgi, 1831. [Pedigree No. 509. In addition three cases, in males, of little value.]

69.* Grandidier, L. De dispositione ad haemorrhagias lethales hereditaria. Dissertation. Casselis, 1832. [Not seen. According to Grandidier himself the case herein described is the same as that in his paper in Holscher's *Annalen* 1839 (Bibl. No. 110). The "Auskunftsbureau der deutschen Bibliotheken" informs us that a copy of this rare publication is to be found in Murhardsche Bibliothek der Stadt Cassel. Pedigree No. 485.]

70. [Gröschner of Schlochau]: Bluterfamilie. *Magazin für die gesammte Heilkunde, hrsg. von Johann Nep. Rust.* Berlin, 1832, Bd. xxxii. S. 397. [Pedigree No. 381.]

71. Hughes, James N.: A case of hereditary haemorrhagic tendency. *The Transylvania Journal of medicine and the associate sciences.* Lexington, Ky., 1832, Vol. v. p. 133. [Pedigree No. 532.]

72. Mende, L. J. C.: Ausführliches Handbuch der gerichtlichen Medizin für Gesetzgeber, Rechtsgelehrte, Aerzte und Wundärzte. Leipzig, 1832, Sechster Theil, S. 327. [Refers to a boy of 4 in Göttingen. Although a bleeder his parents and siblings were not affected.]

73. Rueber, Joannes Justus: De dispositione ad haemorrhagias lethales hereditaria. *Diss. inaug. medica.* Berolini [1832], 31 pp. [Valueless account of male aged 12 who bled for days after the slightest injury and after tooth extraction. Father had haemorrhoids and died after tooth extraction.]

74. Robbins, C.: Case of profuse and obstinate haemorrhage after the extraction of a tooth. *The Boston Medical and Surg. Journal*, Boston, 1832—3, Vol. vii. p. 289. [Adult male, family or previous history bleeding not referred to.]

75. Droste, Aug.: Fall von Stomatorrhagie. *Wochenschrift f. d. ges. Heilkunde von Casper.* Berlin, 1833, Bd. i. S. 689. [Male aged 18. Bleeding from a small swelling on the gum, no history in the family.]

76. Elsaesser: Geschichte einer Familie von Blutern in Württemberg. *Journal der practischen Arzneykunde und Wundarzneykunst, hrsg. v. C. W. Hufeland und E. Osann.* Berlin, 1833, Bd. lxxvii. Stück 5, S. 133. [Pedigree No. 393.]

77. Fränzel: Ueble Folgen des Zahnausnehmens bei einem Bluter. *Medicinische Zeitung, hrsg. von dem Verein für Heilkunde in Preussen.* Berlin, 1833, Bd. ii. S. 164. [Haemorrhage after tooth extraction, no heredity.]

78. Heyfelder: Ueber die angeborne Disposition zu Blutungen bei sogennanten Bluter. *Medicinische Zeitung, hrsg. von dem Verein für Heilkunde in Preussen.* Berlin, 1833, Bd. ii. S. 215. [Two families; rickets, scrofula, rheumatism, epistaxis.]

79. Lohmeyer: Ein sogennanter Bluter. *General-Berichte des königl. rheinischen Medicinal-Collegii*, 1832. Koblenz, 1833, S. 143. [Young male affected with epistaxis which also affected his brother, maternal uncle and a cousin, insufficient account.]

80.* Roux: Quoted by Grandidier as being in *Journ. de méd. et de chir. prat.*, 1833, Vol. viii., not found in 1833 which is Vol. iv., nor in 1836, which is Vol. viii.

81. Ussing, P. St: Bemaerkninger over den arvelige Disposition til dödelige Vorblödninger. *Journal for Medicin og Chirurgie.* Kjöbenhavn, 1833, Bd. ii. S. 289. [Family of two daughters and three sons. One of the daughters had epistaxis, ecchymoses, and she bled profusely after a contusion. One of the sons had epistaxis and ecchymoses, another son in addition bleeding from leech bites. The mother had epistaxis, haemorrhoids and menorrhagia, and died at 33 of phthisis.]

82. Woodward, E.: A case of hereditary disposition to haemorrhage from slight wounds. *The Boston Medical and Surgical Journal.* Boston, 1833, Vol. viii. p. 219. [Male aged 4, bled for some days from cut on his finger. Ten weeks later got a gash over his left eye. At first, bleeding was controlled, but suppuration set in, and haemorrhage started again. Incision followed by bleeding to the point of syncope. Ultimate recovery. A sib (sex?) had a similar constitution and died at the age of 2 or 3.]

83. Kuhl, C. A.: [Pr.] De idiosincrasia haemorrhagica. Particula 1—5. 4°, Lipsiae, 1833—4 (particula ix.—xiii. of Quaestionum Chirurgicarum). [Particula 1—4 general account. Particula 5 contains the same four cases as in Bibl. No. 93.]

84. Lereboullet, A. De l'hérédité dans les maladies. *Thèse (présentée et soutenue devant le jury du concours).* Strasbourg, 1834, p. 10. [General account of haemophilia, no original cases.]

85. Salomon: Beitrag zur Geschichte der Bluter. *Wochenschrift für die ges. Heilkunde von Casper.* Berlin, 1834, No. 7, S. 97. [Pedigree No. 551.]

86. Burdach: Bluterfamilie. *Sanitätsbericht der Provinz Brandenburg*, 1835. Berlin, 1837, S. 191. [Four cases. (1) Child aged 14 days. At end of left middle finger and second joint of right middle finger appeared small red spots which opened and bled. Death on 16th day. Parents had already lost two children from haemorrhage from navel: one at the age of 4 years, the other at 3 weeks. (2) Child aged 4 months, bled much from left and then from right eye; recovery: no other data. (3) Furuncle on fontal bone of a child. Spontaneous rupture, haemorrhage, death. No family history. (4) Haemorrhage from dental cavity in a boy aged 10, slow recovery, no other data.]

87. Cramer: Eine Bluterfamilie. *Wochenschrift für die gesammte Heilkunde von Casper.* Berlin, 1835, No. 33, S. 529. [This case has been reviewed under the erroneous name of Crasner in the *Gazette méd. de Paris*, 1836, iv. ser. 2, p. 599, where it is stated to have been extracted from the *Wissenschaftliche Annalen der gesammten Heilkunde.* This, however, is also erroneous. Pedigree No. 400.]

88. Escherich: Geschichte eines Bluters. Leichte Hiehwunde in der rechten Wange, unstillbare parenchymatöse Blutung; veränderte Qualität des Blutes, Adstringentia innerlich und äusserlich: das Glüheisen. Tod nach 44 Stunden. Sektion, Offenseyn des Foramen ovale. *Medicinisches Correspondenz-Blatt des Württembergischen aerztlichen Vereins.* Stuttgart, 1835, Bd. v. No. 19, S. 147. [P. D., student of medicine, aged 22. At 2, symptoms of haemophilia so well marked that Schönlein was consulted. Haemorrhage from the tongue, great bleeding after wounds,

and leech bites. Two months before date small abrasion of forehead bled three weeks. In spite of his diathesis he engaged in a duel with the result described in Escherich's title. No history of haemophilia except that he thought one of his numerous sibs was similarly affected.]

89. Lafargue: Diathèse hémorrhagique héréditaire. *Journ. hebdomadaire des progrès des sciences et institutions médicales.* Paris, 1835, Tome III. p. 238. [Pedigree No. 546.]

90. Osborne, Jonathan: Account of a haemorrhagic diathesis existing in a family. *The Dublin Journal of Medical and Chemical Science.* 1835, Vol. VII. p. 32. [Pedigree No. 579.]

91. Wardrop, James: On blood letting. London, 1835, p. 16. [Pedigree No. 395.]

92. Burdach (of Finsterwalde): Bluter. *Med. Zeitung.* Berlin, 1836, Bd. v. S. 169. [Two cases described, one referring to three babies in a family, death from haemorrhagic sepsis and cachexia, the other purpura and haemorrhage from the eyes in a child 4 months old.]

93. Kuhl, C. A.: Beobachtungen über Bluter. *Beitrag zur praktischen Heilkunde.* Leipzig, 1836, Bd. II. S. 345. [Four cases; (1) male, aged 16, bled severely from cut thumb and from scalp wound caused by a falling tile; (2) male, aged 22, haemorrhage from skin of left knee; (3) girl, aged 19, strong and healthy, one day felt an itching in the corner of her eye and found a drop of blood exuding from the caruncle. This continued for several days, and could not be looked upon as vicarious menstruation as she remained entirely regular. After spontaneous arrest this condition recurred twice: no mention of inheritance. This is the case referred to by Grandidier (Bibl. No. 214, S. 88) as one of the "merkwürdige Fälle von Hämophilie bei Frauen"; (4) male aged 5 with severe epistaxis.]

94. Laborie: Diathèse hémorrhagique héréditaire. *Gazette des hôpitaux.* Paris, 1836, Tome x. p. 178. [Imperfect account of Lafargue's case (Bibl. No. 89 and Pedigree No. 546).]

95. Sanson, L. J.: Des hémorrhagies traumatiques. (Concours pour une chaire de clinique chirurgicale vacante à la faculté de médecine de Paris.) Paris, 1836, 352 pp. [On page 21 reference to a female aged 17 with bleeding from the mouth in the course of the growth of a vast tumour of the shoulder.]

96. Schaefer: Erfolgreiche Anwendung des Secale cornutum bei einem Bluter. *Medicinische Zeitung hrsg. von dem Verein für Heilkunde in Preussen.* Berlin, 1836, Bd. v. S. 130. [Male aged 34, scrofulous, alcoholic, and cachectic. For two years he had lost large quantities of blood from the mouth. Cured by extract of ergot.]

97. Taynton, R. T.: Case of fatal haemorrhage from lancing the gums. *The London Med. Gazette.* London, 1836, Vol. XVII. p. 659. [Child six months as in title, no family history.]

98. Beckhauss: Blutungen bei einem neugebornen Kinde. *Medicinische Zeitung hrsg. von dem Verein für Heilkunde in Preussen.* Berlin, 1837, Bd. VI. S. 208. [Umbilical haemorrhage.]

99. Bicking: Idiosyncrasie gegen Feuchtigkeit bei einer Bluterfamilie. *C. W. Hufeland's Journ. der practischen Heilkunde fortgesetzt von Dr E. Osann.* Berlin, 1837, Bd. LXXXIV. Stück IV. S. 110. [Instances of bleeding in the family of "Pr. W. in Z." These include piles, haemoptysis, epistaxis and umbilical haemorrhage, and in no way suggest haemophilia.]

100. Grandidier: Nachricht von einer Bluterfamilie in Kurhessen. *Allgemeine med. Zeitung.* Altenburg, 1837, No. 69, S. 1089. [Pedigree No. 483.]

101. Lebert: Recherches sur les causes, les symptomes et le traitement des hémorrhagies constitutionnelles. *Archives générales de médecine.* Paris, 1837, série 3, Tome III. p. 36. [Contains the history of a porter aged 24. Had suffered from epistaxis and haemorrhage from slight wounds; nearly bled to death from leech bites. Dislocation of thumb with great swelling diagnosed as an aneurism; incision, haemorrhage, ligation of ulnar and radial arteries, death.]

102. Lucas (of Erkelenz): no title. *General-Berichte des königl. rheinischen Medicinal-Collegii*, 1835. Koblenz, 1837, S. 116. [Female aged 57 with purpura, epistaxis and bleeding from the gums, recovery. Not haemophilia.]

103. Nevermann: Bemerkungen über die erbliche Disposition zu tödtlichen Verblutungen von P. St Ussing vorgetragen in der Gesellschaft "Philatrien" zu Kopenhagen. *Medicinische Annalen.* Heidelberg, 1837, Bd. III. S. 424. [Abstract of Ussing's case, Bibl. No. 81.]

104. Rösch, Carl: Untersuchungen aus der Gebiete der Heilwissenschaft. Stuttgart, 1837, Erster Theil, S. 249. [Female aged 12, bruises started to appear after slight trauma when she began to walk. Epistaxis, haematoma, the result of blow. No family history of bleeding.]

105. Thormann, F.: Haematocele bei einem Jünglinge aus einer Bluterfamilie. *Schweizerische Zeitschrift für Natur- und Heilkunde.* Heilbronn, 1837, Bd. II. S. 19. [Same as Bibl. No. 115 and Pedigree No. 373.]

106. Du Bois: Observation remarquable d'hémorrhaphilie (disposition héréditaire aux hémorrhagies). *Gaz. Méd. de Paris*, 1838, Tom. VI. p. 43. [Pedigree No. 405.]

107. Liston, R.: Haemorrhagic idiosyncrasy. *The Lancet*, 1838—1839, Vol. II. p. 136. [Pedigree No. 535; cf. Kendrick, Bibl. No. 63.]

108. Convers: Observation d'hémorrhagie consécutive. *Gaz. Méd. de Paris*, 1839, T. VII. p. 315. [Haemorrhage secondary to a wound of arm.]

109. Gabriel, P. G.: De haemorrhagia hereditaria. *Inaug. Diss.* Berolini [1839]. [General account, no cases.]

110. Grandidier, L.: Ueber die erbliche Neigung zu tödtlichen Blutungen oder die sogennanten Bluterkrankheit. *Hannover'sche Annalen für die ges. Heilkunde hrsg. v. G. P. Holscher*, 1839, Bd. IV. S. 1. [Pedigree No. 485.]

111. Mende: Geschichte eines Bluters. *Mittheilungen aus dem Archiv der Gesellschaft practischer Aerzte zu Riga.* Leipzig, Riga and Mitau, 1839, erste Sammlung, S. 69. [Case of a woman who lost her five children from haemorrhage as follows: (1) male, was well up to 6 weeks when his mother found him exsanguine in his cradle; death. (2) female, who some days after birth showed a blue streak on the forehead, a week later it began to bleed and she died. (3) female, in the 4th week a red spot appeared on the arm and began to bleed, death occurring in 36 hours. (4) male, umbilical haemorrhage, death. (5) female, scratched her nose and bled to death. The father of these children had syphilis when young.]

112. Schneider, P. J. (in Offenburg): no title. *Schmidt's Jahrbücher der in- und ausländischen gesammten Medicin.* Leipzig, 1839, Bd. XXIV. S. 354. [Review of Siebenhaar's Handb. der gerichtl. Arzneikunde, with a general account of haemophilia in its forensic aspects by Schneider. S. records a case of his own in which a family of five sons and six daughters suffered from profuse epistaxis. One of the sons, aged 17, was also troubled with gout and died of epistaxis which could not be controlled. The autopsy showed disease of the heart involving the musculature and valves.]

113. Schönlein, J. L.: Allgemeine und specielle Pathologie und Therapie. St Gallen, 1839, Theil II. S. 54. [General account.]

114. Evert: no title. *Zeitschrift für die gesammte Medicin mit bes. Rücksicht auf Hospitalpraxis und ausländische Literatur hrsg. von Fricke und Oppenheim.* Hamburg, 1840, Bd. XIII. S. 404. Abstract of a paper in Ars-Berättelse om Svenska Läkare-Sällskapets Arbeten af Souden. Stockholm, 1838, S. 74. [Male infant, death on 20th day after numerous haemorrhagic symptoms. Mother cured of venereal disease two years previously.]

115. Thormann: Seltener Fall einer Haematocele. *Journal der Chirurgie und Augen-Heilkunde hrsg. von C. F. v. Gräfe und Ph. v. Walther.* Berlin, 1840, Bd. XXX. S. 297. [Describes the case of Christian Buchli of Tenna. Pedigree No. 373.]

116. Burnes, David: Haemorrhagic diathesis. *The Lancet.* London, 1840—1841, Vol. I. p. 404. [Pedigree No. 592.]

117. Hooper. [Case quoted in paper of D. Burnes, Bibl. No. 116.]

118. Lane, Samuel: Haemorrhagic diathesis, successful transfusion of blood. *The Lancet.* London, 1840—1841, Vol. I. p. 185. [Two cases: (1) of no value, no family history; (2) case described in James Wilson's paper. Bibl. No. 32 and Pedigree No. 463.]

119. Ray, C.: On the haemorrhagic diathesis. *The Lancet.* London, 1840—1841, Vol. I. p. 823. [Author describes three cases of severe haemorrhage after the extraction of loose teeth, similar to those frequently published under the name "haemophilia." He points out that haemorrhages of this kind are due entirely to the existing local conditions and that before a diagnosis of haemophilia is arrived at, a thorough history must be obtained of other manifestations of the diathesis.]

120. Smethurst, T.: Haemorrhagic diathesis. *The Lancet.* London, 1840—1841, Vol. I. p. 648. [Two cases of bleeding after tooth extraction in women.]

121. Claudi: Ein Bluter. *Oesterreichische med. Wochenschr.* Wien, Jahrg. 1841, S. 435. [Male, aged 21, haemorrhage from wound of head produced by fall of a log of wood.]

122. Johannsen, Rudolf: De haemorrhaphilia. *Diss. inaug. med.* Kiliae, 1841, 31 pp. [Three cases: (1) male, died from small wound made while cutting his toe nail. A brother died at the age of 10 after treading on a piece of glass, while another brother died from a contusion of the forehead produced by a stone; (2) male, was long subject to huge haemorrhages from slight wounds. During pneumonia he was venesected and his wife found him dead next morning, the bed full of

blood. The brachial artery was not wounded as was thought at the time. His mother and three sisters were healthy. His father died from haemorrhage following amputation for gangrene, and a paternal uncle also bled to death early from a cut; (3) girl, aged 6, lost much blood from prick with sewing needle; one attack of epistaxis. Her mother had *post partum* haemorrhage.]

123. Mutzenbecher, F. M.: De haemorrhagicis. *Diss. inaug.* Heidelbergae, 1841. [Pedigree No. 389.]

124. Quadrat: Fälle von Hämorrhaphilie. *Oesterreichische med. Wochenschrift.* Wien, 1841, No. 33, S. 769. [Three cases: (1) married woman aged 34, childless, suffered from menorrhagia for four years due to plethora of the venous system in the abdomen. The veins of the cervix uteri resembled those seen in piles. She had haemoptysis three times and lost large quantities of blood from trivial injuries. In her 19th year on the day before menstruation was due, a swelling formed on her right forefinger and gave rise to a flow of blood. This replaced the menstrual flow lasting three or four days and recurring during six years when the finger became spongy. Her mother and two married sisters were subject to menorrhagia; (2) female, epistaxis in pregnancy; (3) child of case 2, umbilical haemorrhage, death. Case 1 is quoted by Grandidier (Bibl. No. 214, p. 89) as a case of haemophilia.]

125. Tardieu, Ambroise: Observation de diathèse hémorrhagique avec douleurs articulaires. *Arch. gén. de méd.* Paris, 1841, série 3, Tome x. p. 185. [Male, aged 33, frequently under observation in hospital for painful swelling of joints, bleeding from gums, epistaxis, haemoptysis, haematuria and ecchymosis. No history of haemorrhage in family.]

126. Wilmot, S.: A case of haemorrhagic diathesis. *The Dublin Journal of Med. Science.* Dublin, 1841, Vol. xix. p. 234. [Pedigree No. 552.]

127. Cochrane, J.: On the haemorrhagic diathesis. *The Lancet.* London, 1841—1842, Vol. ii. p. 147. [Polemic, of no value.]

128. Roberts, W. A.: Haemorrhagic diathesis; fatal haemorrhage from the extraction of a tooth. *The Lancet.* London, 1841—1842, Vol. i. p. 752. [Same case as D. Hay; Bibl. No. 130, male, aged 31, died of haemorrhage, lasting three weeks, following extraction of a decaying molar.]

129. Allan, J.: Cases of haemorrhagic diathesis. *The London and Edinburgh monthly Journal of Medical Science.* London, Vol. for 1842, p. 501. [Pedigree No. 425.]

130. Hay, David: Case of haemorrhage following the extraction of a tooth and terminating fatally. *The London and Edinburgh monthly Journal of Medical Science.* London, Vol. for 1842, p. 264. [Same as W. A. Roberts, Bibl. No. 128.]

131. Hunt, R. T.: Case of general haemorrhagic tendency. *Prov. Med. and Surg. Journ.* London, 1842, Vol. ii. p. 143. [Not haemophilia, male aged $7\frac{1}{2}$.]

132. Miller, James: On the treatment of the haemorrhagic diathesis. *The London and Edinb. monthly Journal of Medical Science.* London and Edinburgh, Vol. for 1842, p. 567. [No cases.]

133. Steiner, F.: De haemophilia. *Diss. inaug.* Berolini, 1842. [Male, aged 24, first affected at 23 when he had epistaxis, bleeding from the bowel from which he died. His father also had epistaxis when 22 years of age and died of it.]

134. Storrs, R.: Case of venous haemorrhage from an abscess. *Prov. Med. and Surg. Journ.* London, 1842, Vol. i. p. 527. [Colossal erysipelatous lesion of chest and head. Incision. No previous personal or family history.]

135. Storrs, R.: A bleeding family. *Provincial Medical Journal.* London, 1842, Vol. ii. p. 473. [Family of six children, five of whom, two males and three females, had umbilical haemorrhage, one male dying of it. No family history.]

136. West, J. W.: Severe haemorrhage from the tongue. *Provincial Medical Journal.* London, 1842, Vol. ii. p. 438. [Male child. Another child in same family bled badly after slight wound. No further history.]

137. Bérard: ref. in Canstatt's *Die specielle Pathologie und Therapie.* Erlangen, 1843, 2te Aufl. Bd. i. S. 138. [Mere mention of name; worthless.]

138. Von Bippen, Wilhelm: no title. *Amtlicher Bericht über die zwanzigste Versammlung der Gesellschaft deutscher Naturforscher und Aerzte zu Mainz im Sep.* 1842. Mainz, 1843, Sitzung am 24 Sep. S. 290. [The communication of von Bippen is of interest on account of the erroneous estimate of its importance as given by Grandidier and copied from him by many subsequent writers, and from the fact that it is supposed to *describe* the grandchildren of the illustrious Johann Christian Reil. It is stated by Grandidier (1st ed. p. 14) that von Bippen's communication was made in Mainz, 1843, and in Bremen, 1844. "auch beschrieben von

Heyland in der neuen med. chirurg. Zeitung 1844, No. 5." The published account of v. Bippen's communication occupies $2\frac{1}{4}$ lines in which it is stated that four of Reil's grandchildren died chiefly of epistaxis, a girl aged 14 being still alive. Von Bippen's name does not appear at all in the Bremen *Bericht* and Heyland does not refer to him there. In the *Med. chirurg. Zeitung*, however, Heyland, who says that he did not know von Bippen, takes the opportunity, in a communication occupying 12 lines, of asserting that von Bippen's report "enthält viele Unwahrheiten" [!] In the discussion on Bippen's paper a von Ritgen recommended creosote internally and externally. A translation of Bippen's $2\frac{1}{4}$ lines is given in the *Gaz. méd. de Strasbourg*, 1843, p. 408, and this is referred to as a communication by Ritgen by Wickham Legg and many others.]

139. Canstatt, Carl: *Die specielle Pathologie und Therapie vom klinischen Standpunkt ausbearbeitet.* Erlangen, 1843, 2te Aufl. Bd. i. S. 138. [General account.]

140. Kercksig: Bluterkrankheit, *Provinzial-Sanitätsbericht des königl. Medizinal-Kollegiums von Westfalen*, 1843, S. 80. [Pedigree No. 506.]

141. Ritgen: *Gazette Médicale de Strasbourg*, 1843, p. 408. [See remarks on von Bippen, Bibl. No. 138.]

142. Dequevauviller, Jean François: De la disposition aux hémorrhagies et des signes au moyen desquels on peut la prévoir. *Thèse de Paris*, 1844, 43 pp. [Pedigree No. 530.]

143. Dequevauviller: De la disposition aux hémorrhagies et des signes auxquels on peut la reconnaitre. *Journal de Chirurgie*, Paris, 1844, Tome ii. p. 164. [Same cases as in D.'s Thesis, Bibl. No. 542.]

144. Erdmann, E. E.: De haemophilia. *Inaug. Diss.* 8°, Hallis, 1844. [Pedigree No. 396.]

145. [Gobée] cited by many authors as in "*Oppenheims Zeitschrift* 1844" and as an instance of a Belgian bleeder. In the *Zeitschrift für die gesammte Medicin hrsg. v. Oppenheim*, Hamburg, 1844, Bd. xxxvii. p. 210, there is *one line* to the effect that *Gorée* treated a bleeder with Ungt. Hydrargyri, and reference is made to the original source of publication, viz. *Nederl. Lancet*, 1842. On consulting this we find an article by Kerst, entitled Mededeelingen uit het gebied der Genees- Heel- en Oogheelkunde. *Nederlandsch Lancet, practisch Tijdschrift aan de Geneeskunde*, Utrecht, 1842—1843, Bd. v. S. 300. At p. 311, *Gorée* now appears as Chir. Majoor Dr *Gobée*, and it states that he communicated to Kerst the fact that he treated with great success a so-called bleeder, with Ung. Hydrargyri. Worthless.]

146. Guépratte, Alph.: Un cas d'hémorrhaphilie. *Journal de connaissances médico-chirurgicales.* Paris, 1844, Tome x. p. 239. [Recovery from haemorrhagic condition allied to scurvy rickets in a boy after receiving suitable diet.]

147. Heyland: no title. *Tagesgeschichte Bremen Oct. I. in Neue medicinisch-chirurgische Zeitung* hrsg. v. G. Ludwig Ditterich. München, 1844, Bd. iv. (neuer Folge zweiter Jahrgang), No. 45, Dec. 3, S. 189. [Worthless. See remarks on Bibl. No. 138.]

148. Hunt, R. T.: Case of haemorrhagic tendency complicated with purpura and with strumous diathesis. *Prov. Med. and Surg. Journ.* London, 1844, No. 2, p. 21. [A dissipated musician, aged 24, bleeds for the first time from carious teeth.]

149. [Luke]: Haemorrhagic diathesis. *The Lancet.* London, 1844, p. 423. [Secondary haemorrhage from scalp wound. Previous haematuria and epistaxis.]

150. Melicher, L.: Haemorrhagia gravis ex cellula dentis molaris ultimi. *Oesterreichische med. Wochenschrift.* Wien, Jahrg. 1844, S. 869. [Male, aged 19, tooth extraction, fracture of alveolus of jaw, haemorrhage: had haemorrhage from extraction before, also epistaxis.]

151. Miling: Ein Bluter-Familie. *Medicinische Zeitung hrsg. von dem Verein für Heilkunde in Preussen.* Berlin, 1844, Bd. xiii. S. 31. [Trifling account of a family; no value.]

152. Schultz, J.: De idiosyncrasia haemorrhagica historia morbi adnexa. *Diss. inaug.* Berolini [1844]. [Male, aged 28, epistaxis and wandering pains. Severe haemorrhage from cut on thumb, nearly bled to death after tooth extraction. No history of haemophilia in the family.]

153. Wolff, Jean-Daniel: De la diathèse hémorrhagique héréditaire. *Thèse de Strasbourg*, 1844. Série 2, No. 123, 32 pp. [Male, aged 42. Swelling of hip, discharge of pus, death. Two brothers died of epistaxis and tooth extraction. A cousin on paternal side bled from wound of head for 15 days.]

154. Besserer: Blutungssucht. *Medicinisches Correspondenz-Blatt rheinischer und westfälischer Aerzte. hrsg. von F. Nasse und J. F. H. Albers.* Bonn, 1845, Bd. iv. S. 353. [Pedigree No. 558.]

155. Druitt, W.: Case of compound fracture of the leg in a patient of the haemorrhagic diathesis. *Prov. Med. and Surg. Journ.* London, 1845, p. 260. [Male, aged 27, no family history, not haemophilia.]

156. Haime: Note sur un cas de diathèse hémorrhagique extraordinaire. *Recueil de travaux de la société médicale du département d'Indre-et-Loire.* Tours, 1845, p. 100. [Female, aged 42: in menopause, epistaxis, haematemesis and anaemia relieved by small venesections. Title of paper misleading.]

157. Heyland: no title. *Amtlicher Bericht über die zwei und zwanzigste Versammlung deutscher Naturforscher und Aerzte in Bremen im September* 1844. Bremen, 1845, S. 163. [See von Bippen, Bibl. No. 138, and Ritgen, Bibl. No. 141.]

158. Nasse, Fr.: Die Blutungssucht. *Medicinisches Correspondenz-Blatt rheinischer und westfälischer Aerzte hrsg. von F. Nasse und J. H. Albers.* Bonn, 1845, Bd. iv. S. 207. [Case quoted in which a daughter of a bleeder family bled for 24 hours from a cut on finger. No details of family.]

159. Pluemicke, G. C.: De haemorrhagia hereditaria. *Inaug. Diss.* Berolini [1845]. [General account of haemophilia, no original cases.]

160. Baucek, F.: Fall von einem Bluter. *Oesterreichische med. Wchnschr.* Wien, 1846, column 554. [Short account of three boys of healthy parents, Nos. 1 and 2 had haemorrhage from gums, tongue and nose, No. 3—a baby—had epistaxis.]

161. Bjørkman: no title. *Hygiea.* Stockholm, 1846, Bd. viii. S. 513, and 1847, Bd. ix. S. 723. [Pedigree No. 507.]

162. Clay: Haemorrhagic diathesis, ten days haemorrhage after the extraction of a tooth. *The Medical Times.* London, 1846, Vol. xiii. p. 293. [Pedigree No. 590.]

163. Lange: Ein Bluter. *Medicinische Zeitung hrsg. von dem Verein für Heilkunde in Preussen.* Berlin, 1846, Bd. xv. S. 24. [Male, aged 3, probably a bleeder although there was no mention of the disease in the family. At 1 year of age he had discoloured swellings on arms, feet, belly, breast, neck and head. At 2 he cut his forehead and bled for 10 days. On two occasions he bled from the nose for 14 days. On another occasion he bled from the palate. At 3, he fell and sustained a swelling of his knee for which six leeches were applied. With considerable difficulty the bleeding from five of the bites was arrested, Lange being called to deal with the haemorrhage which was still going on from the sixth leech bite.]

164. [Vieli]: Observations sur les bluters ou hommes saignants. *Journal de médecine et de chirurgie pratiques à l'usage des médecins praticiens par Lucas Championnière.* Paris, 1846, Tome xvii. p. 340. [Pedigree Nos. 373, 375, 376.]

165. Adams, G. H.: On the effects of acetate of lead and opium in haemorrhagic diathesis. *The London Med. Gazette.* London, 1847, Vol. iv. p. 58. [A lad suffering from haematemesis treated with lead acetate.]

166. Fuchs, C. H.: Lehrbuch der speciellen Nosologie und Therapie. Göttingen, 1847, Bd. ii. S. 620. [In the course of a general accouut there is short mention (p. 624) of a male aged 40, said to have been affected from youth onwards.]

167. Küster, F. and R.: Die Bluterkrankheit. *Wochenschrift für die ges. Heilkunde (Casper).* Berlin, 1847, No. 18, S. 282. [Male "Curgast" 28—30, received injury to finger from the rope of a swing. No primary haemorrhage, but later on, and largely as the result of the treatment, some haemorrhage. The patient stated that the sons in the family had been bleeders from time immemorial.]

168. Lange: Die geographische Verbreitung der Bluterkrankheit. *Medicinische Zeitung hrsg. v. d. Vereine für Heilkunde in Preussen.* Berlin, 1847, Jahrg. vi. S. 124. [Short account.]

169. Schmidt, B. A. B.: De haemophiliae casu quodam. *Diss. Inaug.* Regiomonti Prussorum, 1847. [Male, aged 25, epistaxis and intestinal haemorrhage, death. Autopsy. He had bled from smallest cut. No history of haemorrhage in the family.]

170. Schneider (in Fulda): Die Bluter erbliche Blutung oder sogennante Bluterkrankheit betrachtet in medicinisch-gerichtlicher und polizeilicher Hinsicht. *Adolph Henke's Zeitschrift für die Staatsarzneikunde.* Erlangen, 1847, Bd. liii. S. 1. [Review, no cases.]

171. Dubois, Emile-Amable: De l'hémorrhagie ombilicale après la chute du cordon. 35 pp. *Thèse de Paris,* 1848, No. 241. [Male child died on 13th day, mother healthy.]

172. Kleeberg: De idiosyncrasia haemorrhagica. *Inaug. Diss.* Berolini, 1848. [General account, no original cases.]

173. Liedbeck, P. J.: no title. *Hygiea, medicinsk och pharmaceutisk månadsskrift utgiven af svenska läkaresällskapet.* Stockholm, 1848, Bd. x. S. 363. [See Bjørkman, Bibl. No. 161 and Pedigree No. 507.]

174. Thore: Observation d'hémorrhagie par le tubercule ombilical. *Gazette médicale de Paris*, 1848, p. 191. [Male infant, umbilical haemorrhage and pyaemia.]

175. André: no title. *Hygiea.* Stockholm, 1849, Bd. xi. S. 677. [Family of six children of whom the last four (3 ♀ and 1 ♂) died after a period of bilious vomiting associated with livid spots and haemorrhages from skin. Prof. K. Petrén of Upsala, who abstracted André's paper for this paper, suggests Barlow's disease as the proper diagnosis.]

176. Barbieri: Singolare emorragia da un dito affeto di infiammazione. *Gazzetta medica lombardia.* Milano, 1849, serie seconda, Tome ii. p. 68. [Male, aged 28, with spontaneous haemorrhage from one of his fingers. No family history of haemophilia.]

177. Bühring: Ein Fall von schwer zu stillenden nach Extirpation einer Tonsille, erfolgreiche Anwendung des Ergotin. *Deutsche Klinik.* Berlin, 1849, Bd. i. S. 55. [Case of a male aged 25, no family history.]

178. Modoni, Giuseppe: Emorragia in seguito ad estrazione di dente ricorrente a periodi e vinta col sulfato di chinina. *Raccoglitore medico.* Fano, 1849, Vol. xxiv. p. 276. [Male of middle age. Great and repeated haemorrhage after the extraction of the three lower molars. Ultimate arrest with sulphate of quinine. No family history.]

179. Papillaud, Lucien: Observation d'hémorrhagie mortelle ayant eu lieu par diverses voies, à la suite d'extraction de dents. *Gaz. méd. de Paris*, 1849, T. iv. p. 104. [As in title, male aged 25—28, no family history.]

180. Ray, Edward: On haemorrhage from the umbilicus after the separation of the funis. *The London Med. Gazette or Journal of Practical Medicine.* London, 1849, new series, Vol. viii. p. 423. [As in title, three males in one family, no history of bleeding in the family.]

181. Wachsmuth, Carl Otto Theodor: Die Bluterkrankheit, Versuch einer Monographie derselben nach eigenen Beobachtungen und Erfahrungen. Magdeburg, 1849, 60 pp. Separat-Abdruck aus dem *Zeitschrift des deutschen Chirurgen Vereins.* Magdeburg, 1849, Bd. iii. S. 459. [Pedigree No. 409.]

182. Donkersloot, N. B.: Over de erfelijke bloedingen, bloederziekte (haemorrhophilia) uit het oogpunt der geregtelijke geneeskunde en geneeskundige policie. *Nederl. Lancet.* Gravenhage, 1849—1850, 2 S. Bd. v. S. 416. [Pedigree No. 503.]

183. Amyot, Thomas: Haemorrhagic diathesis. *The Med. Times—a Journal of Medical and Chemical Science.* London, 1850, Vol. xxi. p. 449. [Three cases described: (1) an infant dying on the 5th day of haemorrhage from scrotum, umbilicus and mucous membranes. (2) female infant bled after lancing of gums. (3) infant with lacerated wound (7 inches) of scalp, suture with twine by midwife.]

184. Bowditch, Henry I.: On haemorrhage from the umbilicus in new born children with cases. *The American Journal of the Medical Sciences.* Philadelphia and London, 1850, new series, Vol. xix. p. 63. [Two children in a family of four.]

185. Dunlap, I. B.: Case of hemorrhagic diathesis following suppression of the catamenia and attended by vicarious discharge from the gums—terminating fatally from haemorrhage following scarification. *New York Journ. of Medicine.* 1850, Vol. iv. p. 314. [Case of gall stones (autopsy) in a young lady.]

186. Manley, John: On haemorrhage from the umbilicus after the separation of the funis. *The London Medical Gazette.* London, 1850, new series, Vol. x. p. 755. [As in title.]

187. Van der Scheer, A.: Drie gevallen van bloederziekte, waargenomen in Drenthe en Groningen. *Nieuw practisch Tijdschrift voor de Geneeskunde in al haren Omvang.* Gorinchen, 1850, Bd. ii. S. 414. [Pedigree Nos. 543, 544.]

188. Stoehr, G. J.: Ueber Haemophilie oder die erbliche Anlage zu tödtlichen Blutungen. *Inaug. Diss.* Erlangen, 1850. [Pedigree No. 454.]

189. Uhde, C. W. F.: Fall von einem Bluter (ἀμυχαιμορραγής). *Deutsche Klinik, Zeitung für Beobachtungen aus deutschen Kliniken und Krankenhäusern hrsg. v. Alex. Göschen.* Berlin, 1850, Dec. 7, No. 49, Bd. ii. S. 537. [Male, aged 50, who received a contusion on R. thigh from a barrel. A fortnight later, under the impression that it was pus, Uhde incised it and found it was blood: there was considerable haemorrhage. Ultimate recovery. Patient had previously bled for three weeks from a cut on his finger, and for 18 weeks after the extraction of a tooth. A maternal uncle is said to have bled easily from the slightest injury.]

190. Fearn, R. L.: Hemorrhagic diathesis. (Proceedings of the Mobile med. Soc.) *New Orleans Medical and Surgical Journal.* 1850—1851, Vol. vii. p. 178. [Male, aged 9, had bled badly on two separate occasions from trivial injuries, sustained a penetrating wound one inch deep between thumb and forefinger, haemorrhage, suppuration, recovery.]

191. Bordmann, J.-M.: De hémophilie ou de la diathèse hémorrhagique congéniale. *Thèse de Strasbourg.* 1851, 52 pp. [Thesis founded on Wachsmuth's monograph, q.v., no cases.]

192. Fournier, Ulysse: Quelques réflexions à propos d'une observation de purpura hémorrhagica. *Gaz. des hôpitaux.* Paris, 1851, p. 493. [Pedigree No. 534.]

193. Lange: Statistische Untersuchungen über die Bluterkrankheit. *Ztschr. für die gesammte Medicin, hrsg. von F. W. Oppenheim.* Hamburg, 1851, Bd. xlv. S. 145. [Good general and critical account.]

194. Martin, Eduard: Bemerkungen über die Bluterkrankheit. *Jenaische Annalen für Physiologie und Medicin.* Jena, 1851, Bd. ii. S. 307. [Pedigree No. 428.]

195. Meinel, Eug. Aug.: Beiträge zur Geschichte der Haemophilie. *Jenaische Annalen für Physiologie und Medicin.* Jena, 1851, Bd. ii. S. 293. [Pedigree Nos. 600, 601.]

196. Poland, Alfred: (1) Haemorrhagic diathesis, nitric acid, recovery, relapses; (2) haemorrhagic diathesis, actual cautery, recovery. *Guy's Hospital Reports.* London, 1851, second series, Vol. vii. p. 329. [Pedigree No. 429.]

197.* Schwarz: Beiträge zur Heilkunde von der Gesellschaft prakt. Aerzte in Riga. 1851, Bd. i. S. 537. [Not found. This reference is given in Grandidier's monograph. On consulting a copy of the above journal in the library of the École de médecine in Paris it was found that Bd. i. was published in 1849 and Bd. iii. in 1851. In neither could Schwarz's publication be found. The volumes do not extend to 500 pages.]

198. Bailey: Cases of haemorrhage from the umbilicus after the separation of the funis. *The American Journal of the Medical Sciences.* Philadelphia, 1852, new series, Vol. xxiii. p. 432. [Two children affected in a family of four.]

199. [Felt]: Extraordinary bleeders. *The Boston Medical and Surgical Journal.* Boston, 1852, Vol. xlv. p. 85. [Abstract from Felt's *History of Ipswich* published in 1834. It refers to four bleeder families living in Hamilton, once a part of Ipswich. At the time that Felt wrote there were five living bleeders in the town. Felt describes the symptoms of the disease accurately, and says it first appeared in the Appleton family who brought it with them from England. See Hay's case, pedigree No. 408.]

200. Van Hasendonck: Observation de diathèse hémorrhagique, haemophilia. *Annales de la Société de médecine d'Anvers.* 1852, p. 284. [Case of a woman aged 32, suffering from epistaxis during the sixth month of pregnancy. She had previously bled from a cut on the hand: no family history.]

201. Minot, Francis: On haemorrhage from the umbilicus in new born infants with an analysis of 46 cases. *The American Journal of the Medical Sciences.* Philadelphia, 1852, new series, Vol. xxiv. p. 310. [Not haemophilia.]

202. Pickells: Haemorrhagic diathesis exemplified in two brothers with remarks on its hereditary tendency. *The Edinb. Med. and Surg. Journal.* Edinburgh, 1852, Vol. lxxvii. p. 1. [Male aged 20, died from haemorrhage from his wrist which had been gashed by a reaping hook. His brother, aged 14, died from haemorrhage from cut thumb. No doctor present in either case. No tendency to bleed before and none in relatives.]

203. Gintrac, E.: Cours théorique et clinique de pathologie interne et de thérapie médicale. Paris, 1853, Tome iii. p. 110. [A long and exhaustive account of haemorrhage in its different forms, two original cases described in two brothers: (1) Stanislaus N., born 1810, subject to epistaxis and bruising. At 12 haematuria and again at 18. At 7 spontaneous swelling of knee, and later of ankles and elbows. Tendency to bleeding stopped at 20, but at 22 he developed obscure nervous phenomena and died of "cerebral congestion" at 24: no autopsy. (2) younger brother Aristide, also had haemorrhages from nose and mouth, ecchymoses and swellings of his joints: severe bleeding after tooth extraction, death at 20 of pulmonary disease.]

204. Johnson, Z.: Notes of a singular development of purpura in a remarkable purpuric diathesis. *The Dublin Med. Press.* Dublin, 1853, Vol. xxix. p. 98. [Two brothers suffering from purpura, which, along with scurvy, was common in the locality at that time.]

205. Roger, Henri: De l'hémorrhage ombilicale après la chute du cordon. *L'Union médicale.* Paris, 1853, Tome vii. pp. 138, 142, 146. [Female aged 12 days, as in title.]

206.* Tripplin: In Bibliotheka warzawska. 1853, Bd. iv. S. 79. [Not found. Reference in Grandidier's monograph.]

207. Armstrong: (Fatal haemorrhage). *Transactions of the Belfast Clinical and Pathological Society for the Session* 1853—54. Belfast, 1854, p. 66. [Tooth extraction in male, aged 28, who was subject to haemorrhage from trivial causes: death.]

208. Lees, Cathcart: Cases of haemorrhage from the bowels, lungs and nose, in which the ergot of rye was successfully used. *The Dublin Hosp. Gazette.* Dublin, 1854, Vol. i. p. 289. [Three cases: (1) enteric fever, (2) spotted fever, (3) pulmonary apoplexy.]

209. Virchow, R.: Die Bluterkrankheit, Beiträge zur speciellen Pathologie und Therapie. Separat-Abdruck aus dem *Handbuche der speciellen Pathologie und Therapie.* Erlangen, 1854, S. 263. [General account.]

210. Zaar [J.] P. [M.]: De Haemophilia. *Inaug. Diss.* Berolini [1854]. [General account, with the case of a boy who had suggillations and bleeding from the gums. Father not affected. Mother had menorrhagia.]

211. Abt: Terpentinöl gegen Blutungen. *Medicinisches Correspondenz-Blatt des Württembergischen ärztlichen Vereins.* Stuttgart, 1855, Bd. xxv. S. 124. [Three cases: (1) male, aged 18, with great epistaxis and petechiae; (2) female, aged 23, epistaxis and petechiae; (3) male, aged 9, who had always bled from slight injuries, although his parents and siblings were not bleeders. He cut his finger and bled profusely. Oil of turpentine was exhibited with success in all three cases.]

212. Fischer, C.: Tödtlicher Ausgang einer Zahnextraction bei einem Bluter. *Zeitschrift für Wundärzte und Geburtshelfer.* Stuttgart, 1855, Bd. viii. S. 96. [Male aged 18, tooth extracted, followed by great haemorrhage which could not be arrested, death. No other data.]

213. [Friedreich, J. B.]: *Blätter für gerichtliche Anthropologie für Aerzten und Juristen von J. B. Friedreich.* Nürnberg, 1855, Jahrg. vi., Heft v. S. 43. [General account of haemophilia from forensic standpoint.]

214. Grandidier, Ludwig: Die Haemophilie oder die Bluterkrankheit nach eigenen und fremden Beobachtungen monographisch bearbeitet. Leipzig (Otto Wigand), 1855, 159 pp. [Pedigree Nos. 373, 375, 376, 480, 484, 485, 486, 487, 488.]

215. Heider: Ueber Blutungen aus den Alveolen. *Oesterreichische Zeitschrift für praktische Heilkunde.* Wien, 1855, Bd. i. S. 377. [No family history.]

216. Hoffmanns: Ein Bluter stirbt an einer leichten Hautwunde. *Deutsche Ztschr. f. die Staatsarzneikunde.* Erlangen, 1855, Bd. vi. S. 174. [Male, as in title. In youth was subject to bleeding, no family history.]

217. Schnepf, B.: Recherches historiques sur l'hémophilie. *Gaz. médicale de Paris.* Paris, 1855, pp. 671, 707, 733. [Epistaxis.]

218. Smith, Stephen: Remarks on haemorrhage from the umbilicus of infants, with a table of seventy-eight cases. *The New York Journal of Medicine and the Collateral Sciences.* N.Y. 1855, Vol. xiv. p. 73. [As in title, no evidence of haemophilia.]

219. Vezin, Hermann: Tod durch Blutung aus der Nabelschnur. *Vierteljahrsschr. für gerichtliche und öffentliche Medecin.* Berlin, 1855, Bd. vii. S. 336. [Male infant died of umbilical haemorrhage shortly after birth, mother alleged to have come from bleeder family. Two siblings affected.]

220. Weichelt: De diathesis haemorrhagica hereditaria. *Diss. inaug. med.* Berolini, 1855. [General account. No new cases.]

221. Erichsen, J. E.: Extravasation of blood into the calf of the leg. *The Lancet.* London, 1856, Vol. i. p. 511. [Male, 34, feeble and emaciated. Haematoma formed after spraining calf and extended from popliteal space to heel. Incision; great haemorrhage, incipient gangrene, amputation of thigh, death, no family history.]

222. Hobart, Henry S.: Case of haemorrhagic diathesis. *The Dublin Quarterly Journal of Med. Science.* Dublin, 1856, Vol. xxi. p. 239. [Male in whom wounds sometimes bled freely, stoppage of haemorrhage when suppuration started.]

223. Huss, Magnus: Haemophilia. *Hygiea.* Stockholm, 1856, Bd. xviii. S. 129. [Female aged 23. Up to 19 healthy. At this age was severely struck on the head, and in a fit following, again struck her head repeatedly. Blood sweated out of the hair follicles on each side of coronal suture, no visible injury. Haemorrhage from eyes, ear and mouth. This state continued with attacks of torpor, hemiplegia and fits, any excitement determining a haemorrhage. No family history.]

224. Jacoby, A. L. A.: De haemophilia. *Inaug. Diss.* 8°, Halis Sax., 1856. [Male aged 54, always bled freely. During an attack of toothache with swelling of the gum, leeches were applied and the bites bled some hours. Scarification of gums led to haemorrhage which lasted some days. Later he was in the hospital with headache, weakness, and an affection of the eye. No history of haemophilia in the family.]

225. Miller, John: Case of haemorrhagic diathesis in which the bleeding from the gum was arrested only by the extraction of the tooth in its immediate vicinity. *The Edinburgh Med. Journ.* Edinburgh, 1856, Vol. I. p. 638. [Middle aged male with past history of bleedings, no family history.]

226. Salter, Hyde: On haemorrhagic diathesis (Clinical lecture). *The Med. Times and Gazette.* London, 1856, Vol. xxxiii. p. 253. [References without value.]

227. Sewell, S. C.: Haemorrhagic diathesis. *Medical Chronicle.* Montreal, 1856, Vol. iv. p. 1. [Short account of two families of first cousins children of a brother and sister who themselves had no disposition to bleeding. Two or three out of each family have died of traumatic haemorrhage from slight injuries and others have been at death's door from bleeding; one, a boy, bled five days from scratch of hand and died six months later from slight wound. Insufficient account. In Cumberland, Ottawa.]

228. Thore (fils): Observation à l'hémophilie. *Gazette médicale de Paris*, 1856, p. 653. [Boy. At 18 months large ecchymoses on buttock followed by others especially on legs. At 3 he fell and ruptured fraenum of upper lip and bled for three days. At 4 he bruised his forehead and bled four days; at 5 severe haemorrhage from wound caused by a bit of glass; at 6 injured his lip with a piece of broken plate and lost 1 lb. of blood in one day. White swelling of knee. Has never had epistaxis. No family history of bleeding.]

229. Uhde, C. W. F.: Beiträge geburtshilflichen Inhalts. (4) Verblutung eines neugebornen Kindes (Bluterkrankheit). *Monatsschrift für Geburtskunde und Frauenkrankheiten.* Berlin, 1856, Bd. viii. S. 24. [Male infant, purpura, death.]

230. Wunderlich, C. A.: (Habituelle haemorrhagische Diathese) in *Handbuch der Pathologie und Therapie.* Stuttgart, 1856, 2te Aufl., Bd. iv. S. 586. [General account.]

231. Knolz, J. T.: Tödtlicher Ausgang einer leichten Hautverletzung bei einem Bluter. *Oesterreichische Zeitschrift für praktische Heilkunde.* Wien, 1856—7, Bd. II. S. 645. [Male bled for 45 hours from wound on cheek, death.]

232. Brandmeyer, A. H.: Ein Fall von Diathesis haemorrhagica. *Organ f. die gesammte Heilkunde hrsg. von dem Verein rheinischer Medico-Chirurgen.* Berlin, 1857, Bd. vi. S. 97. [Copious epistaxis and petechiae in a male aged 21. No family history.]

233. Gould: Family of bleeders. *The Boston Med. and Surg. Journal.* Boston, 1857, Vol. lvi. p. 500. [Pedigree No. 538.]

234. Helbich: O Krwaweach. Pamiętnik Towarzystwa Lekarskiego Warszawskiego. Warszawa, 1857, Vol. xxxvii. p. 57. [General account, with report of a case communicated to Helbich by Dr Graer. It was that of a boy, aged 4, with abscess on the jaw, incision, great haemorrhage. Helbich's own case was that of a lay brother with haemorrhage from the neck from incision into a steatoma. Treatment by ligation. Of no permanent value.]

235. Laveran: Note sur un cas d'hémophilie avec leucocythémie et alteration de la rate. *Gaz. hebdomadaire de Méd. et de Chirurgie.* Paris, 1857, Tome iv. p. 621. [Soldier, aged 22, with repeated epistaxis for eight months, cachexia, leukaemia, death.]

236. Lemp, C. C. B.: De haemophilia nonnulla adjecto morbi specimine rariori. *Diss. inaug. med.* Berolini [1857]. [Pedigree No. 491.]

237. Schrey, Julius Aemilius: De haemophilia. *Diss. inaug. med.* Berolini [1857]. [Pedigree No. 441.]

238. Townsend, W. E.: Haemorrhagic diathesis. *The Boston Med. and Surg. Journal.* Boston, 1857, Vol. lv. p. 447. [Male aged seven weeks, epistaxis, wasting, swelling of extremities, death. Two sibs died in infancy of general haemorrhage.]

239. Trautmann: De haemorrhagia hereditaria. *Inaug. Diss.* Berlin, 1857. [General account. No original cases cited.]

240. Jenkins, Foster: Report on the spontaneous umbilical haemorrhage of the newly born. *The Transactions of the American Med. Assoc.* Philadelphia, 1858, Vol. xi. p. 263. [Exhaustive account of bleeding from navel.]

241. Finger: Haemophilie. *Oesterreichische Zeitschrift für praktische Heilkunde, ausserordentliche Beiläge zum 5ten Jahrgang.* Wien, 1859, S. 21. [Long account of haemophilia containing description of a Jew, aged 23, suffering from epistaxis. He bled severely from leech bites at 6 and at 10 years, but never had spontaneous bleedings. Has two daughters not affected. His father had epistaxis in youth and died of haemoptysis in middle age. His paternal grandfather died in middle age of cerebral haemorrhage.]

242. Heymann: Ein Fall von Hämophilie. *Virchows Archiv f. path. Anatomie und Physiologie.* Berlin, 1859, Bd. xvi. S. 182. [Pedigree No. 573.]

243. Leudet, E.: Remarques sur la diathèse hémorrhagique qui se manifeste quelquefois dans le cours de la phthisie pulmonaire et dans d'autres affections aigus ou chroniques. *Gaz. méd. de Paris,* 1859, Tome xiv. 3 S., pp. 814, 829. [Title explanatory—not haemophilia.]

244. Parrot, Jules: Étude sur la sueur de sang et les hémorrhagies neuropathiques. *Gaz. hebdomadaire de Méd. et de Chirurgie.* Paris, 1859, Tome vi. pp. 633, 644, 678. [Not haemophilia.]

245. Benavente, Mariano: Diatesis hémorrágica hereditaria. *El siglo medico (Boletin de medicina y gaceta medica).* Madrid, 1860, Tome vii. No. 331, p. 289. [Pedigree No. 424.]

246. Germain: Ablation d'un sarcocele; hémophilie; mort. *Gaz. hebdomad. de Méd. et de Chirurgie.* Paris, 1860, Tome vii. pp. 1, 38. [No previous or family history of bleeding. *Vide* No. 252.]

247. Laycock, Thomas: Case of aneurism of the aorta opening into the sinus arteriosus of the R. ventricle. Hepatic and splenic disease; consecutive haemophilia. *Edinb. Med. Journal.* Edinb. 1860, Vol. v. p. 36. [Male aged 56, as in title, healthy until 50, not haemophilia.]

248. Richardson, B. W.: On the medical history and treatment of diseases of the teeth and adjacent structures. London, 1860, p. 27. [General account of the haemorrhagic diathesis in its bearings on dental practice; no original cases.]

249. Wunderlich, C. A.: Pneumonie bei habitueller haemorrhagischer Diathese mässigen Grades. *Archiv der Heilkunde.* Leipzig, 1860, Bd. i. S. 89. [Male, 35, as in title. At 32 had haemarthrosis.]

250. Elsässer: Bericht über die Ereignisse in der Gebäranstalt und in der Hebammenschule des Catharinen Hospitals in Stuttgart vom 1 Juli 1859 bis zum 30 Juni 1860. *Med. Correspondenz-Blatt des Württembergischen Ärztlichen Vereins.* Stuttgart, 1861, Bd. xxxi. S. 25. [Female, ophthalmia neonatorum, haemorrhage from leech bite, umbilical and intestinal haemorrhage, death.]

251. Gavoy, E.-A.: Hémophylie ou diathèse hémorrhagique. *Thèse de Strasbourg.* 1861. [Boy, aged 14, epistaxis, three days bleeding from small cut on knee, died of epistaxis in Strasburg military hospital. Autopsy. Father and grandfather said to have been liable to haemorrhages.]

252. Germain: Observation d'hémophilie. *Gaz. hebdomadaire de médecine et de Chirurgie.* Paris, 1861, Tome viii. p. 119. [Same case as Bibl. No. 246.]

253. Resal, Victor Antoine: Quelques pages sur l'hémophilie en général et spécialement sur l'hémophylie spontanée. *Thèse de Paris.* 1861, 81 pp. [Three cases: (1) Epistaxis frequently fatal in numerous members of a family. (2) Female aged 22; haemoptysis every morning for 12 years; pregnant. Her father healthy. Mother married twice. Five male children of first marriage died of haemorrhage of a source not stated; thirteen children by second marriage; twelve died of haemoptysis, the only survivor being Resal's case. (3) Male aged 44, haemorrhage from the gums. His 20 sibs unaffected. (It was by the wholesale addition of cases of this kind that the figures of haemophilia were swelled by Grandidier.)]

254. Finger: Ein Fall von Hämophilie. *Oesterreichische Zeitschrift für praktische Heilkunde.* Wien, 1862, Bd. viii. S. 273. [Pedigree No. 578.]

255. Foot, A. W.: Cases in medicine and surgery. *Dublin Med. Press.* 1862, Vol. xlvii. p. 238. [(1) Male, aged 10, haemorrhage from bite of tongue at 5, from tooth extraction and from slight cut on finger. Haematuria and melaena, no details: in hospital with epistaxis, pain in joints and oedema. One brother died after tooth extraction. Evidence of haemophilia insufficient. (2) Child bleeding from lip.]

256. Laycock, Thomas: Clinical lectures on the physiognomical diagnosis of disease. *The Med. Times and Gazette.* London, 1862, Vol. i. p. 499. [On p. 500, few general remarks on haemorrhagic diathesis.]

257. Momberger, R.: Beitrag zur Lehre von der Haemophilie. *Inaug. Diss.* Giessen, 1862. [Male, aged 14, epistaxis and ecchymoses, tuberculosis of joints, no reliable family history.]

258. Wijmans, M.: De Bloederziekte. *Inaug. Diss.* 8°, Leyden, 1862. [Pedigree Nos. 514, 515.]

259. Winkler, A. A.: De haemophilia. *Inaug. Diss.* Berolini [1862]. [The case of Otto Rothacker, aged 12, in Romberg's clinic. At the age of 1 he had spontaneous and traumatic haemorrhages often lasting for eight days. After his first bleedings rheumatic pains and great swelling of the knee occurred. Convulsions resembling epilepsy were prominent symptoms and preceded and attended the haemorrhages. His parents were alive and healthy but father often had epistaxis and one of the patient's brothers bled to death at the age of 3 from the puncture of an ecchymosis on the head. Under the name Ludovicus Rothacker, this patient is again described in C. Beier's dissertation, Bibl. No. 274, additional information being added. He was also described by R. Otte, Bibl. No. 279.]

260. Adelmann: in Grandidier. *Vide* Bibl. No. 267, S. 331. [Male, aged 30, living in Esthonia, great haemorrhage after tooth extraction, and bleeding from a granulating wound of occiput.]

261. Darblade, Jean-Laurent: De l'hémophilie. *Thèse de Paris*, 1863, 53 pp. [Pedigree No. 499.]

262. Deutsch, M.: Fall von Hämophilie. *Wiener medizinal Halle, Zeitschrift für praktische Aerzte.* Wien, 1863, Bd. iv. S. 218. [14 days after birth a boy exhibited blue elastic swellings in various parts of his body. They all burst, bled and suppurated.]

263. Foot: Haemorrhagic diathesis. *The Dublin Quarterly Journ. of Med. Science.* Dublin, 1863, Vol. xxxvi. p. 449. [Exhibition of viscera of boy described in Bibl. No. 255.]

264. Fritz, E.: De l'hémophilie. *Archives gén. de médecine.* Paris, 1863, Série 6, Tome i. p. 591. [General account, no cases.]

265. Gans, D. S.: Haemorrhagic diathesis. *Cincinnati Lancet and Observer.* Cincinnati, 1863, Vol. vi. p. 652. [Female, aged 25, bled from slight abrasions. Removal of tumour in submaxillary region, rather severe bleeding. No family history.]

266. Gerken, Ludovicus Carolus: De haemophilia. *Diss. inaug. medica.* Berolini [1863]. [Three cases: (1) Male, 17, petechiae, bruises and epistaxis from youth upwards, frequently bled severely when he cut himself. No family history. (2) Female, aged 22, epistaxis and bleeding from slight injuries. (3) Female, aged 19, "in cujus familia haemophilorum dyscrasia erat haereditaria," died of haemorrhage following rupture of the hymen.]

267. Grandidier: Bericht über die neueren Beobachtungen und Leistungen im Gebiete der Hämophilie seit 1854. *Schmidt's Jahrbücher der in- und ausländischen gesammten Medecin.* Leipzig, 1863, Bd. cxvii. S. 329. [Short general account of haemophilia with a description of three new cases by (1) Adelmann, Bibl. No. 260; (2) Henschel, Bibl. No. 268; (3) Merkel, Bibl. No. 270.]

268. Henschel: in Grandidier. *Vide* Bibl. 267, S. 331. [Pedigree No. 481. Also a female 3 months old, who died of epistaxis and bleeding from scratches and vaccination wounds in the course of whooping cough. No family history of bleeding.]

269. Mauthner, Max: Apoplexia cerebralis ex haemophilia. *Wiener med. Wchnschrift.* Wien, 1863, Bd. xiii. S. 71, 90. [Male, aged 26, epistaxis and apoplexy, no family history.]

270. Merkel: in Grandidier. *Vide* Bibl. No. 267, S. 331. [Male, aged 2 days, blood flowed from excoriations on his buttocks, and he died on the 11th day. Parents, brothers and sisters all healthy. Pedigree No. 482.]

271. Römer: Habituelle hämorrhagische Diathese (Bluterkrankheit) ohne erbliche Anlage. *Medicinisches Correspondenz-Blatt des Württembergischen ärztlichen Vereins.* Stuttgart, 1863, Bd. xxxiii. S. 255. [Female, aged 17, started epistaxis and metrorrhagia at 16, purpura, haemorrhage from gums, death.]

272. Sedgwick, W.: On the influence of sex in hereditary disease. *The British and Foreign Medico-Chirurgical Review.* London, 1863, Vol. xxxii. p. 186. [In a family observed by author, three brothers suffered from haemorrhagic diathesis, no details.]

273. Virchow, R.: Eine ältere bisher unbekannte Beobachtung von Hämophilie. *Virchows Archiv f. path. Anatomie und Physiologie.* Berlin, 1863, Bd. xxviii. S. 426. [Reference to case of Bibl. No. 5.]

274. Beier, Conrad: De haemophilia. *Diss. inaug. med.* Berolini [1864]. [Same case as that described by Winkler (Bibl. No. 259), and Otte (Bibl. No. 279), with the additional information that the patient suffered greatly from epistaxis and when six years old he bled for a week after the extraction of a tooth. In 1864 he had great pains in his scapulo-humeral and ankle joints and there was a continuation of the epileptiform convulsions described in Winkler's thesis. Suggillations also occurred on his ankles, thighs and arms.]

275. Stromeyer, Louis: *Handbuch der Chirurgie.* Freiburg i. Br. 1864, Bd. ii. S. 121. [(1) Reference to a bleeder aged 60, who had had all sorts of haemorrhages. In 1856 he sustained a subfascial haemorrhage of L. thigh with paralysis of sciatic nerve; (2) reference to the Hanoverian bleeder boy described by Camman (Bibl. No. 324), and by Reinert (Bibl. No. 309, S. 11.)]

276. Wagner, F.: Haemorrhagic diathesis. *Cincinnati Lancet and Observer.* Cincinnati, 1864, Vol. vii. p. 142. [Three cases: (1) Female, aged 11, epistaxis. (2) Two brothers in one family, one with haemorrhage after picking his teeth, the other with a bruise after injury to back. (3) Two brothers send for doctor after every slight wound. No family history.]

277. Babington, B. G.: Hereditary epistaxis. *The Lancet.* London, 1865, Vol. ii. p. 362. [Epistaxis only, in numerous members of a family during five generations.]

278. Baker, W. Morrant: On a case of the "haemorrhagic diathesis." *Med. Chir. Trans. of the Royal Med.-Chir. Soc. of London.* 1865, Vol. xlviii. p. 205. [Male, aged 23, bleeding on numerous occasions from small pimples, on various parts of his body.]

279. Otte, Richard: Ueber die Bluterkrankheit. Leipzig, 1865, 47 pp. [Same case as that described by Winkler (Bibl. No. 259), and Beier (Bibl. No. 274), with some few additional data.]

280. Saint-Vel, O.: Étude sur l'hémophilie. *L'union médicale.* Paris, 1865, Tome xxvii. pp. 515, 533. [Male, aged 15. Epistaxis from infancy. Bled from a boil on foot, and suffered from pain in R. hip. Reference to haemorrhage in grandfather, maternal uncle and elder brother.]

281. Weber, C. O.: Die Gewebserkrankungen im Allgemeinen und ihre Rückwirkung auf den Gesammtorganismus, in *Handbuch der allg. und spec. Chirurgie von Pitha und Billroth.* Erlangen, 1865, Bd. i. Abt. i. S. 127. [Six lines referring to a case seen some years before.]

282. Bruinsma, G. W.: Jets over bloederziekte. 8°, Leeuwarden, 1866. [Pedigree No. 598.]

283. Fischer, C.: Heftige Blutung nach einer Zahnextraction bei einem Bluter. *Zeitschrift für Wundärzte und Geburtshelfer.* Stuttgart, 1866, Bd. xix. S. 5. [Girl with profuse menstruation. After tooth extraction she bled for five days. Enquiry showed that she came of a haemophilic family. No details.]

284. De Fleury: Observation d'hémophylie; considérations sur cette maladie. *Mémoires et bulletins de la Société médico-chirurgicale des hôpitaux et hospices de Bordeaux.* Paris et Bordeaux, 1866, Tome i. p. 299. [Male, aged 21; had considerable haemorrhage from cut on thumb at the age of 14; haematuria and purpuric spots on thighs and calves when 17; epistaxis and swelling of L. knee. Father died of unknown cause, mother alive but a sufferer from epistaxis. One of patient's sisters has the haemorrhagic diathesis.]

285. Giraudeau, P.-A.: De l'hémophylie. *Thèse de Paris.* 1866. [Male, aged 37. Father born in Guadeloupe, mother in Denmark. They had four children (two boys and two girls) of which patient was the second. At 15 months, haematoma on buttocks; incision gave rise to great haemorrhage. Severe bleedings after trivial accidents. At 15, bleedings less severe but purpura frequent. At 23, cellulitis of leg leading to contracture. Tenotomy of tendo Achillis. Haemorrhage for six days. Very doubtful case. No family history of bleeding.]

286. Sentex: (discussion on de Fleury's paper.) *Mém. et bull. de la Soc. médico-chirurgicale des hôpitaux et hospices de Bordeaux.* Paris et Bordeaux, 1866, Tome i. p. 311. [Male, aged 26, with epistaxis; bruising and haemorrhage from trivial causes. Arm crushed by locomotive, amputation, secondary haemorrhage, death.]

287. Dunn, R. W.: Case of haemorrhagic diathesis. *The Brit. Med. Journ.* London, 1867, Vol. ii. p. 182. [Case of a boy aged 9, of whom such details are given from infancy up, that a diagnosis of haemophilia may be arrived at, but his family history is merely that he was one of six, of whom three are alive. Father had scurvy and rheumatism and the father's brother had epistaxis.]

288. Hodges: Reamputation, haemorrhagic diathesis, death. *The Boston Med. and Surg. Journal.* Boston, 1867, Vol. lxxv. p. 384. [As in title, secondary haemorrhage; male aged 30.]

289. Koch, C.: Ueber Hämophilie. *Inaug. Diss.* Greifswald, 1867. [Female, aged 8, no family history of haemophilia. Purpura and bleeding from an extracted loose incisor. Later, haematuria.]

290. Patton, G. R.: Haemorrhagic Diathesis. *Cincinnati Lancet and Observer.* 1867, Vol. x. p. 724. [Male, aged 7, haemorrhage from bitten tongue, no previous instance of bleeding, no family history.]

291. Schünemann, H.: Extraction eines Zahnes bei einem Bluter, Tod. *Virchows Archiv für patholog. Anatomie und Physiologie und für klin. Med.* Berlin, 1867, Bd. xli. S. 287. [Male, aged 21, death after tooth extraction.]

292. Sedgwick, W.: On the influence of age in hereditary disease. *The British and Foreign Medico-Chirurgical Review.* London, 1867, Vol. xxxix. p. 466. [On p. 479 author discusses influence of age in haemorrhagic diseases and gives general account of umbilical haemorrhage with case of his own on p. 481.]

293. Smith: Case of haemorrhagic diathesis. *The Brit. Med. Journ.* London, 1867, Vol. II. p. 6. [Male, aged 6, one of twelve children, of which seven are alleged to have died of haemorrhage from nose and mouth. At one month spots like bruises appeared. Of late epistaxis.]

294. Spahn, W.: Drei Fälle von Haemophilie. *Inaug. Diss.* Giessen, 1867. [Three cases: (1) male, aged 10, ecchymoses, pain in shoulder and knee joints, the latter being swollen, later anasarca, epistaxis and death. Autopsy. No haemorrhage in family; (2) male, aged 7, umbilical bleeding immediately after birth. Epistaxis, haematemesis and haematuria. No joints affected. Death. No family history; (3) female infant, haemorrhage from excoriations, death: mother syphilitic.]

295. Thoms: Hereditary tendency to the haemorrhagic diathesis. *The Medical Record.* New York, 1867—8, Vol. II. p. 19. [No cases.]

296. Broca, P.: Puissante intervention du galvanocautère dans un cas d'hémophilie opiniâtre. *Journal de médecine et de chirurgie pratiques à l'usage des médecins praticiens.* Paris, 1868, Tome XXXIX. p. 401. [Solitary instance of haemorrhage from the R. eyebrow in a male aged 30.]

297. Buss, Henry: Misplacement of the spleen in connexion with the haemorrhagic diathesis. *The Med. Times and Gaz.* London, 1868, Vol. II. p. 530. [Male, aged about 20, death from rupture of displaced spleen.]

298. Delmas: Hémophilie héréditaire remontant à la 4me génération. *Mémoires et bulletins de la société médico-chirurgicale des hôpitaux et des hospices de Bordeaux*, 1868, Tome III. p. 336; also in Delmas, Manuel d'hydrothérapie, Paris, 1885, p. 448. [Pedigree No. 474.]

299. Durham, Arthur E.: Case of haemorrhagic diathesis. *Guy's Hospital Reports.* London, 1868, 3rd series, Vol. XIII. p. 489. [Pedigree No. 394.]

300. Heath, Christopher: Two cases of hereditary haemorrhagic diathesis. *The Brit. Med. Journal.* London, 1868, Vol. I. p. 25. [Pedigree No. 540.]

301. Roth, Theodor: Ueber die Nabelblutungen der Säuglinge. *Journal für Kinderkrankheiten.* Erlangen, 1868, Bd. LI. S. 1. [General account and three cases as in title. On p. 9 reference to a male, aged 22, who came of a healthy family but who himself bled easily. After the extraction of two molars he sustained an enormous haemorrhage, but ultimately recovered. By his two wives he had 11 children all free from haemophilic taint.]

302. Woelky, J. E. B.: Ueber Haemophilie. *Inaug. Diss.* Berlin, 1868. [Pedigree No. 383.]

303. Anon.: Prince Leopold. *The British Medical Journal.* London, 1868, Vol. I. p. 125. [Account of one of his haemorrhages.]

304. Assmann, Richard: Die Hämophilie. *Inaug. Diss.*, sm. 8°. Berlin, 1869. [Pedigree No. 423.]

305. Brenner und Januszkjewitsch: Galvanokaustische Stillung einer nach dem Ausziehen eines Zahnes entstandenen Blutung. *St Petersburger med. Ztschr.* St Petersb., 1869, Bd. XVII. S. 15. [Case of a female aged 50.]

306. Castan, A.: De l'hémophilie. *Montpellier médical, journal mensuel de médecine*, 1869, Tome XXIII. p. 289. [Long general account of haemophilia containing a short description of two brothers, the one aged 10 (stated in another place to be 6) who had bleeding from the gums and suffusions of blood. Haemorrhage after leech bites behind the ear, lasted 25 days; painful joints. The other, aged 4, had had bleedings from the age of eight months. Epistaxis daily, blood tumours on body and limbs, no joint pains. Maternal grandmother had haematemesis from 28th—40th year, but died at 80.]

307. Cousins, J. Ward: A case of haemorrhagic diathesis. *The Medical Times and Gazette.* London, 1869, Vol. II. p. 277. [Pedigree No. 580.]

308. Higginbotham: Zwei Beobachtungen über Bluter. *St Petersburger medicinische Zeitschrift.* St Petersburg, 1869, Bd. XVI. S. 111. [Two cases: (1) female, aged 15, who lost 3000 ounces of blood from the genitals in 7½ months, as the result of a brutal rape; (2) male, aged 27, eight days' haemorrhage from two carious teeth; successful exhibition of "alter starker Porter, double Stout genannt."]

309. Reinert, Heinr.: Ueber Haemophilie. *Inaug. Diss.* Göttingen, 1869. [Pedigree No. 501.]

310. Tamele, J.: Rodina krvácivá a případ krvácivosti. *Časopis lékařův českých.* v. Praze, 1869, Vol. VIII. p. 153. [Refers to three alleged typical bleeders, viz. one male and two females. Our abstract made by Professor Maixner in Prag contains reference only to the male who was repeatedly treated by Tamele for subcutaneous and intramuscular haemorrhages, also for cerebral haemorrhage and for almost fatal haemorrhage from leech bites and other injuries. One maternal uncle described as a typical bleeder.]

311. Parker, R.: Injury to the urethra in a child, the subject of haemorrhagic diathesis. *The Medical Times and Gazette.* London, 1870, Vol. i. p. 728. [Male, aged 12, ruptured urethra; no evidence of diathesis; vague family history of haemorrhage.]

312. Traneus, L.: Haemophilia in a child with effusion of blood into the ventricles. *St Louis Med. and Surg. Journal.* St Louis, 1870, n. s. Vol. vii. p. 535. [Male, aged 2, umbilical haemorrhage, death.]

313. Waterhouse, Frederick: Mechanical injuries in a case of congenital purpura. *The Brit. Med. Journal.* London, 1870, Vol. i. p. 128. [Pedigree No. 560.]

314. Waterhouse, Frederick: Cases of inherited purpura or hereditary haemorrhagic diathesis. *The Brit. Med. Journal.* London, 1870, Vol. ii. p. 679. [Pedigree No. 560. Continuation of history of cases in Bibl. No. 313.]

315. Higgens, Charles: Notes of two cases of haemorrhagic diathesis. *Trans. of the Royal Medical and Chirurgical Soc. of London*, 1871—5, Vol. vii. p. 268. [Abstract of twelve lines with reference to six bleeders, no details.]

316. Catlin: On some of the difficulties and accidents which happen in the practice of dental surgery. *Trans. of the Odontol. Soc. of Great Britain.* London, 1871, Vol. iii. n. s. p. 131. [Few remarks, no cases, no value.]

317. Delmas: Observation d'un cas d'hémophilie traité avec succès par la médication hydrothérapique. *Mém. et bull. de la société médico-chirurgicale des hôp. et des hospices de Bordeaux*, 1871, Tome vi. p. 164; also in Delmas, Manuel d'hydrothérapie, Paris, 1885, p. 452. [Case of Pétronille Béron, aged 32. She was well up till 15 years of age, when after the onset of menstruation she developed menorrhagia, epistaxis and haemoptysis. This occurred again when she was married, at 19, again at 24 after her first confinement, and again at 28 after her fourth confinement. No family history of haemophilia. See remarks on Franck (Bibl. No. 326).]

318. Grandidier, L.: Die freiwilligen Nabelblutungen der Neugeborenen. Cassel, 1871. [General account.]

319. Harris, R. P.: Reviews and books, notes. *Philadelphia Medical Times.* Philadelphia, 1871—2, Vol. ii. p. 273. [Trifling reference in the course of a book review, no value.]

320. Legg, J. Wickham: Two cases of haemorrhagic diathesis in women. *The Med. Times and Gazette.* London, 1871, Vol. ii. p. 673. [Two cases in females: (1) unaffected up to time of marriage at 18, *post partum* haemorrhage, epistaxis, excessive bleeding from cuts, swollen knee. No family history of bleeding except in mother and a daughter, both of whom had epistaxis; (2) married at 19, ill with epistaxis and flooding, not considered to be haemophilia by author.]

321. Legg, J. Wickham: Four cases of haemophilia. *St Bartholomew's Hosp. Reports.* London, 1871, Vol. vii. p. 23. [Pedigrees Nos. 464, 465, 466, 467.]

322. Poncet, A.: Observation d'hémophilie. *Lyon médicale*, 1871, Tome viii. p. 785. [Male, aged 16, enlarged glands of neck, rheumatic pains in joints, epistaxis, severe haemorrhage following incision into a black swelling of knee, death from haemorrhage following the sloughing of tissue after the twelfth application of the actual cautery. Autopsy. Evidence of haemorrhage into joints. No family history.]

323. Brigstocke, C. A.: Cases of haemophilia. *The Brit. Med. Journal.* London, 1872, Vol. ii. p. 122. [Pedigree No. 439.]

324. Cammann: in Grandidier's paper (Bibl. No. 327, p. 91.) [Same case as that described by Strohmeyer (Bibl. No. 275) and Reinert (Bibl. No. 309, p. 11).]

325. Cantani, Arnaldo: L' emofilia ed il Penghawar Djambi. *Il Morgagni.* Napoli, 1872, Tome xiv. p. 305. [Pedigree No. 374.]

326. Franck, François: De l'hémophilie. *Bordeaux médicale.* Bordeaux, 1872, Tome i. pp. 189 and 194. [Under the name Jeanne Mérieux, aged 34, Franck, in reporting a lecture by Gintrac, gives an account of a case which is identical in every detail with that of Pétronille Béron described by Delmas (see Bibl. No. 317).]

327. Grandidier: Bericht über die neuern Beobachtungen und Leistungen im Gebiete der Hämophilie seit dem Jahre 1863. *Schmidt's Jahrbücher der in- und ausländischen gesammten Medicin.* Leipzig, 1872, Bd. cliv. S. 81. [Contains account of Schulz's and Cammann's Cases.]

328. Grossheim: Macht "Bluterkrankheit" zum Militärdienst unbrauchbar. *Deutsche militäraerztliche Zeitschrift.* Berlin, 1872, Bd. i. S. 319. [Case of a recruit who bled for eight days after the extraction of a carious tooth. He also had great haemorrhage from a furuncle on his arm and also epistaxis. See also M. Fischer, Bibl. No. 541 and pedigree No. 426.]

329. Güntner: Unterbindung des rechten Carotis communis wegen nicht zu stillenden Blutungen aus der Wundfläche eines abgetragenen Mandel bei einem Bluter und mit Syphilis behafteten Manne. *Oesterreichische Zeitschrift für praktische Heilkunde.* Wien, 1872, Bd. XVIII. S. 841. [Man aged 31 as in title, liable to haemorrhage from childhood.]

330. Inman, T.: On male chlorosis and allied diseases. *The half yearly abstract of the Medical Sciences* (Rankin). London, 1872, Vol. LIV. p. 24. [As in title, not haemophilia.]

331. Legg, J. Wickham: A treatise on Haemophilia sometimes called the hereditary haemorrhagic diathesis. London, 1872, 158 pp. [The most authoritative publication in English on the general subject of haemophilia.]

332. Namias, Giacinto: Intorno alla emofilica. *Giornale veneto di scienze mediche.* Venezia, 1872, 3 s. Tomo XVII. p. 33. [Clinical lecture with demonstration of two cases, one being a male aged 39, suffering from epistaxis and pains in the joints.]

333. Schulz: in Grandidier (see Bibl. No. 327 p. 90). [Female. From early childhood had epistaxis and bleeding from the ear, also ecchymoses of skin, gums, palate, death at the age of 13 from subarachnoidal haemorrhage. Father had aortic aneurism, mother anaemic.]

334. Walker, J. W.: On haemophilia. *The Brit. Med. Journal.* London, 1872, Vol. I. p. 605. [Pedigree No. 477.]

335. Brébant: Un cas d'hémophilie. *Bulletins de la société médicale de Reims.* Reims, 1873, No. 12, p. 103. [Male, aged 26, suffering from extensive inflammatory condition of face associated with bloody discharge.]

336. Grandidier: Weitere Mittheilungen über Hämophilie. *Schmidt's Jahrbücher.* Leipzig, 1873, Bd. CLVII. S. 35. [A few notes on cases published by Wickham Legg, Güntner, Brigstocke, etc.]

337. Legg, J. Wickham: The urine in Haemophilia. *The Brit. Med. Journal.* London, 1873, Vol. I. p. 141. [Pedigree No. 469.]

338. von Meurers: Die haemorrhagische Diathese und ihr Vorkommen bei Recurrens. *Inaug. Diss.* Berlin [1873]. [Account of a case of haemorrhagic diathesis with recurrent fever.]

339. Moreau, Joseph: De l'hémorrhagie consécutive à l'extraction des dents. *Archives générales de médecine.* Paris, 1873, VI. sér. Tome XXII. pp. 149, 226. [Analysis of a large number of cases of haemorrhage after tooth extraction in connection with haemophilia and otherwise.]

340. Sotti, Leandro: Sopra un caso di emofilia grave. *Gazzetta Italiana provincie venete.* Padova, 1873, T. XVI. p. 9. [Long account of an hysterical girl of 19 suffering from menstrual irregularities, haematemesis, etc., and cured by kreosote, ergotin and electricity; not haemophilia.]

341. Austin, F. J.: A résumé of the present state of our knowledge of haemophilia. *Canada Med. Record, a Monthly Journal of Medicine and Surgery.* Montreal, 1874—5, Vol. III. p. 549. [General account, no cases.]

342. Chase, S. B.: Hemorrhagic diathesis. *The Medical and Surgical Reporter.* Philadelphia, 1874. Vol. XXXI. p. 446. [Case of an American lawyer, aged 30, nearly bled to death from extraction of incisor tooth, later great haemorrhage after removal of a first molar, and on another occasion of a second molar. The extraction of a wisdom tooth by Chase led to haemorrhage for several weeks. Patient also had severe epistaxis, and his three year old daughter had profuse uterine haemorrhage three times. His brother also bled from kidneys and bowel for more than a year.]

343. Gissler und Wentzel: Eine Lammbluttransfusion. *Aerztliche Mittheilungen aus Baden.* Karlsruhe, 1874, Bd. XXVIII. S. 69. [A young man bleeding from mouth and anus, gave history of haemophilia in his family. One brother had haemorrhage after tooth extraction: another died after the same. Patient very weak with rapid pulse and subnormal temperature. Transfusion of lamb's blood, recovery. See also Max Fischer, Bibl. No. 541 and Pedigree No. 426.]

344. Hadlock: Haemorrhagic diathesis. *Clinic.* Cincinnati, 1874, Vol. VII. p. 241. [Mulatto male, aged 7, died 48 hours after tooth extraction. Uncle died from slight cut with a scythe, father from a scratch with a briar, insufficient account.]

345. Holton, William M.: Constitutional haemorrhage. *The American Journal of the Medical Sciences.* Philadelphia, 1874, n. s. Vol. LXVII. p. 414. [Reference to a few members of T. D. Dunn's fourth family. Pedigree No. 433.]

346. Lemoine: Cas d'hémorrhagie cérébrale à forme insolite. *Gazette des hôpitaux.* Paris, Année 1874, p. 299. [Male, aged 69, epistaxis and cerebral haemorrhage. Had an hereditary disposition to haemorrhage—no details.]

347. Osterloh: Haemophilia hereditaria. *Berichte und Studien aus dem königl. sächs. Entbindungs-Institute in Dresden.* Leipzig, 1874, Bd. I. S. 220. [(1) female infant, umbilical haemorrhage, purpura, death; (2) not haemophilia, imperfect family history.]

348. Palle (d'Épernay): Hémophilie. *Bulletin société médicale de Reims.* Reims, 1874, No. 13, p. 99. [Trivial account of a male child three months old suffering from epistaxis and ecchymoses. Father bled easily from the nose, and one of his nephews was "subject to frequent haemorrhages," discussion on the treatment.]

349. Ranger, W. G.: On severe haemorrhage after teeth extraction. *St Thomas's Hosp. Reports.* London, n. s. 1874, Vol. VI. p. 121. [Male, middle aged: oozing of blood for a short time from a very loose tooth. A brother died after tooth extraction.]

350. Simon, F.: Recherches sur l'hémophilie. *Thèse de Paris*, 1874. [Pedigree Nos. 512, 513.]

351. Bureau, N.: Essai sur l'hémophilie. *Thèse de Paris.* 1875, No. 112, 45 pp. [Three cases. The first a boy aged 15 attacked for the first time at 12, with epistaxis, haemoptysis, haematemesis, haematuria and ecchymosis. Similar attack at 15. No haemophilia in the family. The second case—a male bleeding after tooth extraction. The third case—a child—died of haemorrhage after lancing gums.]

352. Hakenschmied: *Medicinisch-chirurgisches Centralblatt.* Wien, 1875, Bd. X. S. 206. [History of a Bohemian mountaineer aged 30, suffering from pleuropneumonia. When venesection was proposed he asserted that he bled furiously from trivial cuts and had bled severely from tooth extraction. He was venesected notwithstanding and recovered. Two years later he was stabbed with a hunting knife which entered his R. elbow and shaved off a lamella of bone from the external condyle. Uncontrollable haemorrhage; death.]

353. Langelaan, H.: Een geval van haemophilie met letalen afloop. *Weekblad van het Nederlandsch Tijdschrift voor Geneeskunde.* Amsterdam, 1875, Bd. XI. S. 177. [Girl aged 16. Three sisters healthy, one sister aged 8, chlorotic. Parents, uncles and aunts healthy except one maternal uncle who had epistaxis. The girl herself had ecchymoses, epistaxis, profuse menstruation, fever, haemorrhages into joints and died bleeding from the nose, mouth and eyes.]

354. Lapeyre, Charles Laurent: Recherches sur la nature de l'hémophilie. *Thèse de Paris.* 1875. 42 pp. [Four separate cases. Three females, one male, with epistaxis and other haemorrhages which cannot be considered haemophilic.]

355. MacCormac, William: On some cases of "Bleeders." *St Thomas's Hospital Reports.* London, 1875, Vol. VI. p. 111. [Pedigrees Nos. 448, 450, 451; also case of a male aged 24. At 16 severe bleeding after biting tongue, also epistaxis and bleeding from gums. He died after tapping and subsequent suppuration of a large swelling at lower end of femur. No family history.]

356. Thompson, Sir H. (under the care of): Haemorrhagic diathesis, synovitis of knee relieved. *The Med. Times and Gaz.* London, 1875, Vol. I. p. 634. [Male $6\frac{1}{2}$, frequent epistaxis. Three days' haemorrhage from bitten tongue. Bruised and bled easily. Knees swollen, hot and tender. Two brothers had slight tendency to bleed.]

357. Wagstaffe: Haemorrhage following tooth extraction. *The Lancet.* London, 1875, Vol. II. p. 527. [No family history.]

358. Barlow, Thomas: Case of sporadic haemophilia. *The Medical Times and Gazette.* London, 1876, Vol. II. p. 591. [Pedigree No. 571.]

359. Cadet de Gassicourt: De l'hémophylie. *La France médicale.* Paris, 1876, Tome XXIII. p. 117. [Pedigree No. 559.]

360. Dieulefoy: Du rôle de l'hérédité dans la production de l'hémorrhagie cérébrale. *Gaz. hebdomad. de méd. et de chirurgie.* Paris, 1876, Tome XIII. p. 595. [To show that cerebral haemorrhage is inherited.]

361. Epstein, Alois: Zur Aetiologie der Blutungen im frühesten Kindesalter. *Oesterreichisches Jahrbuch für Paediatrik.* Wien, 1876, Bd. VII. S. 119. [Congenital syphilis.]

362. Jenner, Sir W.: Fatal haemophilia. *The Lancet.* London, 1876, Vol. II. p. 716. [Pedigree No. 553.]

363. Kehrer, F. A.: Die Haemophilie beim weiblichen Geschlechte. *Archiv für Gynaekologie.* Berlin, 1876, Bd. X. S. 201. [One of the principal papers on haemophilia in the female. The author himself records three cases, viz. (1) female, aged 30, epistaxis in youth: first pregnancy normal; second pregnancy associated with uterine and nasal haemorrhages as also in her third pregnancy. Anaemia, abortion, recovery; (2) female, aged 23, aborted three times. In fourth pregnancy, repeated epistaxis and haematuria, abortion in the eighth month; death one month later; (3) female, who in second half of third pregnancy suffered from profuse epistaxis, later metrorrhagia with blood casts of uterus. Severe anaemia, death.]

364. Ledoux, P.: Un cas d'hémophylie avec hémorrhagie de la moelle épinière. *Journal des sciences médicales de Louvain.* Louvain, 1876, Tome I. p. 129. [Pedigree No. 459.]

365. Legg, J. Wickham: A case of haemophilia complicated with multiple naevi (a paper read before the Royal Medical and Chirurgical Society of London). *The Lancet.* London, 1876, Vol. II. p. 856. [Male aged 65 suffering from epistaxis and numerous small naevi on face and body. Epistaxis in the family.]

366. Lehmus: Ein Fall von hämorrhagischer Diathese bei hereditärer Lues. *Berichte und Studien aus dem königl. sächs. Entbindungs-Institute in Dresden über die Jahre* 1874 *und* 1875. Leipzig, 1876, Bd. II. S. 118. [As in title.]

367. Stilling, B.: Grosses Lipom auf dem Rücken eines Bluters. Exstirpation unter Anwendung von Listers Principien der Wundbehandlung. Bedeutende Nachblutung und Blutanhäufung unter der Haut. Heilung ohne Fieber und ohne Eiterung. *Deutsche Med. Wochenschr.* Berlin, 1876, Bd. II. S. 607. [Male aged 58. Severe bleeding after slight injuries. No family history.]

368. Tittel, M.: Ein Fall von Hämathidrosis. *Archiv der Heilkunde.* Leipzig, 1876, Bd. XVII. S. 63. [Not haemophilia.]

369. Fütterer, Robert: Zur Casuistik der Hämophilie. *Inaug. Diss.* Greifswald, 1877. [Male aged $5\frac{1}{2}$ in surgical policlinic in Greifswald. Had bled eight days after removal of carious molar; had spotted eruption on his face some weeks before. In his second year he broke his forearm; slight blood extravasation. Nine months later he fell down stairs and developed an extravasation the size of a thaler (!). A 4 cm. wound with a slate bled freely and required two stitches (!). No evidence of haemophilia.]

370. Gibert: Hémophylie, éruption pétéchiale chez un sujet hémophilique; hémorrhagies dans les cavités buccale, pharyngienne et laryngienne; diphtherite secondaire; mort. *Gazette médicale de Paris.* 1877, 4e sér. T. XVI. p. 529. [Male $5\frac{1}{2}$ years old, born in Havana. No family history.]

371. Grandidier: Die neueren Leistungen im Gebiete der Hämophilie. *Schmidt's Jahrbücher.* Leipzig, 1877, Bd. CLXXIII. S. 185. [Literature only.]

372. Grandidier, Ludwig: Die Hämophilie oder die Bluterkrankheit, nach eigenen und fremden Beobachtungen monographisch bearbeitet. Leipzig (Otto Wigand), Zweite neu bearbeitete Auflage. 1877, 227 pp. [Pedigrees Nos. 373, 375, 376, 480, 481, 482, 483, 484, 485, 486, 487, 488.]

373. Hill, P. E. Fatal case of haemophilia in an infant. *The Brit. Med. Journ.* London, 1877, Vol. II. p. 136. [Child 17 months, ruptured ala of L. nostril, death from haemorrhage. "Haemorrhagic diathesis appeared to run in the family of the mother."]

374. Hutchison, James: no title. *Trans. of the Medical Society of the State of New York for the year* 1877. Albany, N.Y., 1877, p. 268. [Pedigree No. 570.]

375. Lossen, Hermann: Die Bluterfamilie Mampel bei Heidelberg. *Deutsche Ztschr. für Chirurgie.* Leipzig, 1877, Bd. VII. S. 358. [Pedigree No. 389.]

376. Mackenzie, Stephen: Haemophilia, purpura, retinal haemorrhages (purpura of retina), death. *The Medical Times and Gazette.* London, 1877, Vol. I. p. 258. [Female aged 13, bled easily all her life, e.g. three weeks haemorrhage after tooth extraction. She was easily bruised and had epistaxis. Began to menstruate at 13 and was admitted to hospital for severe bleeding, from uterus, which had gone on for three weeks. Temporary recovery, but recurrence of symptoms, ecchymosis, menorrhagia, death. No autopsy. No family history of bleeding.]

377. Newcombe, F. W.: Cases of haemophilia. *Northumberland and Durham Medical Journal.* Newcastle-upon-Tyne, 1877, pp. 91—94. [Pedigree No. 539.]

378. Ritter: Das Verhältniss der temporären Haemophilie der Neugeborenen zu Blutungen in späteren Alter und zu der Bluterkrankheit. *Prager medicinische Wochenschrift.* Prag, 1877, Bd. II. S. 425, 445. [General remarks developing the view that haemorrhagic diathesis of the newly born and haemophilia are not in their essence different.]

379. Rotch: Umbilical haemorrhage. *The Boston Med. and Surg. Journal.* Boston, 1877, Vol. XCVII. p. 390. [One case as in title.]

380. Strong: Haemophilia. *The British Medical Journal.* London, 1877, Vol. I. p. 28. [Female aged 13. Whole account occupies three lines. No value.]

381. Alcina, B. Historia clinica de uno hemofilico. *Cronica oftalmologica.* Cadiz, 1878, Vol. VIII. (Suppl.), p. 45. [Male aged 23 with past history of haemorrhage. Incision into furuncle led to severe and persistent bleeding. Digital compression. Recovery. A brother at the age of 4 cut his tongue and bled to death. Father died of heart disease.]

382. Aquilar y Venegas, Joaquin: Un caso de hemófilis. *La Andalucia medica, Revista médico-quirurgica fotografica y de ciencias accesorias.* Córdoba, 1878, año III. num. I. p. 4. [Two cases. (1) Boy aged 6 who lost a considerable quantity of blood during and after vaccination. Later bled copiously from eight leeches which were applied to the abdomen for gastroenteritis, a poultice which had subsequently been applied being found floating in a pool of blood from the leech bites. Later he fell, knocked out a tooth and bled to death. No family history of haemophilia. (2) Girl aged 23 with severe bleeding after tooth extraction.]

383. Bastard, H.: Hémophylie avec purpura haemorrhagica. *Gazette des hôpitaux.* Paris, 1878, p. 1034. [Menorrhagia in a girl of 13.]

384. Börner, E.: Ueber Bluterkrankheit in ihrer Bedeutung für die Gynaekologie. *Wiener med. Wochenschr.* Wien, 1878, S. 891, 946, 971, 988, 1017. [Cases of uterine haemorrhage not uncommon in gynaecological practice.]

385. Darby, J. T.: Clinical lecture on the haemorrhagic diathesis. *Hospital Gazette.* New York, 1878, Vol. III. p. 231. [Sex and age not stated; dilated superficial venules, death from epistaxis. "The disease was here transmitted through four generations."]

386. Deschamps: Bosse sanguine, hémophilie. *Archives médicales belges, organe du corps sanitaire de l'armée.* Bruxelles, 1878, 3me sér. Tome XIII. p. 262. [Male aged 22, a lancer, developed haematoma, the size of a nut, from slight cut on forehead. Had had a similar swelling on his back from kick by a horse; alleged that he bled easily and had constant epistaxis. His father and eight brothers were similarly affected but in a less degree.]

387. Eppinger, Hans: Haemophilia neonatorum. *Wiener med. Presse.* Wien, 1878, Bd. XIX. S. 798, 864. [Umbilical haemorrhage considered by author to be due to an infection.]

388. Förster, R.: Haemorrhagische Diathese. *Handbuch der Kinderkrankheiten* hrsg. von C. Gerhardt. Tübingen, 1878, Bd. III. S. 245. [Pedigrees Nos. 549, 550.]

389. Hémard: Hémorrhagie consécutive à l'extraction d'une dent chez un hémophile et ayant nécessité la ligature de la carotid primitive: guérison. *Recueil de mémoires de médecine, de chirurgie et de pharmacie militaires.* Paris, 1878, 3e série, Tome XXXIV. p. 490. [Soldier aged 19. A small wound on scalp at age of 7 necessitated ligation of a vessel: frequent and grave epistaxis. At 16, bled severely from tooth extraction. At 19, removal of second upper L. molar, followed by haemorrhage so great that Hémard ligated the carotid on the eighth day, and the wound in the neck also bled violently after the seventh day. Secondary haemorrhage; recovery. No family history of bleeding.]

390. Kidd, Percy: A Contribution to the pathology of haemophilia. *Med. Chir. Trans.* London, 1878, Vol. LXI. p. 243, 1 pl. [Case of a male aged 6 unaffected till 2. Haematoma of forehead from a blow, epistaxis and bleeding from the mouth. Bled freely from cuts, e.g. seven days after a scratch. Under observation for haemorrhage from gums lasting 14 days. Death. Autopsy. Youngest child of family had diarrhoea and is stated to have died of haemorrhage.]

391. Rossignol, Joseph: De l'hémophylie. *Thèse de Montpellier.* 1878. [Male aged 45. At 8, profuse epistaxis, prodromal palpitations, etc. At 14, eight days' haemorrhage from contusion on forehead. At 20, ten days' bleeding from wound of palm. At 22, two days' bleeding from leech bites on contused buttocks. Later, severe haemorrhage from lacerated wound of cheek. Finally, seven days' haemorrhage from finger tip after blow from hammer. Father had epistaxis.]

392. Stuart, J. A. E.: Haematemesis in the newly born child (illustrated by a case associated with the haemorrhagic diathesis in the mother). *Edinb. Monthly Journ.* Edinburgh, 1878–9. Vol. XXIV. p. 1081. [As in title, not haemophilia.]

393. Weigersheim: Einige Mittheilungen über Bluterkrankheit. *Berliner klin. Wochenschrift.* Berlin, 1878, Bd. XV. S. 617. [Pedigree No. 478.]

394. Whitall: Haemorrhagic diathesis. *Hospital Gazette.* New York, 1878, Vol. III. p. 181. [Infant. Umbilical haemorrhage, ecchymoses, bleeding from mouth.]

395. Zeroni, H., sen.: Zwei Fälle von Morbus haemorrhagicus nach Typhus abdom. und ein Fall im Verlauf eines Febris recurrens. *Memorabilien.* Heilbronn, 1878, Bd. XXIII. S. 500. [As in title.]

396. Eppinger, H.: Haemophilia neonatorum. *Medicinisch-chirurgisches Centralblatt.* Wien, 1879, Bd. XIV. S. 195, 219. [Case of haemorrhage in a new born child with micro-organisms in the viscera.]

397. Felsenreich: Haemophilie in der ersten Lebenswoche. *Wiener med. Presse.* Wien, 1879, Bd. XX. S. 242. [Female infant. Death from umbilical haemorrhage on the eleventh day.]

398. Hémard: Hémorrhagie consécutive à l'extraction d'une dent chez un hémophile et ayant nécessité la ligature de la carotid primitive. *Bull. et mêm. de la soc. de chirurgie de Paris.* Paris, 1879, Tome v. p. 392. [Same case as in Bibl. No. 389.]

399. Hirschberg: Retinitis ex haemophilia. *Archiv für Augenheilkunde.* Wiesbaden, 1879, Bd. viii. S. 174. [A young male who bled from injuries and tooth extraction. Haematuria three times. Haemorrhages in retina believed to be haemophilic. His family was said to be haemophilic. No data.]

400. Homolle, G.: De l'hémophilie et de ses relations avec le rhumatisme. *La France médicale.* Paris, 1879, Tome xxvi. p. 1; 9. [Female aged 32. Morbus cordis and ulceration of cervix uteri. Father and two brothers bled abundantly from the least cause.]

401. Immermann, H.: Hämophilie in v. Ziemssen's *Handbuch der speciellen Pathologie und Therapie* Leipzig, 1879, 2te Aufl. Bd. xiii. 2te Hälfte, S. 519. English Translation, *Cyclopaedia of the Practice of Medicine*, edited by H. v. Ziemssen. London, 1878, Vol. xvii. p. 3. [Exhaustive general account.]

402. Kloss, Fritz: Ueber haemorrhagische Diathese bei Leukaemie und dadurch contraindicirte Splenotomie. *Inaug. Diss.* Greifswald, 1879. [Not haemophilia.]

403. Liegey: Observations relatives à la diathèse hémorrhagique ou hémophylie. *Journ. de médecine, de chirurgie et de pharmacologie.* Bruxelles, 1879, Tome lxix. pp. 135, 227. [Two cases of epistaxis in elderly females.]

404. Vigneau: no title. *Congrès international de médecine légale.* Paris, 1879, p. 149. [Death following blow on nose. Had bled easily before. No family history.]

405. Andrew: A case of haemophilia. *Medical Press and Circular.* London, 1880, n.s., Vol. xxx. p. 391. [See S. Davies, Bibl. No. 407.]

406. Buchanan: Haemorrhagic diathesis, protracted haemorrhage after the extraction of a tooth, application of actual cautery and subsequent use of ergotine subcutaneously. *The Glasgow Med. Journal.* Glasgow, 1880, Vol. xiv. p. 419. [Male aged 24. Grandfather sometimes bled freely from gums.]

407. Davies, S.: A case of haemophilia. *The Medical Press and Circular.* London, 1880, Vol. ii. p. 391. (See Andrew, Bibl. No. 405.) [Male aged 32 subject to bleeding from early childhood. At 22 haematuria, when examined an extravasation was found on left thigh, gums spongy, elbows and one knee stiff, effusion into eyelid. No family history.]

408. Dubay, Miklós: Haemophilia multiloc. spont. egy kóresete. *Gyógyászat.* Budapest, 1880, Bd. xx. S. 401. [A feeble male infant, the first born alive in three pregnancies. General haemorrhagic symptoms, death in five days.]

409. Finch, H.: Treatment of haemophilia. *The Lancet.* London, 1880, Vol. ii. p. 556. [Stout female aged 50—60. Suddenly attacked with epistaxis, which was instantly arrested by venesection. Brother died of haemorrhage from trivial wound and aunt bled copiously from slightest cause.]

410. Harkin, Alex.: Chlorate of potash in the haemorrhagic diathesis. *The Brit. Med. Journ.* London, 1880, Vol. ii. p. 700. [As in title, not haemophilia.]

411. Hertzka, Hermann: Ueber Hämophilie. *Wiener med. Presse.* Wien, 1880, Bd. xxi. cols. 523, 560, 635, 706, 774. [Long general account of haemophilia. On col. 774 same case as in Bibl. No. 412.]

412. Hertzka, H.: Ueber Haemophilie. *Mittheilungen des wiener medicinischen Doctoren-Collegiums.* Wien, 1880, Bd. vi. S. 189—194. [Pedigree No. 511.]

413. Jacubasch: Tuberkulose und haemorrhagische Diathese. *Jahrb. f. Kinderheilkunde und physische Erziehung.* Leipzig, 1880, Bd. xv. S. 167. [Tuberculous male aged 4, foundling. No family history. Death from purpura and epistaxis.]

414. Emrys-Jones: Haemorrhagic diathesis. *The British Med. Journ.* London, 1880, Vol. ii. p. 850. [Male aged 14. Profuse haemorrhage from ruptured eyeball.]

415. Lindemann, S. H.: A case of haemorrhagic diathesis; haemorrhage from the mouth, death. *The Lancet.* London, 1880, Vol. ii. p. 397. [Male aged 18 as in title. He also had haemoptysis and purpuric lumps on chest. Joints not affected. "Four of his relatives had died of the same thing."]

416. Luton: Rhumatisme hémophilique. *Union médicale et scientifique du nord-est.* Reims, 1880, Tome iv. p. 366. [Male, tailor, aged 21, had attacks of rheumatism, especially in spring and autumn, and associated with profuse epistaxis. Cured by potassium iodide. No family history.]

417. Mosler, Fr.: Ueber haemorrhagische Diathese und dadurch contra-indicirte operative Eingriffe bei Leukaemie und ihr verwandten Processen. *Ztschr. f. klin. Med.* Berlin, 1880, Bd. I. S. 265. [Haemorrhages in leukaemia in a doctor aged 40.]

418. Ribemont, Alban: Des hémorrhagies chez le nouveau-né. *Thèse présentée au concours pour l'agrégation en chirurgie.* Paris, 1880, 213 pp. [p. 111 no new cases free from obvious syphilis.]

419. Rizzo-Matera, S.: Contributo clinico all storia della emofilia e della peliosi reumatica. *L'osservatore medico.* Palermo, 1880, 3 s. T. x. p. 385. [Male aged 21 who had severe haemorrhages from trifling wounds. Swelling of R. knee was incised by a surgeon who however found only coagulated blood. An uncle and a cousin are said to have died of haemophilia.]

420. Stahel, H.: Die Hämophilie in Wald. *Inaug. Diss.* Zürich [1880]. [Pedigrees Nos. 407, 413, 414, 415.]

421. Teuffel, J.: Fall von acuter hämorrhagischer Diathese. *Medicinisches Correspondenz-Blatt des württembergischen ärztlichen Vereins.* Stuttgart, 1880, Bd. L. S. 58. [Male 31, bleeding after tooth extraction; later purpura, haematuria, death.]

422. Thorowgood: A case of haemorrhagic diathesis; haemorrhage from the mouth, death. *The Lancet.* London, 1880, Vol. II. p. 377. [Case under Thorowgood described by Lindemann. See Bibl. No. 415.]

423. Walker: Haemorrhagic Diathesis. *The Brit. Med. Journ.* London, 1880, Vol. II. p. 17. Also *Med. Times and Gaz.* London, 1880, Vol. II. p. 25. [Male aged 38, haemorrhage after tooth extraction.]

424. Whittaker, J. T.: Haemorrophilia. *Cincinnati Lancet and Clinic.* Cincinnati, 1880, n.s., Vol. v. p. 263. [Purpura and death from rupture of a blood blister on foot.]

425. Winter, W. H. T.: Some clinical observations on two cases of haemophilia. *The Dublin Journ. of Medical Science.* Dublin, 1880, 3. s., Vol. LXX. pp. 202—208. [Male 21. Crushed toe, amputation, death from secondary haemorrhage. Had had epistaxis. Two brothers similarly affected. Also Pedigree No. 385.]

426. Zielewicz: Traumatischer Haemarthros des Kniegelenks bei einem Bluter. Incision und Drainage des Gelenks. Tod durch Verblutung. *Centralblatt für Chirurgie.* Leipzig, 1880, Bd. VII. S. 243. [Male aged 11 as in title. Haemorrhage after tooth extraction. Autopsy. No family history.]

427. Ceci, Antonio: Ueber die haemorrhagische Infektion. *Arch. f. exp. Path. und Pharmacologie.* Leipzig, 1880—1, Bd. XIII. S. 461. [Case of haemorrhagic smallpox. Bacteriological investigation.]

428. Hopkins, St G. L.: Hematophilia; or the hemorrhagic diathesis. *Pacific Med. and Surg. Journal.* San Francisco, 1880—1, Vol. XXIII. p. 55. [General account. No cases.]

429. Packhard, J. H.: Death from haemorrhage occurring in a child fourteen days old, due to scratch of the lip. *Philad. Med. Times.* Philad., 1880—1, Vol. XI. p. 380. [As in title, eight lines.]

430. Bannister, H. M.: Haemophilia; haemorrhagic diathesis. *Cyclopaedia of Practical Medicine* (Ziemssen). New York, 1881. Suppl. p. 785. [Addition to Immermann's Article in the German Edition of "Ziemssen."]

431. Blailock, W. R.: A case of haemorrhage [haemophilia]. *Trans. of the Mississippi Medical Association.* Jackson, 1881, Vol. XIV. p. 142. [Male, white, aged 21. Subject from infancy to epistaxis increasing in severity on approach of puberty. Extensive haemorrhage from a septic hand. No hereditary data.]

432. Bokelmann, W.: Ueber die Natur und Bedeutung der hämophilen Gelenkaffektionen. *Inaug. Diss.* Göttingen, 1881. [Richard Rose, 13, under the care of König; no family history except that the father died of osteosarcoma of thigh and the mother eight days after birth of patient. Was well up to $1\frac{1}{2}$ years but then got haemarthros of L. knee followed later by similar affection of R. knee. The account then passes to describe him at the age of 13 while in hospital. Ecchymoses present. Knee joint disorganised. No haemorrhage from skin or mucosae. Elbows attacked with discolouration of skin while lying in bed. Aspiration of knee revealed pure blood. Treatment being unsatisfactory and a suspicion of tubercle being present the joint was opened and drained. It was found to be typical of haemophilia. No haemorrhage till the evening, then the bandages were found drenched, death in two days. P.M. no finding of interest. Macro- and microscopic description of joints.]

433. Chvostek: Ein Fall von allgemeiner haemorrhagischer Diathese im Beginne einer Recidive von Ileotyphus. Heilung. *Medicinisch-chirurgisches Centralblatt.* Wien, 1881, Bd. xvi. S. 373. [Epistaxis, typhoid fever in a male aged 21.]

434. Fano: Un cas d'hémophilie chez une jeune fille ayant subi l'amputation du segment antérieur de l'œil. *Le Courrier médicale et la réforme médicale.* Paris, 1881, Tome xxxi. p. 326. [Girl $11\frac{1}{2}$ years, iridochoroiditis from injury, ablation of anterior segment of eye, series of haemorrhages after the operation. Father had a boil on nates and bled for eight days after it was opened. He also bled three days from a tooth extraction. His brother bled for 19 days after tooth extraction.]

435. Foucherand, A.: Diathèse hemorrhagique? Anémie grave; mort. Autopsie. *Rev. de méd.* Paris, 1881, Tome i. p. 333. [Male aged 31, epistaxis and haemorrhage from gums started at 27. History of malaria and dysentery, haematuria, haematemesis and purpura; death.]

436. Hertzka, Hermann: Ein Fall von Haemophilie. *Arch. für Kinderheilkunde.* Stuttgart, 1881, Bd. ii. S. 339. [Male aged 13 months, after vaccination an eruption appeared over the whole body. At 8 months in hospital with diarrhoea; eventually died of epistaxis. Father aged 38 and paternal uncle both had epistaxis and vague reference is made to haemorrhage after injury.]

437. Legg, J. Wickham: Report on haemophilia with a note on the hereditary descent of colour blindness. *Saint Bartholomew's Hospital Reports.* London, 1881, Vol. xvii. p. 303. [Pedigree No. 468.]

438. Legg, J. Wickham: Tissues of a case of haemophilia. *The Lancet.* London, 1881, Vol. ii. p. 999. [Exhibition of the tissues of a boy who died after a small wound on the lip.]

439. Mehrer, H.: Blutungen aus verschiedenen Organen nach vorausgegangener allgemeiner Gefässaufregung. *Wiener med. Presse.* Wien, 1881, Bd. xxii. col. 176. [Epistaxis in a deaf mute aged 14, after being excited during a fire.]

440. Morales Peres, A.: La hemofilia en el concepto quirurgico. *Gaceta médica catalana.* Barcelona, 1881, Vol. i. pp. 457, 495, 534, 560. [A long and diffuse general account in which some six cases are incidentally described without any evidence being adduced that they were haemophilia *in sensu strictiore.* (1) Male, great haemorrhage after tooth extraction by a "key." (2) Female with severe bleeding after tooth extraction. (3) Severe and recurrent haemorrhage after stab in infraclavicular region. (4) Male, with epistaxis. (5) Female, aged 44, epistaxis. (6) Haemarthrosis of knee. We are indebted for the transcript of Morales' paper to Dr Cervera of Madrid and for the translation to Mr Huÿssen, London Hospital.]

441. Packhard: Case of hemophilia in an infant. *The American Journ. of Obstetrics.* New York, 1881, Vol. xiv. p. 686. [Female. Same case as No. 429; twelve lines.]

442. Thiersch, C.: Extirpation einer Balggeschwülst bei einem Bluter. *Verhandlungen der deutschen Gesellschaft für Chirurgie.* Berlin, X. Congress, 1881, S. 402. [As in title. Patient, a male, had been diagnosed as a bleeder by Oppolzer 30 years before, although there was no history of haemophilia in his family. After Thiersch's operation there was very extensive bruising and considerable haemorrhage. Ultimate recovery.]

443. Pepper, W.: Haemorrhagic diathesis. *The Philadelphia Medical Times.* 1881—2, Vol. xii. p. 109. [See T. D. Dunn's third family, Pedigree No. 435.]

444. Dedolph, F.: Transfusion in a case of haemophilia. *Transactions of the Minnesota State Medical Society.* St Paul, 1882, Vol. xiv. p. 89. [Severe haemorrhage occurring after incisions, into both tonsils, performed during "the dim morning light." On the twelfth day of the bleeding transfusion was performed directly from a sheep, eight ounces of the blood of which caused arrest. No evidence of haemophilia.]

445. Finlayson, James: Haemorrhagic diathesis in three generations. *The Glasgow Medical Journal.* Glasgow, 1882, Vol. xviii. p. 8. [Pedigree No. 522.]

446. Francis, C. R.: A case of haemophilia. *The Brit. Med. Journal.* London, 1882, Vol. ii. p. 615. [Quarter-master sergeant (age?). In 1859 nearly died of bleeding from a blow in the face. Haemorrhage after tooth extraction which did not stop till tooth had been replaced. Severe epistaxis and on one occasion haemoptysis. Insufficient data.]

447. Grenaudier, Louis: Contribution à l'étude de l'hémophilie. *Thèse de Paris.* 1882. [Four previously unpublished cases of which Obs. V. may be one of haemophilia, but owing to the paucity of details in the family history it need not be considered at length. Father, mother and 12 brothers and sisters of patient were healthy. Patient himself, aged 54, was admitted into the hôpital Laennec suffering from a large haematoma in the psoas region. Up to age of 12 affected

with epistaxis. Tooth extraction or cuts caused severe haemorrhage. He also suffered from haematuria, bruises and bleeding from gums, and rheumatic pains had been present from infancy. No definite haemarthroses. Detailed reference is given to his alcoholic habits and other matters.]

448. Jago, Fred. W. P.: Blood discs in a case of haemorrhagic diathesis. *The Brit. Med. Journ.* London, 1882, Vol. I. p. 576. [Thirteen lines with reference to male who had a tendency to bleed and whose blood discs were half as thick as normal.]

449. Kurz, Anton: Příspěvék ku haemofilii (contribution to haemophilia). *Časopis lékařův českých.* v. Praze, 1882, Vol. XXI. pp. 483, 501. [Case of a Jew aged 34. He bled severely after circumcision, and at 2 bled 14 days after two leech bites. Haematuria and bleeding from the bowel yearly. Admitted into clinic for haemoptysis. No haemophilia in the family. Kurz emphasises the relation of haemophilia to leucopenia.]

450. Legg, J. Wickham: A case of haemophilia with the results of the examination of the tissues. *Transactions of the Pathological Society of London.* London, 1882, Vol. XXXIII. p. 412. [Pedigee No. 471.]

451. [Richter]: Zur Casuistik von Purpura haemorrhagica und Haemophilia. *Schmidt's Jahrbücher der in- und ausländischen ges. Medecin.* Leipzig, 1882. Bd. CXCVI. S. 142. [Note by the Editor of a case already published by Kurz (see Bibl. No. 449) from information received from Richter.]

452. Rothschild, N.: Ueber das Alter der Haemophilie. *Inaug. Diss.* München, 1882. [Attempt to prove that haemophilia is described in the Talmud.]

453. Savery, W.: Hemorrhagic diathesis. *Med. News.* Philadelphia, 1882, Vol. XLI. p. 552. [Male aged 5, severe bleeding after scratching himself on a nail. A few days later he fell down and his face became enormously swollen and bruised.]

454. Steinitz, Julius: Ueber die haemorrhagische Diathese, die Hämophilie. *Breslauer aerztliche Zeitschrift.* Breslau, 1882, Bd. IV. S. 256. [Case of fatal haemorrhage following circumcision performed in the fourth week. Parents healthy. Mother had four children, viz. three girls living and healthy, one stillborn; then after an eight years' pause twins, viz. a boy (the patient) and girl (stillborn). Bleeding unknown in the family.]

455. Townsend, F. jun.: Hemophilia, complications, pregnancy. *Albany Medical Annals.* Albany, N.Y., 1882, Vol. III. p. 259. [Female aged 30 had had three living children and two miscarriages, one an abortion at the third month, the other a premature birth. During her sixth pregnancy she had uterine haemorrhage in the eighth month and labour ensued and was associated with copious bleeding. A sister died of haemorrhage from mouth, ears, nose, and eyes said to have been induced by fright.]

456. Vogel, M.: Tod durch Verblutung aus der Nabelschnur in Folge von Hämophilie. *Centralbl. f. Gynäkologie.* Leipzig, 1882, Bd. VI. S. 417. [Female infant, death from umbilical haemorrhage. No history in family.]

457. Melchior y Sendin, C. Un caso de nefrorragia por diátesis hemorragica. *Conferencias cientificas del Cuerpo de Sanidad de la Armada.* San Fernando, 1882—3, Vol. I. p. 136. [Sailor aged 21, suffering from persistent haematuria with great losses of blood at times. Author considers the condition due to haemorrhagic diathesis but gives no family history.]

458. Noir: Diathèse variqueuse, hematemesis, mort. *Société des sciences médicales de Gannat. Compte-rendu des travaux de l'année* 1882—83. Paris, Tome XXXVII. p. 66. [Male with haematemesis lasting off and on for 11 years, death.]

459. Dunn, Thomas D.: Haemophilia. *The American Journal of the Medical Sciences.* Philadelphia, 1883, Vol. LXXXV. p. 68. [Pedigrees Nos. 433, 434, 435, 436, 437.]

460. Eichberg, J.: A case of multiple hemorrhagic infarcts with some remarks on the pathology of the disease. *Cincinnati Lancet and Clinic.* 1883, n.s., Vol. XI. p. 525. [Female aged 23, nothing to do with haemophilia.]

461. Frazer, W.: On "Bleeders" and sudden death resulting from cerebral haemorrhage in such cases. *Trans. of the Acad. of Medicine in Ireland.* Dublin, 1883, Vol. I. p. 182. [Four cases: (1) woman died after the excision of a wart, (2) delicate girl aged seven bled badly after tooth extraction, died later of galloping consumption, (3) and (4) elderly people, death from cerebral haemorrhage.]

462. Lambert, S. H.: Haemophilia notes from practice. *Journ. of Amer. Med. Assoc.* Chicago, 1883, Vol. I. p. 349. [Female aged 63 with rounded tumour in breast, suffered from epistaxis and haemorrhage from gums. Two brothers had tumours, site not given.]

463. Port, H.: A case of haemophilia with joint disease. *Abstract of the Transactions of the Hunterian Society. Session* 1882—1883. London, 1883, p. 27. [Pedigree No. 505.]

464. Recklinghausen, F. v.: Handbuch der allgemeinen Pathologie des Kreislaufs und der Ernährung. Deutsche Chirurgie hrsg. von Billroth und Lücke. Lieferung 2 und 3. Stuttgart, 1883, S. 91. [Short general account of theories of haemophilia.]

465. af Schultén: En familj af blödare. *Finska läkaresällskapets Handlingar.* Helsingfors, 1883, Bd. xxv. S. 321. [Pedigree No. 432.]

466. Schumann, Eugen: Ueber haemorrhagische Diathese und deren Beziehung zur Gravidität. *Inaug. Diss.* Berlin [1883]. [Female aged 32, pregnant with fourth child. Purpura, haematuria, discolouration of skin of face and eyelids. Child born in clinic, death of mother half an hour after. Autopsy (Jürgens) showed intraparietal haemorrhage in R. auricle of heart, cystitis, pyelitis haemorrhagica, universal anaemia and multiple petechiae.]

467. Tiedemann, E. F.: A case of haemophilia, various haemorrhages continuing for fifteen days. *Med. News.* Philadelphia, 1883, Vol. xliii. p. 269. [Female aged 18, epistaxis, purpura, haemorrhage from gums and other places, septic condition of nasal cavities, rigors. No history of haemorrhage in family except epistaxis.]

468. Vulpian: De quelques particularités intéressantes dans un cas d'hémophilie. *Le Practicien.* Paris, 1883, Année vi. p. 112. [Male aged 30, suffered from epistaxis for a number of years. When seen by Vulpian he had great anaemia, fever, otitis and enlargement of spleen, no family history of bleeding.]

469. Eymery: Quelques cas de mort subite chez les hêmophiles. *Journ. de médecine de Bordeaux.* 1883—4, T. xiii. pp. 473, 487. [Three cases: (1) infant with ecchymoses, death from haemoptysis at 11 months, (2) two female children with ecchymoses and epistaxis, death from haemorrhage from the mouth. These two belonged to a part of a large family who were all of an haemorrhagic tendency and physically resembled the father, (3) female aged $3\frac{1}{2}$, died of whooping cough with haemorrhagic symptoms.]

470. Baege, Ernst: Ein Beitrag zur Lehre von der Haemophilie und Leuchaemie. *Inaug. Diss.* Berlin, 1884. [Male aged 27, brewer's drayman in Charité hospital with large swelling of L. thigh; incision evacuated large quantity of blood and cavity filled up with blood clot; two days later ligature of femoral below Poupart's ligament, later great bleeding. Later he was again in hospital with leukaemia (examination of blood by Ehrlich). He had suffered from epistaxis in youth. His father, said to have bled easily, died of phthisis at 40. Grandfather died in 1813 from an injury. An elder brother had a tendency to bleed but patient's sisters and his two children were not affected.]

471. Cornillon, J.: L'hémophilie, est-elle une contre-indication au traitement par les eaux de Vichy. *Gaz. d. hôp.* Paris, 1884, 7me sér. Tome i. p. 246. [No cases, diffuse account, no value.]

472. Flynn, E. F.: A case of haemophilia. *The Brit. Med. Journ.* London, 1884, Vol. ii. p. 714. [Male aged 20, in Sunderland Infirmary, with synovitis of hip, developed acute tonsilitis and bled for 15 days from incision made into it. At the age of 10 he had bled for a week from a small wound on forehead, at 14 three leech bites induced great haemorrhage. Liable to great spontaneous and traumatic haemorrhages. Two uncles died from haemorrhage; the one, the mother's brother, succumbing to bleeding from a small wound on the tongue. The haemorrhagic tendency in the other case was not definite.]

473. Forcheimer, F.: Hemophilia, case. *Cincinnati Lancet and Clinic.* 1884, n.s. Vol. xii. p. 231. [Male aged 11, bled persistently from slight injuries. At 2 bled for one week from bite of tongue, and at 3 from injury to knee. Since then had had four haemorrhages. The absence of a similar condition in other members of family clearly established.]

474. Fürth, L.: Die Nabelblutungen (haemorrhagia ex umbilico). *Archiv f. Kinderheilkunde.* Stuttgart, 1884, Bd. v. S. 305. [General account of the subject.]

475. Hernandez, Carlos: Hémorrhagies successives, hémophylie. *Thèse de Paris.* 1884. [Pedigree No. 537.]

476. Hirsch, Aug.: Article "Albucasis" in Hirsch's Biographisches Lexikon der hervorragenden Aerzte aller Zeiten und Völker. Wien und Leipzig, 1884, Bd. i. S. 170.

477. Hughes, C. H.: Haemophilia. *The Weekly Medical Review.* Chicago and St Louis, 1884, Vol. x. p. 517. [Brief account of a male aged 11 who from infancy bled profusely from slight causes, mother had epistaxis and haemorrhage from ears and lungs.]

478. Keneagy, S. Post mortem appearances and brief history of a case of haemorrhagic diathesis (haemophilia). *The Practitioner.* Lancaster, Pa., 1884, Vol. II. p. 25. [Fatal case of epistaxis in a male aged 46. The haemorrhagic diathesis "can be traced back to the great-grandmother of deceased and more or less through all the descendants." Of the patient's three children, two, a boy and a girl, suffered from epistaxis.]

479. Liegey: Influence de la diathèse hémophilique ou hémorrhagique sur la marche d'un cancer du sein. *Journal de médecine de chirurgie et de pharmacologie.* Bruxelles, 1884, Tome LXXVIII. p. 591. [Cachectic and haemorrhagic state in a female with cancer.]

480. Macnamara: Remarkable case of haemophilia; excision of the breast; recovery; remarks. *The Lancet.* London, 1884, Vol. I. p. 294. [Female aged 51 as in title, healing by first intention. She and other members of the family subject to epistaxis.]

481. Raven, T. F.: Haemorrhage from the umbilicus in haemophilia. *The Brit. Med. Journal.* London, 1884, Vol. II. p. 907. [As in title, no evidence of family history of haemophilia.]

482. de Rute, F.: Uno caso de hemofilia. *La medicina contemporanea.* Madrid, 1884, Vol. I. p. 682. [Girl aged 19 with sudden haemoptysis. Author learned that her parents had lost eight children ("hijos") "from cerebral haemorrhage." Fourteen months later a sister of the girl, recovering from enteric fever, died suddenly from cerebral haemorrhage. The patient herself was attacked four years later with epistaxis but recovered. No other data.]

483. Smith, J. Greig: Two cases of haemophilia. *Bristol Med.-Chir. Journal.* Bristol, 1884, Vol. II. p. 264. [Pedigrees Nos. 574, 575.]

484. Soederbaum, P.: Om faror vid små operationer. *Eira—Tidskrift för helso- och sjukvård.* Goteborg, 1884, Vol. VIII. No. 7, p. 215. [Female aged 26, haemorrhage after tooth extraction.]

485. Anon.: The haemorrhagic diathesis. *The Brit. Med. Journal.* London, 1884, Vol. I. pp. 686, 690. [Account of illness and death of H.R.H. the Duke of Albany. In Jan. 1868 he bled severely for a week, later had swellings of one knee joint. On March 27, 1884, he slipped in the cercle nautique at Cannes and during the night was found breathing stertorously and in an unconscious state in which he died at 4 a.m. next morning, aged 31 years.]

486. [Verneuil]: Hémorrhagies attribuées à l'hémophilie. *Journal de méd. et de chirurgie pratiques.* Paris, 1884, Tome LV. p. 299. [Refers to the slipshod way in which the diagnosis of haemophilia is frequently made in the absence of detailed clinical examination. As examples he cites a case of splenic and hepatic tumour and osteosarcoma of pelvis both associated with haemorrhages.]

487. Acland, Theodore D.: Changes in the thymus gland in a case of haemophilia and in one of purpura. *Trans. of the Pathological Soc. of London.* London, 1884—5, Vol. XXXVI. p. 491. [Pedigree No. 548.]

488. Bemiss, J. H.: Haemophilia. *New Orleans Medical and Surgical Journal.* 1884—5, n.s., Vol. XII. p. 165. [Three cases described, none of which appear to be haemophilia. (1) Male aged 18. First showed purpura at 13, and subsequently purpura, epistaxis and finally malaria. Various haemorrhagic manifestations, enlarged spleen, and acute rheumatism in various members of the family. (2) Death following puncture of R. enlarged tonsil, in a male aged 20. (3) Haemoptysis in a male aged 18.]

489. Legg, J. Wickham: A second case of haemophilia with examination of the tissues and joints. *Transactions of the Pathological Society of London.* London, 1884—5, Vol. XXXVI. p. 488. [Case of Charles B. aged 13. At 3 he bled severely from small wound of tongue; at 6 bled eight weeks after tooth extraction. Bruised easily and had swollen joints. Brought to St Bartholomew's hospital in a collapsed condition bleeding from nose and with a large ecchymosis in his groin. Death on following day. Autopsy. Thymus gland persistent. Both knees and ankles examined. Cartilages worn and thin. Joints preserved in St Bart's museum. One brother died of epistaxis at $3\frac{1}{2}$ years of age.]

490. Taylor, W. H.: Haemophilia. *Maryland Med. Journ.* Baltimore, 1884—5, Vol. XI. p. 441. [Five cases: (1) male infant, haemorrhage from navel and other sites, epistaxis in mother, (2) negro aged 9 had three instances of severe bleeding after wounds, (3) old female who had epistaxis, (4) female mulatto aged 16, epistaxis, ate clay, (5) female aged 9 with purpura.]

491. Forcheimer, F.: Haemophilia without heredity. *The Archives of Pediatrics.* Philadelphia, 1885, Vol. II. p. 728. [Male bleeder aged 11. At 7 months cut upper lip and bled several days; at 2 cut forehead and nearly bled to death. In the last nine years had nearly died of bleeding 10 times. Swellings on thighs, leg, forearm, knees swollen. No history in the family. Mother denied it in her family. Presumably the same case as Bibl. No. 473 but differing somewhat in detail.]

492. Gwynne: Haemophilia. *The British Medical Journal.* London, 1885, Vol. i. p. 234. [Boy of 10 who had been repeatedly treated by G. for haemorrhages from fingers, tongue, nose, scalp consequent on trivial injuries. When one year old he knocked his knee in bed and swelling occurred which ended in slight contracture; he had also suffered from ecchymoses; no family history.]

493. Hœssli, Anton: Geschichte und Stammbaum der Bluter von Tenna (Canton Graubünden). *Inaug. Diss.* Basel, 1885. [Pedigree No. 373.]

494. Holloway, O. E.: Haemophilia. *The Cincinnati Lancet and Clinic.* 1885, n.s. Vol. xv. p. 68. [Pedigree No. 576.]

495. Jones, Handfield: *The Lancet.* London, 1885, Vol. i. p. 801. [In a discussion on J. E. Lane's paper (see Bibl. No. 497) author refers to a family in which the haemorrhagic diathesis had been traced through three generations; no details.]

496. Klippel, Maurice: Note sur un cas d'hémophilie. *Annales médico-chirurgicales françaises et étrangères.* Paris, 1885, Tome i. p. 16. [Female aged 45 had profuse menstruation and haemorrhage after three of her six confinements. Admitted to hôp. Pascal suffering from epistaxis, ecchymoses and metrorrhagia; no family history of haemophilia.]

497. Lane, J. E.: On haemophilia. *The Lancet.* London, 1885, Vol. i. p. 801. [General report of a paper read before the Harveian Society on April 16, 1885. No reference to cases.]

498. Löwit, M.: Die Blutplättchen und die Blutgerinnung. *Fortschritte der Medicin.* Berlin, 1885, Bd. iii. S. 173. [Influence of blood platelets on the coagulation of the blood.]

499. Osler, W.: Haemophilia. Pepper's System of Medicine. London, 1885, Vol. iii. p. 933. [General account, see Hay's case, Pedigree No 408.]

500. Wagner, Paul: Zur Casuistik der Hämophilie. *Deutsche Ztschr. für Chirurgie.* Leipzig, 1885, Bd. xxi. S. 351. [Three cases. (1) Male aged 20, haemorrhage from wounds and tooth extraction. No family history. (2) Male aged 23 bled easily. Was stabbed in hand. Swelling, amputation, death from haemorrhage. (3) Male aged 21, epistaxis began when he went to school. Sudden pain in and swelling of joints. While in hospital ankylosis and contracture. Bleeding from tooth extraction. One brother died of epistaxis, another of scalp wound. No further history.]

501. Buster, O. C.: Case of haemophilia. *The Texas Courier-record of Medicine.* Dallas, 1885—6, Vol. iii. p. 239. [Baby one day old, with alleged heart lesion, death from bleeding from lips, gums, palate, scalp, ear.]

502. Alegre, J.: Caso de hemophilia. *Medicina contemporanea.* Lisboa, 1886, Vol. iv. p. 413. [Case of epistaxis.]

503. Hansen, Søren: Om haemofiliens Arvelighed. *Hospitals-tidende.* Kjøbenhavn, 1886, 3dje Raekke, Bd. iv. S. 241. [Pedigrees Nos. 455, 456, 457, 458.]

504. Kurz, Anton: Případ haemofilie (a case of haemophilia). *Časopis lékařův českých.* v. Praze, 1886, Vol. xxv. pp. 17, 33. [Pedigree No. 379.]

505. Low: Haemophilia. *The British Med. Journal.* London. 1886, Vol. ii. p. 1037. [Male aged 27 with epistaxis, which was prevalent in the family. Abstract of seven lines.]

506. M'Caw, J.: Haemophilia; its causes, symptoms and treatment with an illustrative case. *The Dublin Journ. of Med. Science.* Dublin, 1886, 3 s., Vol. lxxxi. p. 507. [Pedigree No. 547.]

507. Oliver, T.: Clinical notes on haemophilia. *The Lancet.* London, 1886, Vol. ii. p. 526. [Pedigrees Nos. 533, 536.]

508. Payne, R. L., jr.: Haemophilia. *The North Carolina Med. Journal.* Wilmington, 1886, Vol. xvii. pp. 80—87. [Pedigree No. 557.]

509. Pepper, A. J.: A case of haemorrhagic diathesis in which the brachial artery had to be tied for wound in the palm of the hand and the pleural cavity plugged for excessive bleeding from the granulation membrane of an empyema. *The Provincial Med. Journ.* Leicester, 1886, Vol. v. p. 146. [As in title, middle aged male, secondary haemorrhage from septic wound of thumb. No family history of haemorrhage.]

510. Treves, F.: A case of haemophilia, pedigree through five generations. *The Lancet.* London, 1886, Vol. ii. p. 533. [Pedigree No. 493.]

511. Vulpian: Hémophile; pas d'antécédents d'hérédité ou de famille; antécédents scrofuleux dans l'enfance et la jeunesse; accidents saturnins à plusieurs réprises; syphilis; épistaxis abondantes; saignements des gencives; ecchymoses des oreilles et, plus tard, des mains; mort par épistaxis treize ans après la première hémorrhagie nasale. *Revue de médecine.* Paris, 1886, Tome vi. p. 153 [Title sufficient.]

512. Graham, James: A case of haemophilia. *The Australasian Medical Gazette.* Sydney, 1886—7, Vol. vi. p. 245. [Pedigree No. 602.]

513. Caldwell, J. K. P.: Haemophilia. *Mississippi Valley Medical Monthly.* Memphis, 1887, Vol. vii. p. 299. [Male child, American, 3 weeks old. Bleeding from slight scratches, haematomata (?) over scapula. Inadequately described and studied, possibly complicated with malaria. No data as to hereditary taint, no mention of bleeding in other members of the family having been inquired into.]

514. Chambers, J. W.: [Four cases of haemorrhagic diathesis.] *Maryland Medical Journal.* Baltimore, 1887, Vol. xvii. p. 30. [In two of the cases the condition was apparently induced by the treatment; not of permanent value.]

515. Fuchs, A.: Zur Casuistik der acuten haemorrhagischen Diathese. *Prager medicinische Wochenschrift.* Prag, 1887, Bd. xii. S. 246. [Two cases. (1) Female aged 62; on twelfth day of a pneumonic infection, collapse and haemorrhages in face, trunk, extremities and mucous membrane of mouth: albuminuria, recovery. (2) Female aged 18; cephalalgia, fever, haematuria, petechiae: exitus lethalis.]

516. Hughes, C. H.: The relation of the nervous system to haemophilia, malarial haematuria, etc. *The Alienist and Neurologist.* St Louis, 1887, Vol. viii. pp. 378—387. [As in title.]

517. Kurz, Anton: Ein Fall von Hämophilie. *Medicinisch-chirurgisches Centralblatt.* Wien, 1887, Bd. xxii. S. 556, 568. [Abstract in German of Bibl. No. 504.]

518. Page, H. W.: A case of haemophilia. *The Lancet.* London, 1887, Vol. i. p. 1028. [Pedigree No. 518.]

519. [Pollard, Bilton]: Case of haemophilia. *Report of the Surgical Registrar North London or University College Hospital for* 1886. London, 1887, p. 13. [A note referring to boy aged $3\frac{1}{2}$ who bled six days from a cut lip. A brother had been three times in hospital for haemorrhage: maternal uncle died of bleeding at the age of 13.]

520. Reyes, A.: Una observación de hemorrafilia. *Gaceta médica de Mexico.* México, 1887, Tomo xxii. p. 45. [A native Mexican aged 26 of marked alcoholic habits and living in filthy surroundings, developed malaria and epistaxis followed by bleeding from the mouth, rectum and urethra. He bled copiously after incisions into an inguinal bubo and into a swelling of the face. Subcutaneous extravasations of blood were frequent. He then suffered from hypertrophy of the bladder. No family history of haemophilia. Mr Ronald Macleay, H.M.'s Chargé d'Affaires in Mexico, who kindly procured a copy of the *Gaceta méd. de Mexico*, informs us, in addition, that he has authority for saying that "by an unaccountable blunder the name of Dr Reyes was appended to the article which was really written by Dr Agustin Villalobos."]

521. Rinonapoli, E.: Poche considerazioni sulle esplicazioni della diatesi emorragica, caso di porpora perniciosa. *Archivio di pathologia infantile.* Napoli, 1887, Tomo v. p. 200. [Clinical history of a case of purpura haemorrhagica in a child previously in good health. Death on the third day.]

522. Savoye: Cas d'hémophilie. *Bulletin de la société de médecine pratique de Paris.* Année 1887, p. 221. [Girl aged $8\frac{1}{2}$ passed blood per rectum and had petechiae on arms and legs; later epistaxis and haemorrhage from ears, face, neck, gums. No family history of haemorrhage.]

523. Shattuck, F. C.: Haemophilia. *The Boston Med. and Surg. Journal.* Boston, 1887, Vol. cxvi. p. 497. [Male aged 52. Haemorrhage after incision into tonsillar abscess. 150 attacks of haematuria. He was easily bruised when a child. Epistaxis once. Reference to other affected members in the family.]

524. Skelton, H.: Three cases of haemophilia in the same family. *The Lancet.* London, 1887, Vol. i. p. 874. [*Vide* Greig-Smith, Bibl. No. 483, and Pedigree No. 574.]

525. Speidel: Haemorrhophilie, Beitrag zur Bluterkrankheit. *Ztschr. f. Wundärzte und Geburtshelfer.* Hegnach, 1887, Bd. xxxviii. S. 40. [Pedigree No. 577.]

526. Villard: Des maladies hémophiliques. *Marseille médical.* Marseille, 1887, T. xxiv. pp. 449, 530, 581. [Lecture on haemophilia, scurvy and purpura. One case, female, aged 21, of purpura.]

527. Wendt, Edmund C.: Congenital haemophilia with the history of a remarkable case. *Medical Record.* New York, 1887, Vol. xxxi. pp. 208—210. [Male infant, umbilical haemorrhage, purpura neonatorum, death.]

528. Wiener, M.: Ueber haemorrhagische Erkrankungen bei Schwangeren und Wöchnerinnen. *Archiv für Gynaekologie.* Berlin, 1887, Bd. xxxi. S. 281. [Female aged 28 and her sister; death from puerperal septicaemia.]

529. Bailey, S.: Hemophylia; a family of bleeders. *Progress.* Louisville, Ky., 1887—8, Vol. II. p. 495. [One case of purpura neonatorum; few remarks upon some other members of the family suffering from conditions known not to be haemophilic.]

530. Fisher, E. A.: Nearly fatal hemorrhage following the removal of a tooth, owing to the hemorrhagic diathesis. *The Cincinnati Med. Journ.* 1887—8, Vol. III. p. 289. [Male aged 20; maternal uncle bled once in a similar way; brother not affected.]

531. Hope, James W.: A case of haemophilia. *The Australasian Med. Gazette.* Sydney, 1887—8, Vol. VII. p. 7. [Pedigree No. 554.]

532. Carson, S. C.: Haemophilia. *Gaillard's Medical Journal.* New York, 1888, Vol. XLVII. [XLVI.] p. 314. [Umbilical haemorrhage in two children of same mother.]

533. Eagle, H. F. C.: Haemophilia. *Brit. Med. Journ.* London, 1888, Vol. I. p. 531. [Male infant; jaundice and oozing of blood from mouth and navel on the fourteenth day; swelling over left scapula; death.]

534. Hubbard, Thomas: Report of a case of haemophilia with family history for three generations. *The Toledo Medical and Surgical Reporter.* Toledo, O., 1888, Vol. I. p. 11. [Pedigree No. 545.]

535. M^{c}Arthur, D. R.: Haemophilia. *Brit. Med. Journ.* London, 1888, Vol. I. p. 793. [Male infant; purpura neonatorum, umbilical haemorrhage on fifth day; death.]

536. Rochard, E.: Hémophylie. *Dict. encycl. d. sc. méd.* Paris, 1888, 4. s., Tome XIII. pp. 291—304. [General account.]

537. Cohnheim, Julius: Lectures on general pathology. London, 1889, Vol. I. p. 397. [An account of the influence of haemorrhage on the coagulability of the blood. Reference to haemophilia.]

538. Coltman, R., jr.: Hemorrhagic diathesis. *The China Medical Missionary Journal.* Shanghai, 1889, Vol. III. p. 12. [Post-operative haemorrhage after removing hard growth from penis of a Chinaman.]

539. Duany-Soler: Hémophilie. *Le Poitou médical.* Poitiers, 1889, Tome III. p. 129. [Male aged 3 who suffered from epistaxis and haemorrhage from the gums. Haemophilia alleged in two male cousins, but without any data.]

540. Eve, F. S., and Bidwell, L.: Haemophilia. *The Lancet.* London, 1889, Vol. II. p. 1002. [Pedigree No. 399.]

541. Fischer, Max: Zur Kenntniss der Hämophilie. *Inaug. Diss.* München, 1889. [Pedigree No. 426.]

542. Hayem, Georges: Du sang et de ses altérations anatomiques. Paris, 1889, p. 999. [Refers to the great rarity of haemophilia in France and to the fact that he himself had never seen an authentic case of the hereditary disease.]

543. Herrick, J. B.: A case of hemophilia neonatorum. *The North American Practitioner.* Chicago, 1889, Vol. I. p. 84. [Male infant died on twelfth day of umbilical haemorrhage; also haemorrhage from a pricked finger, conjunctival haemorrhage and bleeding from the mouth. Father had epistaxis.]

544. Jalaguier: quoted in Hayem's Du sang et de ses altérations anatomiques. Paris, 1889, p. 1001. [Male aged 14; epistaxis, haematuria, repeated haemarthroses and subcutaneous haemorrhages and an haematoma. Family history not given.]

545. Kirmisson: quoted in Hayem's Du sang et de ses altérations anatomiques. Paris, 1889, p. 1002. [Male aged 19 liable to epistaxis, furious bleeding from furuncle on nose, great haemorrhage after tooth extraction, haematoma in calf. Division of tendo achillis by Kirmisson, severe haemorrhage and phlegmon. At 19 had an enormous haemorrhage and nearly died. No history of haemophilia in the family.]

546. Koch, Wilhelm: Die Bluterkrankheit in ihren Varianten. Stuttgart, 1889, 227 pp. (Lieferung 12 of Deutsche Chirurgie hrsg. von Billroth und Luecke.) [Exhaustive work in which the author arrives at the conclusion that all the haemorrhagic diseases, including haemophilia, are but variants of a common toxic-infectious state.]

547. Martin, L.: Zur Casuistik der transitorischen haemorrhagischen Diathese. *Annalen der städtischen allgemeinen Krankenhäuser zu München* 1880—1884. München, 1889, Bd. IV. S. 466. [Good account as in title. M. opines that many cases published as haemophilia are really morbus maculosus Werlhofii.]

548. Ripke, Christlieb: Ueber Haemophilie. *Inaug. Diss.* Freiburg in Baden, 1889, 75 pp. [Pedigree No. 377.]

549. Young, James: On a case of haemophilia. *The Lancet.* London, 1889, Vol. II. p. 951. [Pedigree No. 520.]

550. Arthus, M., et Pagès, C.: Nouvelle théorie chimique de la coagulation du sang. *Archives de physiologie normale et pathologie.* Paris, 1890, 5 s. Tome II. p. 739.

551. Benedict, S. C.: Haemophilia. *The Southern Dental Journal.* Atlanta, Ga., 1890, Vol. IX. p. 133. [Pedigree No. 402.]

552. Bowlby, Anthony A.: Some cases of joint diseases in bleeders. *St Bartholomew's Hospital Reports.* London, 1890, Vol. XXVI. p. 77. [Pedigrees Nos. 403, 404.]

553. Cohen, G.: Ein Fall von Hämophilie. *München. med. Wochenschrift.* München, 1890, Bd. XXXVII. S. 209. [Female aged 38 of neuropathic stock, and displaying a remarkable combination of haemorrhagic and other phenomena.]

554. Cohen, G.: Ein Fall von Hämophilie. *Ztschr. f. klin. Med.* Berlin, 1890, Bd. XVII. Suppl.-Hft, S. 182—201. [Same case as Bibl. No. 553.]

555. Hornbogen, A. H.: Haemophilia. *Med. and Surg. Rep. Cook Co. Hosp.* Chicago, 1890, p. 134. [Male aged 21. Persistent haemorrhage following operation for piles and fistula, epistaxis yearly. "The patient's only brother is affected in a similar manner."]

556. Koch, W. V. M.: Haemophilia occurring in malaria. *Brit. Med. Journ.* London, 1890, Vol. I. p. 1301. [Female aged 6. Violent haemorrhage from mouth, one day after an attack of fever; death. Autopsy showed congested spleen.]

557. Loviot: Placenta praevia chez une primipare âgée, hémorrhagies répétées, rupture artificielle des membranes, version podalique bipolaire, mort par anémie vraie. *Bulletins et mémoires de la société obstétricale et gynécologique de Paris.* 1890, p. 78. [As in title, not haemophilia.]

558. Patterson, J. A.: Rupture of the choroid in a haematophilic. *The Therapeutic Gazette.* Detroit, Mich., and Philadelphia, Pa., 1890, 3 s., Vol. VI. p. 310. [As in title, female aged 22, no evidence of haemophilia.]

559. Sarra, R.: Un caso di diatesi emorragica congenita. *Archivio italiano di pediatria.* Napoli, 1890, Tomo VIII. p. 15. [Protocol of an autopsy on a baby 50 days old. It had been ill for three days with lividity and convulsions: Three brothers had died at the same age of the same symptoms. The autopsy showed enlargement of spleen and subcutaneous and submeningeal haemorrhages.]

560. Stecki: Haemophilia. *Medycyna.* Warsawa, 1890, Vol. XVIII. p. 182. [Male aged 62 with haemorrhage from the gums, which began about 12 years ago and recurred about once or twice a month.]

561. Stengel, W.: Case of haemophilia. *The Pittsburgh Med. Review.* Pittsburgh, 1890, Vol. IV. p. 221. [Death following longitudinal division of prepuce in an infant. No haemorrhagic history in family.]

562. König, Franz: Die Gelenkerkrankungen bei Blutern mit besonderer Berücksichtigung der Diagnose. *Sammlung klinischer Vorträge (R. Volkmann).* Leipzig, 1890—1894, Chirurgie No. 1—25, p. 232. [In the course of his classical description of the pathology of haemophilic joint lesions König mentions two cases. (1) Male aged 17, bled readily from slight wounds; a few months before he sustained a condition of knee illustrating Stage I.; puncture evacuated 100 c.c. of fluid blood; one sib bled badly after tooth extraction. (2) Death three days after incision into haemophilic joint.]

563. Beebe, D. C.: Haemophilia and surgical operations. *The Medical Standard.* Chicago, 1891, Vol. X. p. 125. [Case of female baby, gums lanced, ecchymoses, haematomata, death. The child was a descendant of a "line of bleeders," various female ascendants suffering from epistaxis and uterine haemorrhage.]

564. Butler, W. M.: A case of haemophilia. *The North American Journal of Homoeopathy.* New York, 1891, 3 s., Vol. VI. p. 225. [Haemoptysis with negative result of physical examination of chest.]

565. Delezenne, C.: Note sur un cas d'hémophilie hystérique. *Bulletin médical du nord.* Lille, 1891, Tome XXX. p. 541. [Female with multiple haemorrhages during first menstruation, menorrhagia and dysmenorrhœa. "Crises hystériques" from 26—50 years. Several other relatives had nervous attacks and one brother had epistaxis.]

566. Demme, R.: Fälle von Hämophilie. *Achtundzwanzigster medicinischer Bericht über die Thätigkeit des Jenner'schen Kinderspitales in Bern im Laufe des Jahres* 1890. Bern, 1891, S. 20. [Pedigree No. 411.]

567. Duany-Soler: Observation d'hémophilie. *Bull. de la soc. de méd. et chirurgie de La Rochelle.* 1891, No. 7, p. 57. [Reproduction of Bibl. No. 539, but no reference is made to it.]

568. Hamilton, Allan McLane: A contribution to the pathology of haemophilia esp. in regard to its neurotic aspects, with the presentation of several cases in one family. *Medical Record.* New York, 1891, Vol. xl. p. 617. [Pedigree No. 397.]

569. Hayem, G.: Sur un cas de diathèse hémorrhagique. *Bulletins et mémoires de la société médicale des hôpitaux de Paris.* 1891, 3 s. Tome viii. p. 389. [Female aged 22, epistaxis, menorrhagia after coitus, no examination of uterus.]

570. Jardine, R.: Haemophilia in a newly born child. *Brit. Med. Journ.* London, 1891, Vol. i. p. 636. [Weakly female infant, oozing from navel on ninth day, jaundice; on eighteenth day purpura neonatorum, death.]

571. Keimer, Wenzel: Ueber zwei Fälle von intermittirender Bluterkrankheit. *Inaug. Diss.* Kiel, 1891, 30 pp. [Two cases. (1) Boy one year old with large swelling on forehead and chemosis of eyelid; bandage applied but a colossal haemorrhage occurred underneath it. Diagnosis, haematoma or sarcoma. Removed by operation it proved to be a blood clot. Great subsequent haemorrhage, collapse, exitus. Autopsy showed marked osteophytic formation on skull. His father had epistaxis in youth. (2) Boy aged 10 with recurrent attacks of epistaxis. His grandmother was a "bruiser."]

572. von Limbeck, R.: Zur Casuistik der erblichen Hämophilie. *Prager med. Wchnschr.* Prag, 1891, Bd. xvi. S. 459. [Pedigree No. 452.]

573. Lowry, Isabel: A study of two cases of hemophilia in the same family. *Occidental Med. Times.* Sacramento, 1891, Vol. v. p. 587. [Female, Pole, with menstrual troubles, purpuric eruptions and other complex, partly neuropathic symptoms. Her daughter also had purpura.]

574. Prewitt, T. F.: A case of haemophilia. *The St Louis Courier of Medicine.* Saint Louis, 1891, Vol. iv. p. 15. [Female aged 17, four weeks' oozing from gums the result of dental operations.]

575. Senator, H.: Ueber renale Haemophilie. *Berl. klin. Wchnschr.* Berlin, 1891, Bd. xxviii. S. 1. [Female aged 19, persistent haemoglobinuria. Nephrectomy. Kidney appeared normal. Most members of the family had epistaxis and menstrual troubles.]

576. Taub, Gyula: Haemophilia egy esete. *Gyógyászat.* Budapest, 1891, Vol. xxxi. p. 354. Also transl. [Abstr.], *Pest. med. chir. Presse.* Budapest, 1891, Bd. xxvii. S. 1214. [Pedigree No. 419.]

577. Utley, J. H.: A case of haemophilia. *The Southern Californian Practitioner.* Los Angeles, 1891, Vol. vi. p. 481. [Child 18 months. Epistaxis, previous haemorrhage from cut of upper lip, bruises and bleeding after slight injury. No family history of haemorrhage.]

578. Vanderveer, A.: Report on a case of haematophilia, or a family of bleeders. *Archives of Pediatrics.* Philadelphia, 1891, Vol. viii. p. 756. [Pedigree No. 438.]

579. Wright, A. E.: Upon a new styptic and upon the possibility of increasing the coagulability of the blood in the vessels in cases of haemophilia and aneurism or internal haemorrhage. *The Brit. Med. Journ.* London, 1891, Vol. ii. p. 1306. [As in title.]

580. Neumann, H.: Weiterer Beitrag zur Kenntniss der haemorrhagischen Diathese Neugeborener. *Archiv für Kinderheilkunde.* Stuttg., 1891—2, Bd. xiii. S. 211. [Male child of syphilitic mother, haemorrhage, jaundice, death.]

581. Beebe, D. C.: Hemophilia with mention of a case. *Transactions of the Wisconsin State Medical Society.* Madison, 1892, Vol. xxvi. p. 244. [Same case as No. 563.]

582. Bertrand, Carl: Ueber die Zulässigkeit grösserer Operationen bei Blutern. *Inaug. Diss.* (Heidelberg). Wiesbaden, 1892. [A full account of six male cases, in five of which we consider there is no evidence of haemophilia notwithstanding the statement that Cases II. and IV. were "well marked" bleeders; Case VI. probably a bleeder. In none of the cases was there a family history of haemophilia. Case VI. is the history of a male into whose knee joint an incision was made under the idea that it was tuberculous. It was found to be haemarthrosis and great haemorrhage followed the operation. The patient subsequently admitted that he had frequently had epistaxis, ecchymosis and severe bleedings from small wounds.]

583. Demme, R.: Ein Fall von Haemophilie bei einem $2\frac{1}{2}$-jährigen Knaben. *Allg. med. Centralzeitung.* Berlin, 1892, Bd. lxi. S. 189. Same title, *Medicinisch-chirurgisches Centralblatt.* Wien, 1892, Bd. xxvii. S. 182. [Same case as Bibl. No. 566.]

584. Gepner, B. R.: Przypadek zaniku nerwu wzrokowego po kowotku do oczodolu u hemofilika. *Medycyna.* Warszawa, 1892, Vol. xx. p. 573. [Male aged 15, atrophy of optic nerve from haemorrhage of orbit following a slight blow on eyelid. Two of his brothers had also a tendency to bleeding. No further family history.]

585. Glass: Zur Aetiologie und Therapie der Bluterkrankheit und verwandter Zustände. *Allgemeine medicinische Centralzeitung.* Berlin, 1892, Bd. lxi. S. 653, 677. [General account dealing with aetiology, therapy and dietetics of haemorrhagic conditions. No cases.]

586. Kinnigers: Ueber Haemophilie. *Der praktische Arzt.* Wetzlar, 1892, Bd. xxxiii. No. 6. S. 121. [Case of a boy, 2 years old, who drove a splinter of wood into his forefinger and then injured the mucous membrane of the mouth with it. Bled for eight days and became very anaemic and showed large blue spots on feet and legs. He had previously bled severely from wounds of his fingers. Three older sibs in the family were healthy. Father had epistaxis up to the age of 25, but did not bleed severely after injuries. Bleeding in the boy was easily arrested by ice.]

587. Marcu, L.-A.: Quelques considérations sur l'hémophilie, les hémorrhagies qu'elle provoque après l'accouchement et leur traitement. *Thèse de Paris.* 1892. [Three cases of *post partum* haemorrhage.]

588. Matsumoto, Saburo: Hetsukobio sitsuken ni zukete. (Clinical experiences on haemophilia.) *Rikugun-Guni-Gakkwaisassi.* Tokyo, 1892, No. 54, p. 27, 6 pl. [Two cases. (1) Soldier aged 21, bruised easily and suffered from epistaxis and bleeding from the gums when young, was five months in hospital with epistaxis, afterwards suffered from pleurisy; no family history of haemophilia. (2) Soldier aged 23, suffered from haematemesis and epistaxis, pain and swelling of joints; no family history. Professor Aoyama of Tokyo who abstracted Matsumoto's paper informs us that he himself has never met with a case of hereditary haemophilia in Japan.]

589. Moses, Julius: Die Bluterkrankheit. Haemophilie. *Inaug. Diss.* Greifswald, 1892. [Pedigree No. 588.]

590. Naumann, Hans: Ein Beitrag zur Lehre von der erworbenen haemorrhagischen Diathese. *Inaug. Diss.* Berlin (1892). [Male aged 34, morbus maculosus Werlhofii with localisation of haemorrhages in skin and mucous membranes. Autopsy showed haemorrhagic pericarditis and chronic nephritis. Mother died of cholera. Father, two brothers and one sister alive and well.]

591. Rachford, B. K.: Hemophilia. *The Med. News.* Philadelphia, 1892, Vol. lx. p. 227. [Pedigree No. 542.]

592. Schmidt, Alexander: Zur Blutlehre. Leipzig, 1892. [Classical work on the coagulation of the blood.]

593. Watkins, J. L.: Haemophilia; its pathology and treatment; with report of cases. *The New York Med. Journ.* New York, 1892, Vol. lvi. p. 172. [Male aged 61, epistaxis began at 49. At 59 he bled from every orifice, natural or traumatic, in the body. No family history.]

594. Makins, G. H.: A large blood tumour developing at the mouth of a wound in a case of haemophilia. *Transactions of the Pathological Society of London.* London, 1892—3, Vol. xliv. p. 165. [Boy aged 12, comminuted fracture of humerus with swelling and rigor on the fifth day. In St Thomas's hospital two days later the swelling was incised, fluid serum and blood clot being evacuated. At first progress was good, later clot began to collect at mouth of wound which was again cleared on the sixteenth day, when parenchymatous bleeding set in. He died on the seventeenth with an enormous blood mass extending from the front of the shoulder to midsternum, upwards to above the clavicles, and downwards to nearly the lower edge of ribs. Family history disclosed that he was a bleeder. "Mother's cousin female, mother's niece, one brother and one sister."]

595. Afonski, N. D.: Sluchaĭ krovotochivosti u 12-lïetnyavo malchika. *Meditsinskoye Obozrïeniye.* Moskva, 1893, Vol. xxxix. p. 348. [Boy 12 years old, described as having haemophilic joints, insufficient account.]

596. Albertoni, Pietro: Studi clinici sulle affezioni emorragiche. *Bullettino delle scienze mediche di Bologna.* Bologna, 1893, Anno lxiv. Serie vii. Tomo iv. p. 563. [Two cases. (1) Male aged $19\frac{1}{2}$, a student of arts who suffered from epistaxis from the age of 6 onwards. He was also said to have bled easily from slight scratches. The maternal grandfather—alive at the age of 84—suffered from epistaxis and a relative bled severely after tooth extraction. Two cousins also bled freely. Patient's mother had amenorrhoea, epistaxis, haemoptysis and tuberculosis. Examination of patient's nose by Secchi showed nothing abnormal. (2) Student

aged 23, bruised easily in infancy and suffered from epistaxis. Haemorrhage after tooth extraction, haematuria, blood in stools, etc. No family history of haemophilia.]

597. Chisolm, F. M.: Repeated and profuse haemorrhage subsequent to puncture of a Meibomian cyst. *Medical Record.* New York, 1893, Vol. xliv. p. 523. [Mulatto aged 35, sufficiently described in title.]

598. Dalziel, T. K.: Case of a female patient with symptoms resembling those of haemophilia. *Transactions of the Glasgow Pathological and Clinical Society.* Glasgow, 1893, Vol. iv. p. 214, and 1895, Vol. v. p. 56. [Female aged 21 with abscess on fifth metacarpal bone, not haemophilia.]

599. Dunn, T. D.: Peliosis rheumatica in a bleeder. *Transactions of the College of Physicians of Philadelphia.* 1893, 3 s. Vol. xv. p. 100. And *American Journal of the Medical Sciences.* Philadelphia, 1893, n. s. Vol. cvi. p. 701. [Account of VI. 6, in Dunn's Case V. see Pedigree No. 436.]

600. Kardamates, Joannes P.: Τέσσαρα ἀτυχήματα αἱμοφιλίας ἐκ μητρὸς λευχαιμικῆς. Γαληνὸς. Ἀθῆναι, 1893, Vol. xxiii. p. 646. [Four male children died of haemorrhage of a nature not specified, three before and one synchronously with an attack of leukaemia in the mother.]

601. Moffet, G. E.: A case of haemophilia. *Army Medical Department Report.* London, 1893, Vol. xxiii. p. 397. [Case of fatal haemorrhage after tooth extraction in a soldier aged 18 years, 8 months. He had previously had epistaxis lasting for days and had nearly bled to death from a trifling scalp wound (scar visible). The mother "a well educated and intelligent woman" supplied a family history of bleeders both males and females, the evidence of haemophilia being haemorrhage after slight cuts, and circumcision, epistaxis, *post partum* haemorrhage, severe crush of leg, haemorrhage after extraction of a polypus. In our opinion evidence of haemophilia insufficient.]

602. Müller, O.: Haemophilia congenita; tödtliche Blutung aus den Augenbindehäuten. *Arch. f. Gynaekologie.* Berlin, 1893, Bd. xliv. S. 269. [New born female child, bled to death after the application of lunar caustic solution to the eyes.]

603. Ottava: Ein Fall von Augenoperation an einem hämophilen Individuum. *Gesellschaft der Aertze in Budapest. Sitzung von* 18, iii. 1893. [Tenotomy on left eye, great haemorrhage and extravasation into orbit, exulceration of bulbus, and in six months total symblepharon.]

604. Recht: Metrorrhagie chez une vierge hémophile. *Bulletins de la société anatomique de Paris.* Paris, 1893, lxviii^{e} année, 5me série, Tome vii. p. 207. [Demonstration of blood clots obtained from the uterus of a female who had been very unhealthy and had suffered from haemorrhage and ecchymoses.]

605. Sandelin, E.: Hämofilernas arthropathier. *Finska Läkaresällskapets Handlingar.* Helsingfors, 1893, Bd. xxxv. Häft 10, S. 725. [Male aged 6 was knocked down two years ago and hurt his R. knee. Great swelling occurred at the time and on several occasions later. In the hospital tubercle was diagnosed and the joint opened by transverse incision. Bloodstained serum and blood clots escaped, and the capsule was found to be of a brown-yellow-green colour. At first the bleeding was slight, but later very severe, the whole surface oozing. To stop the bleeding the surface was treated with the Paquelin but within a week infection had taken place. Later abscess formed in popliteal space and recurrence of bleeding from the knee. Epistaxis and bleeding from the gums were superadded and he died about eight weeks after the operation. Autopsy. No history of haemophilia in the family.]

606. Wright, A. E.: On a method of determining the condition of blood coagulability for clinical and experimental uses and on the effect of the administration of calcium salts in haemophilia and actual and threatened haemorrhage. *The Brit. Med. Journ.* London, 1893, Vol. ii. p. 223. [Experiments illustrating the therapeutical value of calcium.]

607. Zoege von Manteuffel: Bemerkungen zur Blutstillung bei Haemophilie. *Deutsche med. Wchnschr.* Leipzig und Berl., 1893, Bd. xix. S. 665. [Pedigree No. 562.]

608. Broca, A.: Hémophilie rénale et hémorrhagies rénales sans lesion connue. *Annales de maladies des organes génito-urinaires.* Paris, 1894, Tome xii. p. 881. [Female aged 28, haematuria at 27. R. renal region tender. Operation. Palpation of kidney revealed nothing abnormal, but although the kidney was left intact the symptoms disappeared.]

609. Caillé, Augustus: Haemophilia (bleeders). *The Postgraduate.* New York, 1894, Vol. ix. p. 141, 1 pl. [Pedigree No. 595.]

610. Clark, Wm.: Haemophilia. *The Cleveland Medical Gazette.* Cleveland, O., 1894, Vol. x. p. 570. [Epistaxis and haematemesis in an alcoholic German aged 31. Death. Inadequate family history.]

611. Knudtzon, H.: To Tilfaelde af Haemofili. *Norsk Magazin for Laegevidenskaben.* Christiania, 1894, 4 R. Bd. ix. S. 98. [Pedigree No. 593.]

612. Olivier: Hémophilie chez une jeune fille de 13 ans, réglée pour la première fois. *Journal de médecine de Paris.* 1894, 2e ser. Tome vi. p. 379. [When young she fell on her head and a large haematoma formed, severe bleeding after tooth extraction. Menstruated for the first time at 13 years when the flow of blood was so great that she was in danger of her life. No family history stated.]

613. Passet, J.: Ueber Haematurie und renale Hämophilie. *Centralbl. für die Krankheiten der Harn- und Sexualorgane.* Leipzig, 1894, Bd. v. S. 397. [As in title, female, no family history of haemorrhage.]

614. Romme, R.: La diathèse hémorrhagique et les hémorrhagies gastro-intestinales chez les nourrissons. *Revue mensuelle des maladies de l'enfance.* Paris, 1894, Tome xii. p. 198. [As in title, not haemophilia.]

615. van der Stok: Bloederziekte. *Nederlandsch militair geneeskundig Archief van de Landmacht, Zeemacht, het Oost en West-Indisch Leger.* Leiden, 1894, Bd. xviii. S. 173. [Boy 7 years old, several attacks of epistaxis, died of bleeding from the nasopharynx. A brother had haematuria, parents healthy.]

616. White, J. W.: A case of haemophilia. *The Lancet.* London, 1894, Vol. ii. p. 740. [Male aged 5, ecchymoses, haemarthros, haemorrhage after tooth extraction. "Hereditary transmission followed the ordinary course."]

617. Wightman, J. P.: Notes and family history of cases of haemophilia. *The Lancet.* London, 1894. Vol. i. p. 535. [Pedigree No. 431.]

618. Zitrin, M. G.: Krovot. *Feldsher.* S.-Peterburg, 1894, Vol. iv. p. 441. [Haemophilia. No heredity.]

619. Daland, J. and Robinson, W. D.: Three cases of spontaneous haemophilia in brothers. *The Maryland Medical Journal.* Baltimore, 1894—5, Vol. xxxii. p. 389. [Pedigree No. 597.]

620. Matas, R.: Hemophilia, failure of hemostatics and suture; arrest of hemorrhage by direct elastic compression. *The New Orleans Medical and Surgical Journal.* 1894—5, n.s. Vol. xxii. p. 251. [Italian baby 9 months old, contusion of upper lip from fall, great haemorrhage, recovery. Professor Matas informs us (Aug. 1909) that 10 years later he saw this child with typical haemarthros of the R. knee, which was aspirated. Recovery. Patient now well.]

621. Barrett, J. W.: Case of haemophilia treated by the administration of lime. *The Australian Medical Journal.* Melbourne, 1895, n.s. Vol. xvii. p. 340. [Male aged 4½, probably haemophilia, no family history.]

622. Fischer, H.: Ueber Hämophilie. *Ztschr. f. Wundärzte und Geburtshelfer.* Fellbach, 1895, Bd. xlvi. S. 338. [Same case as Bibl. No. 623.]

623. Fischer, H.: Ueber Haemophilie. *Medicinisches Correspondenz-Blatt d. württembergischen aerztlichen Landesverein.* Stuttgart, 1895, Bd. lxv. S. 65, 73. [Pedigree No. 416.]

624. Gahbler, Theodor: Beitrag zur Casuistik der primären und secundären haemorrhagischen Diathese. *Inaug. Diss.* Greifswald, 1895. [Male aged 7; from infancy bled severely from smallest wounds. At 6 months was bitten on the head by a fly and bled for four days. At 6 bled for a week from tooth extraction; also epistaxis and haemorrhage from gums. In the hospital he was covered with bruises. No family history of haemorrhage.]

625. Gavriloff, I. M.: Sluchaĭ hemorragicheskavo diateza. *Feldsher.* S.-Peterburg, 1895, Vol. v. p. 332. [Case of haemorrhagic diathesis.]

626. Gayet, G.: Arthropathies et hématomes diffus chez les hémophiles. *Gaz. hebdomad. de méd. et de chir.* Paris, 1895, Tome xxxii. p. 258. [Three cases shortly described, no family history. (1) Male aged 16, no personal history, painful affections of several joints. (2) Male aged 12; since 2, epistaxis, bleeding from gums and ecchymoses. Sudden swelling of left arm, temp. 102·2° F. Ecchymoses. Swelling fluctuated, incision, evacuated clots and bloodstained fluid. (3) Male aged 9, epistaxis since birth, haemorrhage and bruises from slightest injury. Sudden swelling of knee on three occasions in three months. Skin blue over calf, incision, evacuated clots and bloodstained fluid.]

627. Guttmann: Zwei Fälle von Hämophilie. *Zeitschrift für aerztliche Landpraxis.* Frankfurt a. M., 1895, Bd. iv. S. 364. [Male aged 40, persistent haemorrhage after tooth extraction. A year later his son aged 3 had ulcerated glands in his neck. Incision, prolonged bleeding, arrest by sutures. No other data.]

628. Hirsch, Theodor: Die Gelenkerkrankungen bei Hämophilie und deren Behandlungen. *Inaug. Diss.* Würzburg, 1895. [Pedigree No. 586.]

629. Hoersen, E.: Ueber einige Fälle von haemorrhagischer Diathese (Morbus maculosus Werlhofii, Scorbut und Hämophilie). *Inaug. Diss.* Bonn, 1895. [Male with indefinite history of bleeding. No family history of haemorrhage.]

630. Kolster, Rud.: Om hämofili hos qvinnor. *Finska Läkaresällskapets Handlingar.* Helsingfors, 1895, Bd. xxxvii. Häft iii. S. 145. [Girl aged 3½ years. Since the age of 1, occasional blood-stained discharge from vagina. Blood in stools and epistaxis and bleeding from the mouth had also been observed. When Kolster saw her she had been fretting and languid for a few days. The left hand and leg had been swollen and covered with crops of purpuric spots. Two days later similar condition on right side and on the abdomen as high as the umbilicus. On examination her legs especially ankles and knees were swollen and covered anteriorly with purpuric spots. There was effusion of blood into the left knee, although the grounds on which this diagnosis was made are not stated. The hands and arms were in a similar condition. An haematoma was noticed on the vertex. The mucous membranes were unaffected. In the course of her illness which lasted a fortnight haematemesis and haematuria also occurred in addition to a bloody discharge from the vagina. Her face became swollen and slight fever was present. There was a slight relapse after she got up. Her mother always menstruated normally. She however was frequently afflicted with epistaxis and wounds always bled a long time. She had "remittent fever" two years before during which her body was covered with purpuric spots. A brother of this woman suffered very much from bleeding from small wounds. The family described by Kolster was Finnish, but nothing was known about the other members.]

631. Löns, H.: Beiträge zur Haemophilie. *Inaug. Diss.* Halle a. S., 1895. [Pedigrees Nos. 526, 527, 528, 529.]

632. Nason, E. N.: Case of haemophilia. *Birmingham Medical Review.* Birmingham, 1895, Vol. xxxviii. p. 289. [Male aged 30, primary and secondary haemorrhage from wound of wrist. No family history of bleeding.]

633. Nordberg, A.: Ett bidrag till kännedomen om hämofilins förekomst i Finland. *Finska Läkaresällskapets Handlingar.* Helsingfors, 1895, Bd. xxxvii. S. 94. [Pedigree No. 417.]

634. Pague, F. C.: Hemophilia. *Pacific Coast Dentist.* San Francisco, 1895, Vol. iii. p. 72 [Male aged 24, bleeding from the gum round the second upper bicuspid which had been broken off during an unsuccessful attempt at extraction. Haemorrhage lasted 17 days. He had previously bled (1) from a cut on the thumb for six weeks, (2) on another occasion from a scalp wound caused by the kick of a horse. A maternal uncle nearly bled to death after tooth extraction.

635. Rosner, Emil: Ein Beitrag zur Lehre von den Gelenkerkrankungen bei Blutern. *Inaug. Diss.* Breslau, 1895, 31 pp. [Male aged 26 in Mikulicz's Klinik. As a child had epistaxis and great bleeding from slight injuries. In his 8th year was rendered exsanguine from a wound on finger. Elbow joint early affected. In 1887 R. knee swelled and this recurred frequently on both sides. On one occasion 100 c.c. of pure blood were aspirated from the knee. Ultimately he was crippled. Parents and four brothers and sisters all healthy.]

636. Schmidt, Alexander: Weitere Beiträge zur Blutlehre, nach des Verfassers Tode herausgegeben. Wiesbaden, 1895. [Classical work on blood coagulation.]

637. Berggrün, Emil: Ein Fall von haemorrhagischer Diathese mit Hirnblutung. *Archiv fur Kinderheilkunde.* Stuttgart, 1896, Bd. xxi. S. 84. [Case of cerebral haemorrhage with paralysis. Fever with profuse haemorrhage from the genitals in a female aged 9.]

638. Bradley, A. E.: Urethral haemorrhage; haemophilia. *The Medical News.* New York, 1896, Vol. lxviii. p. 15. [Urethral haemorrhage in a man aged 33 who had been treated for stone.]

639. Chauffard, A.: Hémophilie avec stigmates télangiectasiques. *Bulletins et mémoires de la société médicale des hôpitaux de Paris.* 1896, 3 s. Tome xiii. p. 352. [Title explains itself; female aged 50.]

640. Comby, J.: Hémophilie chez une filette de onze mois. *Gazette des hôpitaux.* Paris, 1896, p. 793, and *Bulletins et mémoires de la soc. méd. des hôpitaux de Paris.* 1896, 3 s. Tome xiii. p. 558. [Bleeding from mouth, tongue, intestine, nose, ear and skin. Uncle died of epistaxis at the age of 21.]

641. Dop, G.: Des hémorrhagies alvéolaires chez les hémophiles; observations. *Archives médicales de Toulouse.* Toulouse, 1896, Tome ii. p. 313. [General account of haemophilia with special reference to alveolar haemorrhages: two cases described, (*a*) female aged 34 with pyorrhoea alveolaris, (*b*) male aged 13, whose father is said to have been haemophilic.]

642. Eichorst, H.: Hämophilie in Eulenburg's Real-Encyclopädie der gesammte Heilkunde. Wien und Leipzig, 1896, 3te Aufl. Bd. ix. S. 44. [Good general account of haemophilia.]

643. Elb, G.: Zur Kenntniss der renalen Haemophilie. *Inaug. Diss.* Berlin, 1896. [Not haemophilia.]

644. af Forselles, Arthur: Ein Beitrag zur Kenntniss der Gelenkerkrankungen bei Blutern. *Centralbl. f. Chirurgie.* Leipzig, 1896, Bd. xxiii. S. 19. [Pedigree No. 596.]

645. Linser, P.: Beitrag zur Kasuistik der Blutergelenke. *Beiträge zur klinische Chirurgie.* Tübingen, 1896, Bd. xvii. S. 105. [Pedigree No. 421.]

646. Meynet: Des arthropathies hémophiliques. *Thèse de Lyon.* 1896, No. 1178. [Two original cases, no family history in either. (1) Male aged 18 alleged typical bleeder. At 9 haemarthrosis of both knees continued up to date, epistaxis. (2) Young male with condition of knee diagnosed as tubercle but hearing of epistaxis and rebellious haemorrhage from slight injury diagnosis was altered to haemophilia.]

647. Neeley, J. H.: Haemophilia. *The Dental Journal.* Ann Arbor, Mich. 1896, Vol. v. p. 71. [Slight references to a male aged 12 alleged to be a bleeder of a bleeder family. Two teeth extracted. Haemorrhage not very alarming.]

648. Summers, J. E., jr.: Hemophilia, an unrecognised case of which terminated fatally after excision of the knee joint. *Medical Record.* New York, 1896, Vol. xlix. p. 336. [Male aged 10 as in title. Knee became inflamed at the age of 8; diagnosis tubercle. Purpuric spots were noticed before the operation. No other evidence of haemophilia. Parents were germans and had a family history of tubercle.]

649. Wright, A. E.: On the treatment of the haemorrhages and urticarias which are associated with deficient blood coagulability. *The Lancet.* London, 1896, Vol. i. p. 152. [As in title.]

650. Averill, C.: Idiopathic haemorrhage from the umbilicus in an infant. *Brit. Med. Journ.* London, 1897, Vol. i. p. 393. [Jaundice, purpura, death.]

651. Bienwald, P.: Ein Fall von Hämophilie. *Deutsche medicinische Wochenschrift.* Leipzig und Berlin, 1897, Bd. xxiii. S. 28. [Boy aged 2, who had been frequently under observation for cutaneous blood extravasation, was successfully treated for haemorrhage from a small wound by local application of blood taken from his grandmother. No account of relatives.]

652. Combemale: Un cas d'hématurie chez un hémophilique. *L'Écho médical du nord.* Lille, 1897, Tome i. p. 121. [An account of affection of joints with epistaxis and haematuria in a male. No family history.]

653. Combemale: Sur l'hémophilie. *L'Écho médical du nord.* Lille, 1897, Tome i. p. 455. [Epistaxis and purpura in a female aged 17.]

654. Dejage, L.: Un cas d'hémophilie traité avec succès par le corps thyroide. *Journal de clinique et de thérapeutique infantiles.* Paris, 1897, Tome v. p. 998. [A lady (age?) suffering from purpura on legs, thighs, arms, trunk, treated by thyroid extract; recovery. No family history given.]

655. Finkelstein, H.: Ein Fall von haemorrhagischer Diathese bei einem Neugeborenen. *Charité Annalen.* Berlin, 1897, Bd. xxii. S. 311. [Male infant died on fifth day, general sepsis and syphilis.]

656. Fussell, M. Howard: Two cases of haemophilia. *Brit. Med. Journ.* London, 1897, Vol. ii. p. 1230. [Pedigree No. 566.]

657. Helfrich, Chas. H.: A case of hemophilia with haemorrhage from the lower eyelid. *Transactions of the Homoeopathic Medical Society of the State of New York.* 1897, Vol. xxxii. p. 39. [Haemorrhage from left eye, breasts and umbilicus in a female child 16 days old; death. Mother in early life had epistaxis.]

658. Mühl-Kühner: Ein Fall von Alveolarblutung mit nachgefolgtem Tod. *Münchener med. Wochenschrift.* München, 1897, Bd. xliv. S. 857. [As in title, adult male. Two brothers said to be haemophilic. No data.]

659. Nesterovski, I.: Sluchaĭ obriezaniya hemofilika. *Dĭetskaya meditsina.* Moskva, 1897, Vol. ii. p. 36. [Pedigree No. 564.]

660. Oppenheimer, Seymour: A study of the nares and pharynx in a case of haemophilia. *New York Med. Journal.* New York, 1897, Vol. lxvi. pp. 767, 802. [Female aged 32 with "rheumatic heart trouble" and epistaxis.]

661. Perry, J. Clifford: The treatment of haemophilia with chloride of calcium with report of a case. *The Journal of the American Medical Association.* Chicago, 1897, Vol. xxviii. p. 492. [Male Russian aged 20, profuse haemorrhage following incision into alveolar abscess. He also bled from mere scratches. Two brothers bled to death in infancy from trivial injuries.]

662. Phillips, H. H.: Idiopathic bleeding from the umbilicus in an infant; recovery. *The Brit. Med. Journ.* London, 1897, Vol. i. p. 971. [As in title, no family history.]

663. Reed, R. Harvey: Hemophilia. *The Columbus Medical Journal.* Columbus, O., 1897, Vol. xix. p. 119. [Two cases. (1) Male aged 28, haemorrhage after tooth extraction, ligation of facial and carotid artaries. Haemostasis obtained by red hot poker. (2) Male, general oozing from teeth and roof of mouth, fever, abdominal symptoms, coma, death.]

664. Shaw, J. E.: A case of haemophilia with joint lesions. *Bristol Med.-Chir. Journal.* Bristol, 1897, Vol. xv. p. 240. [Re-investigated Greig-Smith's Case, Bibl. No. 483, and Pedigree 574.]

665. Siffre, A.: Note sur l'hémophilie dans l'avulsion des dents. *Revue odontologique.* Paris, 1897, Tome xvi. p. 161. [Boy aged 11 with carious upper molar. On account of alleged haemophilia Siffre decided to conserve the tooth, but later it broke and had to be extracted. Great haemorrhage in spite of all kinds of styptics. No family history of bleeding.]

666. Silvestrini, Raffaelo, e Baduel, Cesare: Le infezioni emorragiche nell' uomo. *Il Policlinico.* Roma, 1897, Vol. iv., Sezione Medica, p. 13. [Not haemophilia.]

667. Stowell, William L.: Hydraemia, haematoma, sepsis, recovery. *The New York Medical Journal.* N.Y., 1897, Vol. lxvi. p. 80. [Male aged 14, epistaxis. Fell and sustained swelling of buttock. Incision revealed nothing abnormal. Later discharge of serum. Subsequent suppuration.]

668. Valude, E.: Hématome orbito-palpébral à répétition chez une hémophile. *Annales d'oculistiques.* Paris, 1897, Tome cxvii. p. 190. [Female aged 34, always a great tendency to haemorrhage. As in title. No family history.]

669. Valude, E.: Repeated attacks of orbito-palpebral hematoma in a case of hemophilia. *The Journal of Ophthalmology, Otology and Laryngology.* New York, 1897, Vol. ix. p. 323. [Same case as Bibl. No. 668.]

670. Vickery, H. F.: Hemophilia. *The Boston Med. and Surg. Journal.* Boston, 1897, Vol. cxxxvi. p. 226. [Three cases. (1) Robust young man; formerly ecchymoses and epistaxis. (2) Female aged 24, purpura, epistaxis and menorrhagia. (3) Female "blue baby," easily bruised. Bled from teeth. Menorrhagia at 13.]

671. Wagenmann, A.: Spontaner Hämophthalmos bei hereditärer Haemophilie. *von Graefe's Archiv für Ophthalmologie.* Leipzig, 1897, Bd. xliii. S. 207. [Male 25, haemorrhage into vitreous humour, epistaxis. Uncle died of tooth extraction.]

672. Ammann, E.: Das Vererbungsgesetz der Hämophilie bei der Nachtblindheit. *Correspondenzblatt für schweizer Aerzte.* Basel, 1898, Bd. xxviii. S. 623. [Title explains itself. No cases.]

673. Brown, W. H.: A case of haemophilia, oxygen inhalation, recovery. *The Lancet.* London, 1898, Vol. ii. p. 1474. [Pedigree No. 392.]

674. Combemale et Gaudier: L'opothérapie thyroidienne dans les accidents hémophiliques. *L'Écho médical du nord.* Lille, 1898 Tome ii. p. 198. [Epistaxis, metrorrhagia and other haemorrhages in a female.]

675. Dent, Clinton T.: Mental peculiarities in haemophilia. *British Medical Journal.* London, 1898, Vol. i. p. 1066. [Refers to the mental state of a male aged 16 who died of haemorrhage in St George's Hospital after the removal of a tooth. He denied that he was a bleeder and died cursing his mother and "all who had to do with him."]

676. Fry, Albert: The successful treatment of haemophilia by the injection of serum. *Medical Record.* New York, 1898, Vol. liv. p. 131. [Treatment of three brothers, bleeders, with horse serum. From the description the cases are unquestionably the same as in McLane Hamilton's Case, Bibl. No. 568. Pedigree No. 397.]

677. Gage, G. C.: Hemostatic effect of hydrogen peroxide as shown in two cases of hemophilia. *New York Eye and Ear Infirmary Reports.* 1898, Vol. vi. p. 119. [Two cases. First, male aged 18, severe epistaxis, polypus removed from nose, haemorrhage resisting treatment except as in title; severe bleeding after trifling injury. Uncle on mother's side a severe bleeder. Second, female aged 46, formerly had purpura; epistaxis all her life. Enlarged turbinal removed, severe haemorrhage. She thought her aunt was a bleeder.]

678. Goetz, Hermann: Ueber einen Fall von Haemophilie. *Inaug. Diss.* München, 1898, 38 pp. [Male aged 23, had always been well up to the age of 19; after that epistaxis frequent and severe. Is said to have nearly died from incision into abscess of chin. In hospital with epistaxis, gonorrhoea and haematuria. No family history of haemophilia.]

679. Hennessey, J. C.: A case of hemorrhagic diathesis. *The Pacific Medico Dental Gazette.* San Francisco, 1898, Vol. vi. p. 457. [Female aged 17, bled for three days after tooth extraction.]

680. James, John V.: A strange case of haemorrhage. *Indian Med. Record.* Calcutta, 1898, Vol. xiv. p. 317. [Female aged 68, bleeding from eyes, ears, nose and mouth. First attacked at 55.]

681. Jones, C. R.: A case of haemophilia. *Brit. Med. Journal.* London, 1898, Vol. ii. p. 987. [Female aged 7 suffering from haematemesis and bruising. Swelling of both knees and wrists with discolouration a few days later. A pin prick bled 24 hours and a tooth which fell out oozed 24 hours. No family history. Not haemophilia.]

682 Kenefic, J. A.: A case of fatal haemorrhage following adenectomy in a haemophilic child. *The Laryngoscope.* St Louis, 1898, Vol. iv. p. 251. [Death following removal of adenoids in a male aged 4. No evidence of haemophilia. No family history.]

683. Legg, J. Wickham: Haemophilia, System of Medicine (Allbutt). London, 1898, Vol. v. p. 548. [General account.]

684. Ligorio, E.: Contributo alla casuistica delle artropatie negli emofilii. *La Settimana medica, dello Sperimentale.* Firenze, 1898, Tomo lii. p. 445. [Clinical history of a male child, 2 years old, who presented a swelling of R. knee. A surgeon, believing it to be tuberculous, made an incision, but found only some haemorrhagic spots. Severe haemorrhage after the operation, but ultimately complete recovery. A brother died of haemorrhage from a small cut on the upper lip, another brother bruised easily. A sister was healthy. No family history of haemophilia.]

685. Litten, M.: Die Krankheiten der Milz und die haemorrhagischen Diathese. Nothnagel's specielle Pathologie und Therapie. Wien, 1898, Bd. viii. Theil iii. [General account of haemorrhagic diseases.]

686. Nash: A case of haemophilia. *Brit. Med. Journ.* London, 1898, Vol. i. p. 883. [Pedigree No. 567.]

687. Northrupp, W. P.: Haemophilia. An American Textbook of the Diseases of Children (Starr). London, 2 Edit. 1898, p. 377. [General account.]

688. Powell, C. H.: A remarkable case of haemophilia. *The North American Journal of Diagnosis and Practice.* St Louis, 1898, Vol. i. p. 19. [Irish-American, female, first had haemorrhage from stomach and bowel when 23. Two years later menorrhagia, and two years after this bleeding from gums, bowels, nose, vagina, stomach; purpura; recovery. No other member of family subject to bleeding.]

689. Sabrazès, J., and Cabannes, C.: Arthropathies des hémophiles; leur diagnostic radiographique. *Gazette hebdomadaire des sciences médicales de Bordeaux.* 1898, Tome xix. p. 495. [History of a male bleeder aged 35. Parents and grandparents and a brother not affected. At 7 and at 10 bit his tongue and bled many days. At 15 rebellious haemorrhage from small wound on R. temple. At 30 R. knee swelled and cautery was applied. Patient elevated one of the cautery eschars with his finger and sustained a great haemorrhage. The kick of a gun bruised him severely. At 18 a small growth appeared on his tongue and he tied a silk thread round it; great haemorrhage ensued. In 1897 abscess developed in connection with carious molars, and on incision violent bleeding set in and he was taken to hospital, where an examination of his blood and joints by Sabrazès took place. Many of his joints had been affected since he was 4 or 5.]

690. Sadler, Ernest, A.: A family of bleeders. *The Birmingham Medical Review.* London and Birmingham, 1898, Vol. xliv. p. 45. [Pedigree No. 391.]

691. Thébaud, A.: Contribution à l'étude des arthropathies hémophiliques et de leur diagnostic par la radiographie. *Thèse de Bordeaux.* 1898. [General account of haemophilic joints illustrated by cases from the well known publications of König, Sabrazès and others.]

692. Wittner, M.: Ein Fall von Hämophilie bei einem Neugeborenen. *Allgemeine wiener medizinische Zeitung.* Wien, 1898, Jahrg. xliii. S. 199. [In Dorohoiu, Rumania, male aged 8 days who bled profusely after circumcision. The mother said that two other boys had bled to death after circumcision whereas a daughter was alive and well. As Wittner heard this with astonishment an old woman—the grandmother—who had been deep in prayer completed the story that she (the old woman?) had lost eight boys after circumcision. One of her daughters died of haemoptysis, two others being well. A "Brenner F" is suddenly mentioned, the old woman adding that he had been clever, as after losing two boys from circumcision he did not have the third one circumcised. Whether "Brenner F" was merely an acquaintance or relative is not stated. The whole account is most ambiguous and of no permanent value.]

693. Bond, A. K.: The hemorrhagic state in a new born twin. *The Maryland Medical Journal.* Baltimore, 1898—9, Vol. xl. p. 161. [Epistaxis, umbilical haemorrhage and general oozing of blood from cracks in the skin of a baby. No family history of any value.]

694. Chizh, S. F.: Sluchaĭ hemofilii. *Vrachebnîya Zapiski.* Moskva, 1899, Vol. vi. p. 278. [Case, no heredity.]

695. Davies, R. T. E.: Treatment of haemophilia. *The British Medical Journal.* London, 1899, Vol. i. p. 339. [Treatment of haemophilia in a family given without sufficient detail.]

696. Faber, Poul Kuhn: Om Ledtilfoede hos Blödere. *Hosp. Tidende.* Kjøbenhavn, 1899, 4 R. Bd. vii. S. 875. [Pedigree No. 587.]

697. Gabel, W.: Zur Casuistik der Hämophilie. *Wiener med. Wochenschrift.* Wien, 1899, Bd. xlix. col. 62. [Male aged 8, "cephalhaematoma spurium," purpura and epistaxis. No family history of haemophilia.]

698. Gocht, H.: Ueber Blutergelenke und ihre Behandlung. *Archiv für klinische Chirurgie von Langenbeck.* Berlin, 1899, Bd. lix. S. 482. [Pedigree No. 386.]

699. Heymann, Rudolf: Ueber einen Fall von Hämophilie mit erfolgreicher Anwendung der Gelatine-injection. *Münchener med. Wochenschrift.* München, 1899, Bd. xlvi. S. 1109. [Male bleeding after excision of adenoids, treated as above.]

700. Imbert, Léon: Hématurie hémophilique. *Quatrième session de l'association française d'urologie.* Paris, 1899, p. 120. [University student aged 32, had haemoptysis and subcutaneous haemorrhages after injuries when 4 or 5 years of age. At 16 arthritis and epistaxis. At 23 abundant haemoptysis and haematuria lasting 8—15 days. Father died of hepatic disease, mother of variola, two brothers died young.]

701. Launay, Léon: Contribution à l'étude des arthropathies et des hématomes chez les hémophiles. *Thèse de Paris.* 1899, No. 66, 48 pp. [Genuine case of haemophilia, with incomplete account of the family history; male aged 20. At 7 epistaxis lasting 10 days and recurring three or four times a year. Swollen joints. Bled 48 hours from a leech bite: at 10 cut finger and bled a week. Since 5 he has had 23 attacks of swelling in his ankle and three in his knee. In an attack of haemarthrosis of the knee with discolouration of whole thigh, osteomyelitis was diagnosed and the swelling boldly incised. About a litre of blood clot was turned out. Wound packed. There was slight bleeding during the first two or three days but later the wound had to be reopened and more clot evacuated. Great anaemia followed but he ultimately recovered. His father, brother and sister were normal and no history could be obtained among the ascendants or collaterals.]

702. Nichols, J. B.: The hemostatic use of gelatin with report of a case of haemophilia treated by gelatin with recovery. *The Medical News.* New York, 1899, Vol. lxxv. p. 705. [Male aged 24, large wounds of forearm and wrist with broken bottle. Secondary haemorrhage.]

703. Nové-Josserand: Arthrites hémophiliques. *La Province médicale.* Lyon, 1899, p. 366. [Report of a paper given in the Société de Chirurgie, Lyon, séance du 27 juillet 1899. Case of a boy aged $6\frac{1}{2}$ who showed symptoms of haemophilia at the age of 8 months. Slight cuts caused great bleeding; an injury produced a great haematoma on the head; pains in vertebral column and swelling of various joints. No family history of haemophilia.]

704. Nové-Josserand: Arthrites hémophiliques. *Lyon médical.* Lyon, 1899, Tome xcii. p. 231. [Same case as Bibl. No. 703.]

705. Roth, Otto: Ueber einen Fall von Sarkom verbunden mit haemorrhagischer Diathese. *Deutsche med. Wochenschrift.* Leipzig, 1899, Bd. xvii. S. 222. [Female aged 55 as in title.]

706. Siddall, J. B.: The treatment of haemophilia. *Brit. Med. Journ.* London, 1899, Vol. i. p. 531. [As in title, no cases of his own.]

707. Sieczowska, Hélène: Un cas de névrite traumatique chez un hémophilique. *Thèse de Genève* 1899. [As in title. Male aged 11; diagnosis of haemophilia based upon bleeding from gums and purpura since the age of 9; no family history.]

708. Stengel, A.: Haemophilia. *Progressive Medicine.* Philadelphia and New York, 1899, Vol. ii. p. 303. [Short review of some cases not his own.]

709. Sympson, E. Mansel: A note on haemophilia; its treatment by red bone marrow and by the continuous administration of chloride of calcium. *The Lancet.* London, 1899, Vol. i. p. 1289. [As in title with reference to two children, (1) male aged 2 with purpura, haematomata, haematuria, joint lesions, and bleeding after injury; (2) no information.]

710. Wagner, G. W.: Hemophilia. *The Physician and Surgeon.* Detroit and Ann Arbor, 1899, Vol. xxi. p 397. [Two cases. (1) A miner aged 25 bled for three weeks from crushed finger. At 15 bled one week from wound of toe; at 20 bled two weeks after tooth extraction. Parents, three sisters and three brothers healthy, two brothers bled to death, the one at 20 from a

lacerated wound two inches long on the scalp, the other at 30 from the stomach and bowel. (2) Jewish boy aged 4, bleeding from small lacerated wound of mucosa of mouth. No history obtainable.]

711. Kerr, A. A.: Haemophilia with report of two cases. *The Denver Medical Times.* Denver, Col. 1899—1900, Vol. xix. p. 177. [Pedigree No. 565.]

712. Weitz, Geo. J.: Report of a case of haemophilia. *The Indiana Medical Journal.* Indianapolis, 1899—1900, Vol. xviii. p. 294. [Pedigree No. 382.]

713. Buck, L.: Haemophilia in the negro. *Medical Record.* New York, 1900, Vol. lviii. p. 149. [Fifteen lines with reference to two cases of epistaxis in negroes.]

714. Burger, Eugen: Ueber Hämophilie mit Geschichte einer Bluterfamilie. *Inaug. Diss.* Freiburg i. Br., 1900. [Pedigree No. 401.]

715. Coste, Emile: Contribution à l'étude de l'hémophilie. Hematomes. *Thèse de Paris.* 1900, No. 442, 41 pp. [Pedigree No. 525.]

716. Etlinger, N. E. K.: K voprosu o vrozhdennoĭ krovotochivosti sluchaĭ smertelnavo krovotecheniya iz pravavo konyunktivalnavo mĭeskha u 3-nedĭelnavo rebyonka. *Yezhenedĭelnik zhurnala "Prakticheskaya Meditsina."* S.-Peterburg, 1900, Vol. vii. pp. 249, 273. [Congenital haemophilia; fatal haemorrhage from the right conjunctival sac in an infant 3 weeks old. See Bibl. No. 740.]

717. Fillebrown, Thomas: A fatal case of haemophilia. *The International Dental Journal.* Philadelphia, 1900, Vol. xxi. p. 302. [Death on the seventh day following the extraction of roots of two molars in a male aged 25. A large number of mechanical and other devices failed to stop the flow. The undertaker who embalmed the corpse reported that the walls of the arteries were unusually thin. Patient had previously bled freely from cuts and after tooth extraction. His paternal grandfather had epistaxis and his mother was inclined to bleed freely.]

718. Gavrilko, A. S.: K voprosu o krovotochivosti. *Lĭetopis Russkoĭ Khirurgii.* S.-Peterburg, 1900, Vol. v. p. 551. [No heredity.]

719. Gläser, J. A.: Drei Fälle von haemorrhagischer Diathese. *Allgemeine medicinische Central-zeitung.* Berlin, 1900, Bd. lxix. S. 331, 345, 356. [Three cases. (1) Female aged 24, purpura, fever, rigors, death; *post mortem*, haemorrhages in bone marrow and meninges. (2) Male aged 21, diagnosis Weil's disease. (3) Male aged 55, hypertrophy of heart, secondary renal cirrhosis, multiple cutaneous haemorrhages, death.]

720. Gocht, Hermann: Ueber Blutergelenke und ihre Behandlung. *Sitzungsberichte der Phys.-med. Gesellschaft zu Würzburg.* Würzburg, 1900, p. 2. [Short account and demonstration of two haemophilic brothers. Pedigree No. 386.]

721. Guttmann: Ein Fall von abondanter Blutung aus dem Nabel eines Erwachsenen. *Die aerztliche Praxis.* Würzburg, 1900, Bd. xiii. S. 278. [Male aged 45 who burst his umbilicus during attempted defaecation, one litre of blood lost. No other data.]

722. Hahn: Nierenblutungen bei Haemophilie durch Gelatin geheilt. *Münchener med. Wochenschrift.* München, 1900, Bd. xlvii. S. 1459. [Male with haematuria, had also suffered from epistaxis and haemorrhage for four weeks after tooth extraction. A brother also bled for a long time after tooth extraction. An uncle died of bleeding which could not be arrested.]

723. Hogner, R.: Haemophilia diagnosed by means of suprarenal extract. *Journal of Eye, Ear and Throat Diseases.* Baltimore, 1900, Vol. v. p. 332. [Swede aged 21, congested pharynx and adenoids.]

724. Jones, C. R.: Liquor thyroidei in haemophilia. *The Brit. Med. Journ.* London, 1900, Vol. ii. p. 1375. [Same case as Bibl. No. 681 cured by thyroid extract.]

725. Klein, Th.: Beitrag zur Bluterkrankheit. *Deutsche med. Wochenschr.* Leipzig, 1900, Bd. xxvi. S. 390. [Pedigree No. 479.]

726. Kudryashoff, A. I.: Nĭeskolko sluchayev krovotochivosti. *Zubovrachebnîy Vestnik.* S.-Peterburg, 1900, Vol. xvi. p. 403. [Several cases of alleged haemophilia, no heredity.]

727. Manteĭfel, I. R.: O sochlenovnikh krovoïzliyaniyakh u krovotochivîkh. *Vrach.* S.-Peterburg, 1900, Vol. xxi. pp. 788, 823, 852. [Male aged 12 years with haemophilic joints. Parents and sibs healthy.]

728. Masters, J. L.: Hemophilia with report of an aggravated case. *The Medical and Surgical Monitor.* Indianapolis, Indiana, 1900, Vol. iii. p. 307. [Male aged 25, very great haemorrhage from ulceration in naso-pharynx, epistaxis in the family (maternal grandfather, a brother and a sister).]

729. Neumann, Fritz: Ein Beitrag zur Kenntniss der Hämophilie. *Prager medicinische Wochenschrift.* Prag, 1900, Bd. xxv. S. 457. Also *Zahnaerztliche Rundschau.* Berlin, 1901, Bd. x. No. 446. [Various instances of haemorrhage in a family, haemorrhage from tooth extraction, haematuria, menorrhagia. No evidence of haemophilia. See Pedigree No. 605.]

730. Perez, Luis E.: Hemofilia. *La juventud médica. Órgano de la Sociedad cientifica del mismo nombre.* Guatemala, 1900, Vol. II. p. 169. [General account, no cases.]

731. Porter, C. A.: Haemophilia. *International Dental Journal.* Philadelphia, 1900, Vol. xxi. p. 295. Also translated in *Medizinische Rundschau.* Berlin, 1900, p. 594. [General account, no cases.]

732. Russell, F. P.: A case of hemorrhagic diathesis. *Clereland Journal of Medicine.* Cleveland, O., 1900, Vol. v. p. 455. [Male aged 7 fell and drove blade of pocket knife through external auditory meatus into mastoid. Secondary haemorrhage, sepsis. Later, bleeding from tooth. No family history.]

733. Steiner, W. R.: Haemophilia in the negro. *Johns Hopkins Hospital Bulletin.* Baltimore, 1900, Vol. xi. p. 44. [Pedigree No. 504.]

734. Stempel, Walther: Die Hämophilie. *Centralblatt für die Grenzgebiete der Medicin und Chirurgie.* Jena, 1900, Bd. III. pp. 721, 753, 784, 817. [General acconnt of aetiology, prognosis and therapy of cases of haemophilia published betweeu 1889—1899.]

735. Strzelbitski, I. K.: Dva sluchaya prekkodyashtshavo hemorragicheskavo diateza u dvukh rodnikh bratyev. *Meditsinskoye Obozrieniye.* Moskva, 1900, Vol. liv. p. 178. [Two brothers aged 9½ and 7 respectively, alleged to be bleeders, and to have suffered from skin and joint haemorrhages. No family history of bleeding.]

736. Tilmann: Zur Frage der Blutergelenke. *Deutsche Aerztezeitung.* Berlin, 1900, Heft 19, S. 421 [Male aged 22 healthy up to age of 19, when after a blow a swelling formed in his R. knee joint. He was able to serve as a soldier but was invalided for recurrent trouble with his joint into which an incision was made. At the operation the joint contained 36 bodies formed of coagulated blood material and the synovialis was found of a red brown colour. Great haemorrhage followed and after various therapeutic remedies had been tried he died three weeks later. No family history of haemophilia.]

737. Villemin: L'hémophilie. *Gazette des maladies infantiles.* Paris, 1900, Tome II. p. 25. [Boy of 14 seen by Villemin with an enormous haematoma of thigh. Had suffered from epistaxis for four years, and some months previously had a haematoma after a fall. Bruised easily. His brother was affected with epistaxis up to age of 20. Father had epistaxis as a child, and died of unknown cause at 52.]

738. Brook, W. H. B.: A case illustrating an attempt at the antenatal treatment of haemophilia. *Trans. of the Clinical Society of London.* London, 1901, Vol. xxxiv. p. 153. [Pedigree No. 410.]

739. Cheyne, Watson: Knee disease in a haemophilic. *The Clinical Journal.* London, 1901, Vol. xvii. p. 402. [Great haemorrhage following incision into an alleged haemophilic joint, in a boy. No history of previous bleeding or haemophilia in the family.]

740. Etlinger, N. von: Zur Casuistik der Haemophilie in Säuglingsalter. Ein Fall von tödtlicher Blutung aus dem rechten Conjunctivalsac bei einem dreiwöchentlichen Kinde. *Jahrb. f. Kinderheilk.* Berlin, 1901, 3 Folge, Bd. IV. S. 24. [Badly nourished child, jaundice, suppuration from navel, purpura, blennorrhoea of R. conjunctiva, ectropion, haemorrhage, death.]

741. Greef, J. G. Wm.: Hemophilia neonatorum. *New Yorker medicinische Monatsschrift.* New York, 1901, Bd. xiii. S. 214. [Three cases. (1) Male infant, purpura neonatorum and jaundice on fifth day. (2) Infant youngest of four, three others normal, purpura neonatorum, death at 48 hours, bleeding from injury ⅓ inch long on soft palate. Autopsy showed haemorrhage into knees and shoulder joint. (3) A sixth child, others healthy, died in first 24 hours of anaemia, after appearance of haematoma on cheek. Autopsy showed the abdominal cavity to be full of blood; interstitial haemorrhages. No family history of bleeding in any of these cases.]

742. Grusche, Walther: Die Hämophilie oder die Bluterkrankheit. *Inaug. Diss.* Halle a. S., 1901. [Pedigree No. 508.]

743. Hahn, Hermann: Beitrag zur Kasuistik der Orbitalblutungen bei Hämophilie. *Inaug. Diss.* Tübingen, 1901. [Female child who died of haemorrhage from the orbit which contained purulent granulations. No history given.]

743[a]. Holland, W. A. L.: A case of haemophilia. *Queen's Medical Magazine, the Journal of the Birmingham School of Medicine.* Birmingham, 1901, Vol. v. No. 2, p. 39. [Pedigree No. 606.]

744. McKenzie, Dan: Suprarenal gland extract in the epistaxis of haemophilia. *Brit. Med. Journal.* London, 1901, Vol. i. p. 1009. [Male aged 13, epistaxis, insufficient account, epistaxis in father and maternal grandfather.]

745. Malsch, E. A.: A fatal hemophilia of a new born. *The Texas Medical News.* Austin, 1901, Vol. x. p. 205. [Fatal umbilical haemorrhage in a female baby.]

746. Scheffler: Un cas de maladie de Werlhof. Hémophilie, traitement par la médication thyroidienne. *Archives de médecine et de pharmacie militaires.* Paris, 1901, Tome xxxvii. p. 246. [Not haemophilia.]

747. Tomka, S.: Ueber Haemophilie, Ohrblutungen. *Ungarische medizinische Presse.* Budapest, 1901, Bd. vi. S. 175. [Pedigree No. 420.]

748. Tomka, S.: Haemophilie, Blutungen aus dem Ohre. *Pester medizinisch-chirurgische Presse.* Pest, 1901, Bd. xxxvii. S. 349. [See Bibl. No. 747.]

749. Videla, J. Agustín: La hemofilia complicando un ligero traumatismo del cuero cabelludo. *La semana médica.* Buenos Aires, 1901, Vol. viii. p. 49. [Case of a female child, aged 2 years and 8 months, who while playing fell and cut her forehead slightly, large oedematous swelling. Incision, recovery. The mother alleged that after the child received the slightest injury a haematoma formed which took a considerable time to disappear.]

750. Zaviolow: Analyse du sang d'un hémophilique. *Russkiy Archiv Patologii, Klinicheskoĭ Meditsini i Bakteriologii.* Saint Petersburg, 1901, Vol. x. p. 320. [Chemical examination of blood.]

751. Arkwright, J. A.: A case of haemophilia in a woman, with symptoms of defective circulation in the legs and threatened gangrene of the toes; death with cerebral symptoms. *The Lancet.* London, 1902, Vol. ii. p. 737. [Female aged 30. No evidence of haemophilia, slight evidence in one brother.]

752. Courtin, J.: Les arthrites des hémophiles. *Gaz. hebdomadaire des sciences médicales de Bordeaux.* Bordeaux, 1902, Tome xxiii. p. 482. [Three new cases described, of which two may have been haemophilia. No family history.]

753. Mermingas, K.: Beitrag zur Kenntniss der Blutergelenke. *Archiv für klinische Chirurgie.* 1902. Bd. lxviii. S. 189. [Three cases of haemophilic joints in three males in König's Clinic. No family history.]

754. Milligan, William: Suprarenal extract as a haemostatic in haemophilia. *The Brit. Med. Journ.* London, 1902, Vol. i. p. 266. [Boy aged 10, adenoids.]

755. Pincus, L.: Zur Castratio uterina atmocaustica bei Hämophilie. *Centralbl. für Gynaekologie.* Leipzig, 1902, Bd. xxvi. S. 573. [No case.]

756. Piollet, P.: Les arthropathies hémophiliques. *Gazette des hôpitaux.* Paris, 1902, Tome lxxv. p. 385. [Extensive general account with bibliography.]

757. Pittaluga, Gustavo: Paludismo y hemofilia, contribucion clinica. *La medicina de los niños.* Barcelona, 1902, Vol. iii. pp. 268, 299. [Italian girl of 10 living at Ostia. Previous history of bleeding from mucous membrane of pharynx, nose, rectum and vagina. Repeated attacks of malaria, after which there was grave haemorrhage from the gums and mucous membrane of the floor of the mouth; recovery. Mother alleged to have bled in youth. We are indebted to Dr Cervera, Madrid, for the Spanish transcript of Pittaluga's paper and to Mr Huÿssen for its translation into English.]

758. (Türk): No title. *Sitzung der Gesellschaft für innere Med. in Wien.* 1901. *Dec.* 19. *Centralbl. f. innere Med.* Leipzig, 1902, Bd. xxiii. S. 208. [Demonstration of a female belonging to a family of five generations. No details.]

759. Wallis, C. Edward: On the treatment of haemophilia with calcium chloride. *The Brit. Med. Journ.* London, 1902, Vol. i. p. 1141. [Female, haemorrhage after tooth extraction. Teeth all very foul.]

760. Carra, P.: Chez une hémophile, suite grave de la cure radicale du chalazion. *Bulletins et mémoirs de la société française d'ophthalmologie.* Paris, 1903, Tome xx. p. 347. [Female aged 56. No family history.]

761. Dommartin, Jules: Contribution à l'étude de l'hémophilie. *Thèse de Paris.* 1903, 137 pp. [Good general account of haemophilia with extensive bibliography (many errors!). Twenty cases described of which only six are original and bear slight evidence of haemophilia. (1) Male aged 21, epistaxis and bleeding from teeth, haematuria twice. Slight cuts produced great losses of blood, ecchymoses but no joint lesions. No history of haemophilia in family. (2) Male,

neurasthenic, aged 45, bleeding from gums for last two years. No previous history of bleeding, no haemophilia in family. (3) Male aged 5 with epistaxis and atrophic rhinitis, bled easily from cuts. Two maternal uncles and a cousin had had epistaxis. (4) Male aged 7 had suffered from epistaxis since he was 6 months old. (5) Male aged 41, frequent epistaxis since the age of 16, one attack of haematuria at the age of 40. (6) Female aged 12½, epistaxis without cause twice or thrice daily; fell on face and bled from nose and mouth; bled freely after opening of sinus maxillaris. Was never ill till 7. Father's sister had haemorrhagic variola and epistaxis. Dommartin then cites the cases of Jalaguier, Kirmisson, Combemale, Launay, Gayet, Potain, Comby, Hémard, Pinard, Dejage, Duany-Soler, Valude, Hughes, Tardieu, Osborne, Dubois, Perry, Wallis, Wright. Case XXIX. has no name (Limbeck's?), and a second pedigree also numbered XXIX. and called "Klebs" is a mutilated pedigree of the Mampel family.]

762. Faludi, G.: Haemophilia érdekesebb esete. *Gyermekgyógyászat.* Budapest, 1903, p. 44. [Same case as Bibl. No. 766.]

763. Koester: Ueber die Vererbung in der Bluterfamilie Mampel. *Deutsche medicinische Wochenschrift.* Leipzig, 1903. Vereins-Beilage, S. 378. [Pedigree No. 389.]

764. Abderhalden, Emil: Beitrag zur Kenntnis der Ursachen der Hämophilie. *Beiträge zur pathol. Anatomie und zur allgemeinen Pathologie hrsg. von E. Ziegler.* Jena, 1904, Bd. xxxv. S. 213. [Pedigree No. 412.]

765. Blaker, P. Stanley: Haemophilia in a female child. *The Brit. Med. Journal.* London, 1904, Vol. i. p. 189. [Bruises and haemorrhages from the gums following an injury in a female 11 months old, four weeks after measles.]

766. Faludi, Géza: Ein seltener Fall von Haemophilie. *Archiv f. Kinderheilkunde.* Stuttgart, 1904, Bd xxxix. S. 92. [Pedigree No. 422.]

767. Francis, Ernest: A case of haemophilia treated with adrenalin chloride. *The British Med. Journ.* London, 1904, Vol. i. p. 1247. [A wealthy high cast male Hindu; deep purple ecchymosis on R. side of palate, soft palate engorged with oozing of blood, uvula long (2½″), removal; death four days later. He had previously bled from nose, mouth and bowel. No autopsy, no family history.]

768. Froelich: Hémophilie articulaire. *Révue d'orthopédie.* Paris, 1904, 2e sér. Tome v. p. 289. [Pedigree No. 584.]

769. Froelich: Un cas d'arthrite hémophilique. *Revue médical de l'est.* Nancy, 1904, Tome xxxvi. p. 453. [Short report of paper in Bibl. No. 768.]

770. Geier, W. A.: K woprossu o hemofilii. *Meditsinskoye Obozrïeniye.* Moskva, 1904, Vol. lxi. p. 22. [Male aged 22, died of haemorrhage from gastric ulcer. Two brothers were bleeders and had swelling of joints. Uncle on maternal side died of bleeding from wounded finger. Mother died at 50 of bleeding from anus.]

771. Grant, Lachlan: On haemophilia and its treatment. *The Lancet.* London, 1904, Vol. ii. p. 1279. [Same case as in Bibl. No. 772.]

772. Grant, L.: On haemophilia and its treatment. *The Caledonian Medical Journal.* Glasgow, 1904—6, Vol. vi. p. 321. [Pedigree No. 490.]

773. Gutkin, Lea: Behandlung der Hämophilie. *Inaug. Diss.* Freiburg i. Br., 1904, 32 pp. 8°. [Three cases. (1) Boy 1½, bitten tongue, stitch arrested bleeding which however recurred some hours later. Application of clamp, which badly crushed the tongue and it became hard and swollen. Fever, but no further bleeding for six days when it recurred slightly. Ultimately foul sloughing of tip of tongue. Secondary haemorrhage, death. One sib was easily bruised. (2) Robert Muser, (see Burger, Bibl. No. 714). (3) Emil Spiess aged 29, haemorrhage after tooth extraction. Short reference to other bleedings.]

774. Klippel et Lhermitte: Lésions du sang au cours des grandes maladies hémorrhagipares. *Archives générales de médecine.* Paris, 1904, Tome i. p. 257. [Investigation of blood in a case of cutaneous eruption with a view to differentiating between infectious purpura and erythema multiforme.]

775. Monsarrat, Keith: Fracture of the femur in a haemophilic. *The British Journal of Children's Diseases.* London, 1904, Vol. i. p. 494. [Male aged 8, probably haemophilia but no family history.]

776. Naish, A. E.: A case of haemophilia with haemorrhage under the right iliac fossa. *Brit. Med. Journ.* London, 1904, Vol. i. p. 21. [Ten lines with reference to a male aged 17 with joint lesions and one brother who died of a minute cut on the lip.]

777. Pearson, H. B. A.: Notes of a case of haematuria due to the haemorrhagic diathesis. *The Lancet.* London, 1904, Vol. I. p. 91. [15 persons in four generations, dying of haemorrhage, the nature of which was usually not stated; one however was cerebral haemorrhage, another haematemesis, another morbus cordis and a fourth haematuria "evidently associated with some abnormal condition affecting the kidney or its hilum." See Pedigree No. 607.]

778. René Le Fort: Hémorrhagies internes post-opératoires chez des hémophiles latents: un cas d'odontorragie mortelle. *L'Écho médical du Nord.* Lille, 1904, Tome VIII. p. 26. [Three cases. (1) Female aged 45 with fibroid polyp of uterus. Supravaginal hysterectomy and removal of R. ovary; found ex sanguine the same evening, laparotomy disclosed 2 litres of blood in peritoneum; death. Her eight children had died of palatine bleeding. (2) Male aged 27, internal haemorrhage following radical cure of inguinal hernia. No previous personal or family history. (3) Male aged 45 died after tooth extraction and ligation of left carotid artery. No personal or family history.]

779. Wachenheim, F. L.: The hemorrhagic diseases and their allies in the light of modern pathology. *Medical News.* New York, 1904, Vol. LXXXIV. p. 114. [No cases.]

780. Weidmann, Rudolf: Beiträge zur Hämophilie. *Inaug. Diss.* Königsberg i. Pr. 1904, 50 pp. 1 ch. 8°. [Nine cases, seven of which are worthy of no further consideration. (1) "Gottlieb W. of R." aged 15. Swelling of joints (ankle) first occurred at 2; since then hardly ever free. In hospital with knees typical of first stage of haemarthrosis, ecchymoses and bruises, haematuria, epistaxis. Wounds usually bled for several days, but often, especially after a recent attack, there was no bleeding. A maternal uncle died at 18 of bleeding from the knee; he had the tendency similar to that of his nephew. (2) "Heinz B. of D." aged 20, no family history of haemorrhage. At 1 diarrhœa and oral sepsis; at 1½ a basin falling on his head produced a haematoma; at 4 he bit his lower lip through and sustained great haemorrhage; at 9 internal bleeding from kick by horse; at 10 injured mouth with a lead toy soldier and bled eight days. R. knee swelled whenever injured. As he got older tendency to bleeding less. A crushed finger-nail was removed without haemorrhage. Haematuria. In hospital with haemorrhage after tooth extraction. Later haemarthrosis and great bleeding from wound of thigh.]

781. Wiedemann: Ein Fall von Bluterkrankheit. *Deutsche militärärztliche Zeitschrift.* Berlin, 1904, Bd. XXXIII. S. 24. [A musketeer, age not given, bled easily in youth after tooth extraction and had epistaxis. In 1904 severe swelling of knee and discoloration of skin over the joint and over the calf. Later scorbutic affection of gums and extravasation of blood; discharged out of the service as an invalid. No history of haemophilia in the family.]

782. Sahli, H.: Ueber das Wesen der Hämophilie. *Ztschrift für klinische Med.* Berlin, 1904—5, Bd. LVI. S. 264. [Pedigrees Nos. 378, 380, 388.]

783. Ste Marie, P.: Contribution à l'étude de la genèse de l'hémophilie. *La Revue médicale du Canada.* Montreal, 1904—5, Tome VIII. p. 34. [Male aged 28 suffering from epistaxis and haemoptysis. His brother similarly affected. Mother had epistaxis.]

784. Blumenau, N.: Haemophilia on the basis of general tuberculosis. *Vrachebnaya Gazeta.* S.-Peterburg, 1905, Vol. XII. p. 437. [Boy aged 5 months. Father and a 2 years old brother (or sister) died of phthisis. The disease began with ecchymoses in coccygeal region and extended all over body. Autopsy showed generalised tuberculosis.]

785. De Bovis, R.: De l'hémophilie chez la femme. *La Sémaine méd.* Paris, 1905, Tome XXV. p. 421, [Extensive general account of haemorrhagic conditions in women.]

786. Frantz, Charles P.: The hemorrhagic diathesis. *The Medical Fortnightly.* St Louis, Mo., 1905, Vol. XXVIII. p. 444. [Two cases. (1) Male, white, 46, no family history, epistaxis after enteric fever. Small growth on superior turbinate bone considered not to be the cause of the bleeding. (2) Male, white, aged 55, of splendid physique, "gave a history of prolonged and profuse bleeding from the slightest cause." No spontaneous haemorrhage. Bilateral tonsilectomy for some chronic condition. Bleeding lasted 4½ hours. Father healthy. "Mother bled profusely from the slightest abrasion and there was ecchymosis from the merest semblance of a bruise. External bleeding always lasted for hours." Her legs were large and thick, and were often swollen and painful above the knee. Brother, not affected, died at 42. One sister living aged 60. Menorrhagia.]

787. Froelich: Abnorme Formen von Blutergelenken. *Zeitschrift für orthopaedische Chirurgie.* Stuttgart, 1905, Bd. XIV. S. 600. [Two cases not previously described. (1) Male aged 25 with "arthritis haemophilica tardiva." As a child he suffered from epistaxis, and knees and elbows suffered several times. In 1903 pain in R. knee which was cauterised by Paquelin's thermocautery. In 1904 it was resected by Froelich and there was considerable, although not excessive bleeding. A brother had similar symptoms; parents healthy. (2) Male aged 32, of a healthy family. Knee

swollen and regarded as tuberculous. Operation showed it was filled with blood. He was inoculated with tuberculin but did not react. Died of pneumonia 15 days later.]

788. Godfrey, J. M.: Report of a case of hemophilia treated by calcium chloride. *The Hahnemann Monthly.* Philadelphia, 1905, Vol. xl. p. 126. [Male aged 34, bleeding from two lacerations of scalp. Secondary haemorrhage. Mother suffered from epistaxis and prolonged menstruation. Of patient's 12 brothers seven had epistaxis and bled easily.]

789. Goodall, A.: A contribution to the histology and genealogy of haemophilia. *The Scottish Medical and Surgical Journal.* Edinburgh, 1905, Vol. xvi. p. 133. [Family of eight children with mention of parents and maternal grandparents. No tendency to haemorrhage except epistaxis, also a genealogical tree in four generations containing eight bleeders including two females with no data of value.]

790. Kinnicutt, F. P.: A contribution to hemophilia with special reference to the joint symptoms of the disease. *Medical Record.* New York, 1905, Vol. lxvii. p. 881. [Pedigree No. 494.]

791. Lossen, Hermann: Die Bluterfamilie Mampel b. Heidelberg. *Deutsche Ztschr. f. Chirurgie.* Leipzig, 1905, Bd. lxxvi. S. 1. [Pedigree No. 389.]

792. Momigliano, B.: Sopra un caso di ematoma interstiziale da emofilia. *Il Progresso médico.* Torino, 1905, Tom. iv. p. 27. [Child suffering from follicular angina and swelling of R. cheek. Incision into swelling revealed blood. Recovery.]

793. Morawitz, P.: Die Chemie der Blutgerinnung. *Ergebnisse der Physiologie, hrsg. von Asher und Spiro.* Wiesbaden, 1905, iv. Jahrgang, i. und ii. Abt. p. 307. [Exhaustive *résumé* of knowledge of coagulation of blood.]

794. Perthes: Lokale Verwendung defibrinierten Blutes zur Stillung der Blutung bei Hämophilie. *Münchener med. Wchnschrift.* München, 1905, Bd. lii. S. 481. [As in title, male bleeding from gums, no evidence of haemophilia. Brother died of haemorrhage from lower lip.]

795. Pritchard, Eric: A case of haemophilia hereditary in the male line. *Reports of the Society for the Study of disease in Children.* London, 1905, Vol. v. p. 150. [Female aged 6, epistaxis and tonsilitis. Slight reference to father and grandfather.]

796. Sattler, R.: Cases of hemophilia. *The Lancet-Clinic.* Cincinnati, 1905, n.s., Vol. liv. p. 269. [Male with bleeding into a Meibomian cyst after operation. His brother had haemorrhage after enucleation of eyeball.]

797. Weil, P. Émile: Étude du sang dans un cas d'hémophilie. *Comptes rendus de l'Academie des Sciences.* Paris, 1905, Tome cxli. p. 603. [Male aged 45, previously perfectly well and without family history of haemophilia. From childhood he bled from trivial causes. In hospital after extraction of three teeth; haemorrhage and great prostration. Arrest on the eleventh day. Study of his blood by Weil.]

798. Weil, P.-É.: L'hémophilie; pathogénie et sérothérapie. *La Presse médicale.* Paris, 1905, p. 673. [Same case as in No. 797, but with fuller account of his symptoms and the additional facts that at the age of 9 he bled for a day after the extraction of a tooth, and for two days at the age of 11 from the same cause. In 1903 a cut on the thumb bled for five days and necessitated the application of the actual cautery. He never had spontaneous bleedings. Long account of the state of his blood.]

799. Wilson, John J.: Haemophilia. *The Practitioner.* London, 1905, Vol. lxxv. p. 829. [General.]

800. Arnone, L.: Emorragia spontanea della gengiva in una emofiliaca. *La Stomatologia.* Milano, 1905—6, Tomo iv. p. 504. [Haemorrhage from gum in female aged 52.]

801. Sheehan, R. F.: A case of hemophilia. *Buffalo Med. Journ.* Buffalo, N.Y., 1905—6, Vol. lxi. p. 362. [Male aged 16, considerable bleeding after removal of two molars and fracture of alveolar process. No record of family history.]

802. Albers, F.: Ein Fall von Haemophilie. *Inaug. Diss.* Bonn, 1906. [Pedigree No. 418.]

803. Bauer, J.: Ein Beitrag zu den "haemorrhagischen Diathesen." *Archiv für Kinderheilkunde.* Stuttgart, 1906, Bd. xliv. S. 41. [Female child, purpura, death. "Some male members on the maternal side had haemophilia."]

804. Carter, William Wesley: The etiology of the haemorrhagic diathesis. *American Medicine.* Philadelphia, 1906, Vol. xi. p. 434. [Abstract of a general paper read before the society of alumni of Bellevue hospital. One case is quoted of a boy aged 9 who had the haemorrhagic diathesis developed as a result of toxaemia following pneumonia.]

805. Cohn, A.: Die Stauungshyperämie bei Blutern. *Berl. klin. Wchnschr.* Berlin, 1906, Bd. xliii. S. 735. [Male regarded for years as a bleeder, Bier's treatment.]

806. Denis, J.: Hémophilie légère avec albuminurie. *La Policlinique.* Bruxelles, 1906, Tome xv. p. 155. Also *Journal médical de Bruxelles.* 1906, Tome xi. p. 318. [Male aged 37, epistaxis since the age of 6, admitted for blood in stools.]

807. Gilman, T. L.: A case of haemophilia. *Dental Brief.* Philadelphia, 1906, Vol. xi. p. 373. [Male aged 28 who bled profusely after the removal of a cyst of upper jaw. He had always bled excessively from trivial injuries and had one attack of epistaxis. No family history.]

808. Griffith, Frederick: Hemorrhagic diathesis complicating surgical work. *International Clinics.* Philadelphia, 1906, 16 series, Vol. iii. p. 203. [Male aged 21 months, circumcision, some oozing for three weeks. No family history of bleeding.]

809. Günzler, E.: Die gerichtsärztliche Bedeutung der hämorrhagischen Diathese. *Aerztliche sachverständige Zeitung.* Berlin, 1906, Bd. xii. S. 199. [Account of forensic importance of haemophilia, no original cases.]

810. Hirschfeld, Hans: Ueber einen Fall schwerer hämorrhagischer Diathese mit Knochenmarkatrophie. *Folia haematologica.* Berlin, 1906, Bd. iii. S. 429. [As in title, male aged 21, not haemophilia.]

811. Larrabee, R. C.: Haemophilia in the newly born with report of a case. *Amer. Journ. of Med. Science.* Philad. and New York, 1906, n.s., Vol. cxxxi. p. 497. [Pedigree No. 524.]

812. Lezin, W. W.: K woprossu o krovotoczivosti. *Russkij Wratsch.* St Petersburg, 1906, Vol. v. p. 1641. [Two cases. (1) A watchman stabbed with a dagger bled to death in the 14th day. (2) Young healthy male bled to death from stab wound.]

813. Lindsay, J. C.: A case of haemophilia in the new born. *The Chicago Clinic.* Chicago, 1906, Vol. xix. p. 181. [Female infant with large occipital haematoma, death. No family history.]

814. Miles, Alexander: See Thomson, Alexis. Bibl. No. 822. [Pedigree No. 594.]

815. Morris, F.: Notes on a case of haemophilia. *The South African Medical Record.* Cape Town, 1906, Vol. iv. p. 152. [Pedigree No. 387.]

816. Muir, J.: Eight generations of haemophilia in South Africa. *The South African Medical Record.* Cape Town, 1906, Vol. iv. p. 300. [Pedigrees Nos. 581, 582.]

817. Pelissard et Bonhamou: Les injections d'eau de mer isotonique (plasma de Quinton) dans l'hémophilie des nouveau-nés. *Presse médicale.* Paris, 1906, T. xiv. p. 614. [As in title; infant a few days old with general haemorrhagic condition. No instance in family.]

818. Rudnicki, T. F.: Sluchaĭ hemofiliczeskavo stradania sustawow. *Praktícheskiy Vrach.* S.-Peterburg, 1906, Vol. v. p. 473. [Sex not stated; aged 15, alleged haemophilic joints. No history in family.]

819. Ryerson, E. W.: Joint manifestations in hemophilia. *The Journal of the American Medical Association.* Chicago, 1906, Vol. xlvi. p. 1927. [As in title, good general account, no new cases.]

820. Sheldon, John G.: The joint affections of haemophilia. *Medical Record.* New York, 1906, Vol. lxx. p. 654. [Three cases. (1) Female aged 6, swelling of knee, the result of a blow. An uncle "gave a plain history of being a bleeder." (2) Male aged 4 days, acute arthritis, death. (3) Male aged 12, epistaxis, arthritis of L. knee at 11, no history.]

821. Strubell: [Eine Bluterfamilie]. *München. med. Wochenschrift.* 1906, Bd. liii. S. 2553. [Same as Bibl. No. 842.]

822. Thomson, Alexis and Miles, Alex.: Haemophilia, in Thomson and Miles' Manual of Surgery. Edinburgh and London, 1906, 2nd edit. p. 266. [Pedigree No. 594.]

823. Vandamme, G.: Un cas d'hémophylie. *La Policlinique.* Bruxelles, 1906, Tome xv. p. 177. [Male aged 29, epistaxis all his life.]

824. De Walsche, Émile: Une famille d'hémophiliques. *La Clinique, organe officiel des hôpitaux de Bruxelles.* Bruxelles, 1906, Tome xx. p. 641. [Female aged 60 had always a tendency to bleed. Severe bleeding after operation for nasal polypus. Various members of family bled in various ways.]

825. Weil, P. Émile: Recherches cliniques et physiopathologiques sur l'hémophilie d'après six cas. *Bulletins et mémoires de la société médicale des hôpitaux de Paris.* 1906, 3 s. Tome xxiii. p. 1046. Also *La Tribune médicale.* Paris, 1906, n. s., Tome xxxviii. p. 678. [No new cases.]

826. Weil, P. Émile: Etude du sang chez les hémophiles. *Bulletins et mémoires de la société médicale des hôpitaux de Paris.* 1906, 3 s., Tome xxiii. p. 1001. [Weil's principal paper. Pedigrees Nos. 443, 444, 445, 446, 447.]

827. Bouffe de Saint Blaise, G.: Un cas d'hémophilie héréditaire, accidents hémophiliques de vaccination. *Revue pratique d'obstétrique et pædiatrie.* Paris, 1907, Tome xx. p. 38. [Female aged 25 who is described as having suffered from classical haemophilic haemorrhages after tooth extraction, bore a child marked with a few petechiae.]

828. Bramwell, Byrom: Haemophilia. *Clinical Studies—a Quarterly Journal of Clinical Medicine by Byrom Bramwell.* Edinburgh, 1907, Vol. v. p. 368. [Pedigree No. 517.]

829. Broca: Die Blutstillung bei den Hämophilen durch Injektion mit frischem tierischem Serum. *Medizinische Klinik.* Berlin, 1907, Bd. iii. S. 1445. [Pedigree No. 453.]

830. Broca: Hémostase chez les hémophiles. *Bulletins et mémoires de la société de chirurgie de Paris.* 1907, n. s., Tome xxxiii. p. 311. [Short note referring to an alleged haemophilic boy bleeding from the socket of a tooth; serum therapy.]

831. Carrière, G.: Hémophilie. *Congrès français de médecine, neuvième session.* Paris, 1907, Rapports p. 72. [Good general account of the disease.]

832. Chisolm, R. A.: Note on a case of fatal subcutaneous haemorrhage in a new-born child. *The Ophthalmoscope.* London, 1907, Vol. v. p. 708. [Haemorrhage into eyelid of female child 7 days old.]

833. Davidson, R. B.: Recurring epistaxis in a girl. *The Brit. Med. Journal.* London, 1907, Vol. i. p. 563. [No evidence of haemophilia.]

834. Groves, E. W. Hey: The surgical aspects of haemophilia. *The British Medical Journal.* London, 1907, Vol. i. p. 611. [Pedigrees Nos. 500, 502.]

835. Haynes, H. G. L.: The nature and treatment of haemophilia. *The Westminster Hospital Reports.* London, 1907, Vol. xv. p. 80. [General account, no cases.]

836. Julien, L.: Un cas d'hémophilie familiale; accidents mortels post-opératoires à longue échéance (hémorrhagies de la plaie opératoire, hématuries, epistaxis, hémorrhagies cutanées). *L'Echo médical du nord.* Lille, 1907, Tome xi. p. 162. [As in title with following addition; male aged 26, suppuration and gangrene of the wound.]

837. Labbé, M.: Pathogénie de l'hémophilie. *La Médecine moderne.* Paris, 1907, Tome xviii. p. 339. [General account, almost the same as in Bibl. No. 859.]

838. Michel, G. et L.: Arthrite hémophilique. *Revue médical de l'est.* Nancy, 1907, Tome xxxix. p. 249. [Man aged 33 when a soldier received injury on outer side of knee. Aspiration revealed serum. Later knee became painful and was proved to contain blood. Later pains in thighs and epistaxis. His mother, brothers and sisters were all subject to haemorrhages from trifling causes.]

839. Price, F. W.: A case of haemophilia. *The Westminster Hosp. Reports.* London, 1907, Vol. xv. p. 139. [Pedigree No. 556.]

840. Schreiner: Ein Fall von schwerer haemorrhagischer Diathese nach einem Trauma. *Monatsschrift für Unfallheilkunde.* Leipzig, 1907, Bd. xiv. S. 200. [Boy who fell on stones from a height of seven metres. Later he had pain in the back, vomiting, haemorrhages in skin of trunk and elbows, external haemophthalmos and blood swellings on both elbows. Recovery slow. Henoch's purpura.]

841. Scott, E. W. Kerr: Notes on a case of haemophilia neonatorum. *The Australasian Medical Gazette.* Sydney, 1907, Vol. xxvi. p. 178. [Male infant, subcutaneous haemorrhage over face 36 hours after birth. Melaena. No family history.]

842. Strubell: [Eine Bluterfamilie]. *Deutsche med. Wchnschr.* Leipzig and Berlin, 1907, Bd. xxxiii. S. 127. [Demonstration of a "bleeder" (haematuria). Author stated that the family could be traced back as far as 1761 and that the females were not only carriers of the disease but were also affected. No details.]

843. Swanton, A. J.: Haemophilia transmitted through the male. *The Lancet.* London, 1907, Vol. ii. p. 1385. [Five males and five females in three generations, suffering chiefly from epistaxis and swollen joints associated with headache, fever and profuse sweats. Cuts in some cases were difficult to treat.]

844. Toussaint, H.: De l'hémostase chez les hémophiliques. *Bulletins et mémoires de la societé de chirurgie, de Paris.* 1907, n.s. Tome xxxiii. p. 280. [Male adult, severe bleeding after radical cure of hernia, epistaxis. His five sibs were haemophilic. No details.]

845. Vandamme: Un cas d'hémophilie. *La Policlinique.* Bruxelles, 1907, Tome xvi. p. 83. [Short note on blood examination of case described in Bibl. No. 823.]

846. Vannierre, René Gaston: Contribution à l'étude de l'hémophilie articulaire. *Thèse de Nancy.* 1907, 58 pp. [No original cases.]

847. Weil, P. É.: L'hémostase chez les hémophiles. *Bulletins et mémoires de la société de chirurgie de Paris.* 1907, n.s. Tome xxxiii. p. 262. Also in *Revue pratique d'obstétrique et pædiatrie.* Paris, 1907, Tome xx. p. 65. [General account of blood in haemophilia, no new cases.]

848. Anon.: On Weil's new treatment of hemorrhage in hemophilia, purpura, and allied conditions. (Letter from Paris.) *The Bost. Med. and Surg. Journal.* Boston, 1907, Vol. clvii. p. 683. [Serum treatment.]

849. Allen, F. O.: Haemophilia treated by transfusion. *Annals of Surgery.* Philadelphia, 1908, Vol. xlviii. p. 625. [Boy aged 12, haemorrhage from the mouth.]

850. Bonzani, Guido: Sulle iniezioni di siero negli emofili operandi. *La Riforma medica.* Napoli, 1908, T. xxiv. p. 454. [History of a tailor aged 41. Removal of a small lipoma in the suprahyoid region, haemorrhage, injection of anti-diphtheria serum, recovery. A maternal aunt died of a haemorrhage which developed without apparent cause.]

851. Brittin, F. G. M.: A case of haemophilia in the newly born. *The Lancet.* London, 1908, Vol. ii. p. 158. [Sixth child, died 19 days after division of fraenum linguae. After the operation extensive areas of skin became black and ecchymosed.]

852. Broca: Traitement des hémorrhagies chez les hémophiles. *Revue générale de clinique et de thérapeutique* (*Journal des praticiens*). Paris, 1908, Tome xxii. p. 113. [Male aged 13, bled for three weeks, with intervals, from a small wart on his finger, alleged to have bled much since infancy. No family history, serum therapy.]

853. Broca: Tratamiento de las hemorragias en los hemofilos. *Boletín de la Asociación médica de Puerto-Rico.* San Juan, P.R. 1908, Vol. vi. p. 66. [Boy aged 13, who developed a small papilloma on the first phalanx of the R. index finger, as result of a bite. Sudden and severe haemorrhage, compression, recovery. Patient had a sister who was free of haemophilia "which proves nothing since the female sex is less disposed to haemophilia." No other data.]

854. Dahlgren, Karl: Om hämofili och kirurgiska ingrepp hos hämofila personer. *Hygiea.* Stockholm, 1908, Bd. lxx. S. 501. [Two families. In the first, a male, aged 40, died of some generalised septic condition and haemorrhage after an operation for appendicitis. From the age of 28 he had, on three occasions, haemorrhages or extravasation sufficiently accounted for. Three other persons also showed haemorrhagic symptoms. In the second family was also a case of appendicitis. Operation delayed owing to death of man in first family. Appendix gangrenous, death from septic bronchopneumonia. One or two instances of haemorrhage occurred in other members of the family.]

855. Esmein, C.: Note sur l'anatomie pathologique de l'hémophilie. *Arch. des maladies du cœur des vaisseaux et du sang.* Paris, 1908, Tome i. p. 532. [Male aged 16, attacked with epistaxis for the first time, death.]

856. Harris, J. R.: A case of haemophilia neonatorum. *The American Journ. of Clin. Med.* [*The Alkaloidal Clinic*]. Chicago, 1908, Vol. xv. p. 506. [Infant, death from haemorrhage following circumcision, no family history of haemophilia.]

857. Hecker, R.: Haemorrhagic affections, in Pfaundler and Schlossmann's Diseases of Children, Engl. trans. Phila. and Lond., 1908, Vol. ii. p. 169. [General account.]

858. Kingsford, B. H.: Case of acute intestinal obstruction in a haemophilic. *The British Med. Journal.* London, 1908, Vol. ii. p. 545. [Pedigree No. 462.]

859. Labbé, M.: L'hémophilie, pathogénie et traitement. *Revue de médecine,* Paris, 1908, Tome xxviii. p. 103. Same paper also in *Congrès français de médecine, XIX. session.* Paris, 1907. Rapports, p. 117. [Good general account.]

860. Leary, T.: The use of the fresh animal sera in hemorrhagic conditions. *The Boston Med. and Surg. Journal.* Boston, 1908, Vol. clix. p. 63. [Case of purpura simplex in a girl. No history.]

861. Lommel: Ueber Blutstillung mittels Serum bei Hämophilie. *Zentralbl. f. innere Medizin.* Leipzig, 1908, Bd. xxix. S. 677. [Pedigree No. 384.]

862. Mackay, M.: A case of the "haemorrhagic disease of infants" (Holt). *The Montreal Med. Journ.* 1908, Vol. xxxvii. p. 268. [Male infant, purpura neonatorum, no family history.]

863. Mellanby, John: The coagulation of blood. *The Journal of Physiology.* London, 1908, Vol. xxxviii. p. 28. [As in title.]

864. Morawitz, P. and Lossen, J.: Ueber Hämophilie. *Deutsches Archiv für klinische Medizin.* Leipzig, 1908, Bd. xciv. S. 110. [Observations on the blood of a bleeder. The authors conclude that haemophilia is due to a degenerative process in the blood cells and perhaps the tissue cells leading to a diminution in the content of some such body as thrombokinase. Pedigree No. 389.]

865. Mosonyi, A.: A bleeder family. *Budapesti orvosi ujság.* Buda Pest, 1908, Vol. vi. p. 305. [Pedigree No. 569.]

866. Nolf, P.: La nature et le traitement de l'hémophilie. *Le Scalpel et Liège médical.* Liège, 1908—9. Tome lxi. pp. 73, 85. [Three cases the subject of blood coagulation experiments: (1) five lines, male, aged 11, frequent haemorrhage from lips and gums, haematomata, severe bleeding from wound; (2) four lines, male 13, epistaxis, haematuria, ecchymoses, haemarthroses; (3) two lines, brother of preceding aged 12, similar symptoms. No family history of haemorrhage in any case.]

867. de Ponthière, L.: L'hémophilie. *Annales médico-chirurgicales.* Dour, 1908, Tome xvi. p. 70. [Two cases: (1) female, aged 20, haemorrhage following ablation of tonsil, no previous personal or family history; (2) a humorous account of haemorrhage following amputation of the leg of a "grand fort gaillard" aged 30.]

868. Power, D'Arcy: A clinical lecture on a case of haemophilia. *The Clinical Journal.* London, 1908, Vol. xxxii. p. 225. [Pedigree No. 568.]

869. Pratt, Joseph H.: Haemophilia in Osler-McCrae System of Medicine, London, 1908, Vol. iv. p. 717. [Good general account.]

870. Weil, P. Émile: Un cas d'hémophilie spontanée à type de grande hémophilie familiale. *Bull. et mém. soc. méd. des hôpitaux de Paris,* 1908, 3 s. Tome xxv. p. 371. [One case, "Armand L." aged 7 years. No family history of haemophilia. At 2 years he had ecchymoses on calves, elbows and more rarely on face and buttocks. They coincided with joint pains: unilateral haemarthroses, epistaxis. Melaena two or three times. Bleeding difficult to arrest after trivial injuries.]

871. Weil, P. Émile et Boyé: Histoire d'une famille d'hémophiles: la petite hémophilie familiale. *Bull. et mém. de la soc. med. des hôpitaux de Paris,* 1908, 3 s. Tome xxv. p. 377. [Pedigree No. 442.]

872. Weil, P. É. et Claude, O.: Histoire d'un grand hémophile traité pendant un an. *Congrès français de médecine.* Paris, 1908, Compt. rend. p. 22. [Second report of same case as in Bibl. No. 871.]

873. Weil, P. É.: Traitement de l'hémophilie par les injections de sérum sanguin frais. *Congrès français de médecine (neuvième session, Paris,* 1907). Paris, 1908, Compt. rend. p. 17. [General remarks, no data of new cases.]

874. Ziegel, H. F. L.: A fatal case of haemophilia neonatorum. *Archives of Pediatrics.* New York, 1908, Vol. xxv. p. 120. [As in title, haemorrhage from fraenum linguae, and also from prepuce.]

875. Althoff, Hugo: Tödliche Blutung aus den Nabelschnurgefässen bei einem 12-Tage alten Knaben einer Bluterfamilie *Münchener med. Wochenschrift.* München, 1909, S. 2115. [Fatal umbilical haemorrhage 12 days after birth. Father alleged to bleed easily from cuts. Mother had epistaxis and *post partum* haemorrhage. A daughter, aged 6, had epistaxis.]

876. Altstaedt, Ernst: Die Hämophilie im Lichte der genealogischen Forschung. *Inaug. Diss.* Rostock, 1909. [Pedigree No. 489.]

877. Baum, Ernst Wilhelm: Der Werth der Serumbehandlung bei Hämophilie auf Grund experimenteller und klinischer Untersuchungen. *Mittheilungen aus den Grenzgebieten der Medizin und Chirurgie.* Jena, 1909, Bd. xx. S. 1. [Pedigree No. 492.]

878. Baum: Ueber die Erfolge der Serumtherapie bei der Hämophilie. *Münchener med. Wochenschrift.* München, 1909, Bd. lvi. S. 834. [Short summary of author's paper, Bibl. No. 877.]

879. Berthier et Gaillard: Hématome des deux psoas chez un hémophilique; hémarthroses multiples, enterroragie grâve. *Journal des praticiens (Revue générale de clinique et de thérapeutique).* Paris 1909, Tome xxiii. p. 311. [Male, aged 24, haemophilic from infancy. Ecchymoses on buttocks. At 8 left elbow swollen for first time and subsequently on many occasions. At 12 arthritis of R. hip. At 16 great haemarthrosis of L. knee. Later haematoma of psoas. At 22 was attacked with severe pain and swelling in abdomen, after a cycle ride. While in bed he developed haemarthrosis of L. elbow. In the following year severe pain and tumour in R. flank, and subsequently haemorrhage from the bowel. No family history of haemophilia.]

880. Böhm, L.: Hämophilie und Menstruation. *Inaug. Diss.* Breslau, 1909. [Same cases as in Bibl. No. 885.]

881. Busse, W.: Die Behandlung von Gebärmutterblutungen mit Serum. *Zentralblatt für Gynäkologie.* Leipzig, 1909, Bd. xxxiii. S. 236. [Series of 10 cases illustrating uterine haemorrhages in cases not found to be due to local abnormalities. The author considers that "echte Hämophilie bei Frauen bisher nicht bekannt ist."]

882. J. H. C. (Campani): A case of haemophilia with anomalous family history. *Guy's Hospital Gazette.* London, 1909, Vol. xxiii. p 68. [Pedigree No. 519. In a private communication we learn that the article which is signed J. H. C. is by Campani.]

883. Denk, Wolfgang: und Hellmann, I.: Die Verwertung der Koagulationsbestimmung des Blutes in der Chirurgie. *Mittheilungen aus den Grenzgebieten der Medizin und Chirurgie.* Jena, 1909, Bd. xx. S. 218. [Reference to blood coagulation time in bleeders but no details of actual cases.]

884. Fildes, Paul: Haemophilia. *London Hospital Gazette.* London, 1909, Vol. xvi. pp. 48, 81. [General account and history of a case. Pedigree No. 427.]

885. Fränkel, L. und Böhm, L.: Genitalblutungen bei Hämophilie. *Monatsschrift für Geburtshilfe und Gynaekologie.* Berlin, 1909, Bd. xxx. S. 417. [A long paper affirming the existence of haemophila in the female. Seven original cases are cited of bleedings mostly of genital origin, but in five cases there was no family history of haemophilia. The remaining two cases which are regarded as instances of hereditary haemophilia are as follows: (1) girl of 14 with tendency to spontaneous bleedings, bled severely from the genitals on several occasions. Ultimate recovery. Her great-grandfather bled to death from a scythe wound on the foot. The grandmother had epistaxis and bled from trivial wounds. The mother had dysmenorrhoea and profuse menstruation. An only brother of patient was healthy. (2) girl, aged 15, suffering from epistaxis and irregular and profuse menses. Both the mother and father had epistaxis, likewise a sister of the patient. The authors tabulate cases which they consider to be haemophilia in the female. We cannot agree with their diagnosis.]

886. Gaiyoux: Un cas d'hémophilie grave chez l'enfant. *Montpellier médical.* Montpellier and Paris, 1909, Tome xxviii. p. 269. [Girl aged 5, with epistaxis and purpura; cured by antidiphtheritic serum. Mother also had purpura and epistaxis and in addition metrorrhagia.]

887. Gangani, Labindo: Sull' uso del siero come emostatico nelle emorragie degli emofiliaci. *Gazzetta degli ospedali e delle cliniche.* Milano, 1909, Tomo xxx. p. 753. [Child aged 3½ years, early showed himself to be a bruiser. As a result of a fall he developed an haematoma over the bridge of the nose. Incision, haemorrhage, injection of gelatine, and of serum. Recovery. Paternal grandmother and maternal grandfather "affected with haemophilia."

888. Jacob, F. H.: Unpublished case. [Pedigree No. 585.]

889. King, Parks M.: Report of two cases of haemophilia. *The Charlotte Medical Journal.* Charlotte, N.C., 1909, Vol. lx., July, p. 1. [Pedigrees Nos. 473, 475.]

890. Larochie, Guy et Vaucher, E.: L'hémophilie. *Le Progrès médical.* Paris, 1909, 3 s. Tome xxv. p. 265. [Good general account, no new cases.]

891. Løfberg, Otto: Kirurgiskt ingrepp på hemofil individ med dödlig utgång. *Hygiea, medicinsk och farmaceutisk Månadsskrift.* Stockholm, 1909, Bd. lxxi. (Följd ii. 9) S. 380. [Male aged 26, seized with severe abdominal pain and vomiting, laparotomy, great oozing of blood, death 36 hours later. He had suffered in childhood from epistaxis and bruises. Pains and swellings of knees, elbows and other joints had been frequent but slight. No history of haemophilia on either the paternal or maternal side. The patient's four brothers and sisters healthy.]

892. Lombardi, Antonio: Su di un caso non comune di emofilia con ripetute e protratte broncorragie. Patogenesi dell emofilia. *Il Tommasi.* Napoli, 1909. Anno iv. p. 202. [Male aged 48. Epistaxis, haemorrhage from the gums and once after tooth extraction. Malaria: swelling and ecchymosis of L. knee after riding at military exercise when 27 years of age. At 28 probable history of syphilis. Cough, haemoptysis, bronchorrhagia. Family history of epistaxis, metrorrhagia and haematemesis.]

893. M'Causland, J. E.: Haemophilia. *Dublin Journal of Medical Science.* Dublin, 1909, Vol. cxxviii. p. 164. [Pedigrees Nos. 589, 599.]

894. Maixner, Emerich (of Prag): Unpublished case—personal communication. 1909. [Pedigree No. 555.]

895. Masters, J. L.: Unpublished case. [Pedigree No. 541.]

896. Miles, E. Blommart: Unpublished case. 1909. [Pedigree No. 496.]

897. Milne, J. A.: Unpublished case. 1909. [Pedigree No. 449.]

898. Milner, Beverley: The surgical aspects of hemophilia with special reference to hemarthrosis. *The Canadian Practitioner and Medical Review.* Toronto, 1909, Vol. xxxiv, p. 150. [Pedigree No. 523.

899. Müller, J.: Ueber einen Fall von intensiver Darmblutungen bei einem Bluter. *Correspondenzblatt des Vereins deutscher Aerzte in Reichenberg und Umgebung.* Reichenberg, 1909, Bd. xxii. No. 6, S. 3. [Pedigree No. 563.]

900. Nettleship, E. Unpublished case. [Pedigree No. 561.]

901. Pincus, Ludwig: Weibliche Helden. *Centralblatt für Gynäkologie.* Leipzig, 1909, Bd. xxxiii. S. 625. [Short paper referring to a newspaper paragraph stating that the girls of the Canton Graubünden had foresworn matrimony on account of the increase of haemophilia. Inquiry by Pincus showed that this was incorrect and was the work of a joker. See pedigree No. 373.]

902. Rettger, L. J.: The coagulation of blood. *The American Journal of Physiology.* Boston, 1909, Vol. xxiv. p. 406. [Important study on blood coagulation.]

903. Thomson, John: Haemorrhages in new-born children. Allbutt-Rolleston's System of Medicine, 1909, Vol. v. p. 867. [General account.]

904. Thompson, Theodore: Unpublished case. 1909. [Pedigree No. 460.]

905. Trembur, F. Serumbehandlung bei Hämophilie. *Mittheilungen aus den Grenzgebieten der Medizin und Chirurgie.* Jena, 1909, Bd. xx. S. 815. [History of a girl aged 13. From her fifth year onwards repeated history of epistaxis, bleeding from the gums and ears, ecchymoses and large subcutaneous extravasations of blood. In the Clinic at Jena on four occasions. Treated with serum. Parents and eight sibs of patient healthy. Six sibs died young. No history of haemophilia in family.

906. Weber, F. Parkes: A case of haemophilia with fibrous thickening in the appendix region. *Transactions of the Medical Society of London.* 1909, Vol. xxxii. p. 338 [Pedigree No. 531.]

907. Wirth. K.: Die neueren innerlichen Blutstillungsmethoden vorzüglich bei Hämophilie mit besonderer Berücksichtigung der Verwendung von Serum. *Centralblatt für die Grenzgebiete der Medizin und Chirurgie.* Jena, 1909, Bd. xii. S. 217, 258. [Case of a boy aged 14 (p. 221), who bled easily from slight wounds and had epistaxis and gum bleeding. Treatment by serum. Twenty-three other cases of haemorrhages of different kinds.]

908. Wright, A. E.: Haemophilia, in Allbutt-Rolleston, System of Medicine. 1909, Vol. v. p. 918. [General account and Pedigrees Nos. 495, 497, 521.]

909. Oppenheimer, Carl: Reformen im medizinischen Publikationswesen. *Münchener med. Wochenschrift.* 1910, Bd. lvii. S. 27. [General discussion on reform in medical publications and plea for non-publication of useless and inaccessible material.]

910. Gettings, H. S.: Unpublished case. 1910. [Pedigree No. 603.]

911. Thomson, John: Unpublished case. 1910. [Pedigree No. 604.]

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ALPHABETICAL LIST OF NAMES, AND NUMBERS OF PEDIGREES.

Map of Tenna district.

DESCRIPTIONS OF PEDIGREE PLATES[1].

PLATE XXXIV. Fig. 373. *Thormann-Vieli-Grandidier-Hoessli Case.—The bleeders of Tenna.* The first mention, of this great family of bleeders, to be found in medical literature, is from the pen of Dr Thormann, and was published in 1837. The account refers to the case of Christian Buchli (pedigree V. 44), and in addition gives a short account of the leading features of the family disease as communicated to Thormann by the patient. Nine years later the editor of the French *Journal de médecine et de chirurgie pratiques* gave a short abstract of a communication which he had received from Dr Vieli—a physician who practised at Rhäzüns where stands on the Rhine the ancient castle of his family. The abstract, which was not given under Vieli's name, consisted of a general account of the disease as found in the village of Tenna and was illustrated by the history of two cases. It was not however until 1855 that the bleeder families of Tenna became well known. About this time Grandidier, the well-known compiler of haemophilic records, had been in communication with Vieli on the subject, although there is no evidence that they ever met. As a result of their correspondence Vieli undertook the further investigation of the matter. The value of Vieli's work is difficult to gauge, as it would appear that it became considerably distorted by the time it appeared in the two editions of Grandidier's monograph (1855, 1877). Apart from numerous uncorrected printer's errors in both editions we consider that the communication by letter between the two led to error. Vieli himself does not appear to have consulted the Church books, the information being probably supplied to him by the ministers, who alleged that prior to 1828 the records were in great disorder and useless. The information so obtained, together with a few cases personally observed by Vieli and other general information, was all collected in the first edition of Grandidier's monograph (1855). The second edition published twenty-two years later (1877) contains no further data. In 1877 Dr Anton Hoessli, then practising at Thusis (Canton Grisons), undertook a further research at the instigation of Professor Immermann of Basel. His work appears to have been carried out with the greatest care and accuracy for two years, but the publication, which constituted his Inaugural Dissertation for the degree of Doctor of Medicine in Basel, was deferred until 1885 when he had left Thusis. We gather from Dr Wickham Legg (see Bibl. No. 437, p. 306) that the work was completed in 1880, when he visited Hoessli in Thusis. Hoessli's account constitutes the foundation of our abstract. Although it differs from that of Vieli in many respects we consider that it is unquestionably more worthy of credence. Hoessli was unfortunate in the fact that he never saw any of the bleeders, all being dead before he took up his work, but he took great trouble to establish this fact as far as he was able. Hoessli puts it on record that contrary to the statements of Grandidier the Church books were, on the whole, well kept and legibly written, and he added numerous quotations to show that beyond recording the bare fact of death, or birth, the *cause* of death and other information were added.

The village of Tenna as described by Hoessli lies on the south-eastern slopes of Piz Riein in the Canton Graubünden and consisted of several widely separated groups of houses scattered over the meadow slopes. (See accompanying map.) Communication between these houses and the outside world must be established over broken, and in many places dangerous tracts. At the time that Hoessli wrote, there were no driving roads, the journey having to be made on foot, and a traveller would require four to six hours to reach Versam. Tenna is much exposed to the weather, but the combination of a long day's sun and a dry atmosphere renders the village a healthy one. Sanitation was primitive, but better than in many another village in the Canton. The population was on an average about 150, of healthy and sturdy stock, toiling for a simple but sufficient livelihood. Poverty was unknown. Of ailments, pleurisy, pneumonia, bronchitis and arthritis deformans were common; phthisis was rare. Hoessli differs entirely from Vieli over the frequence of acute rheumatism. Organic heart disease, he says, was never seen. Scurvy and purpura were unknown. As an index of health he commented on the fact that a year might often pass without a doctor being called. These facts are of considerable importance in light of the statements of W. Koch that haemophilia and scurvy are one and the same disease, for here we have to do with families healthy in other respects yet the subjects of a hereditary tendency to severe and fatal haemorrhages. Our pedigree is taken from the account of Hoessli, supplemented where possible from Vieli-Grandidier.

First generation. I. 1, Verena Bueler, by whom I. 2, had an illegitimate child. I. 2, Albrecht Walther, born about 1648, married in 1669, and died on March 3rd, 1684. According to the Church books "Herr Amman Albrecht Walther starb im 36 Jahre seines Alters, ist nit lang krank gewesen." I. 3, Ursula Bueler, died 1681. I. 4, Hans Gartmann, married 1694. This man was probably born before 1660, the date to which the Church books of Tenna go back. I. 5, Ursula Walther. With reference to this woman a doubt has arisen as to whether she is identical with II. 8, Hoessli thinks not, for two reasons, firstly because if she were she would only have been 16 at marriage, and secondly because it is highly probable that II. 8, died unmarried (*vide* II. 8).

[1] The *half-blacked* sex symbols followed by a query used throughout the pedigree plates indicate that, although the individuals may have been regarded as bleeders, the writers of the present paper consider that the evidence does not justify the diagnosis of haemophilia.

Second generation. II. 1, Barbla, illegitimate child of I. 1, and I. 2, born about 1668....., "das auf Befelch einer ehrsamen Obrigkeit getauft worden." The subsequent history of this child cannot be traced. II. 2, Samuel, born Nov. 10, 1670, died Jan. 24, 1671. II. 3, Barbla, born Jan. 12, 1672. II. 4, born dead 1675: sex not stated. II. 5, Marie Juon, of Safien, married about 1705, died young, Nov. 17, 1712. II. 6, Samuel Walther, husband of II. 5—a bleeder, born April 6, 1676, died May 8, 1741. The Church books record "Samuel Walther ist allhier zur Erde bestattet worden, den 8 Mai 1741, welcher ein frommer und ehrbarer Rathsherr gewesen, bei 33 Jahre lang, mit der zweiten Ehefrau 26 Jahr, 5 Monat 16 Tag gelebt, hat 7 Tag und Nächt stets im Munde geblutet, daran er gestorben, als er erlebt hat 65 Jahr und 2 Monat"—entered by Pfarrer Pedolin (of Splügen?). It is not known where Samuel Walther lived. Both his wives came from Safien. He died and, apparently, was buried at Splügen. No Walthers are mentioned in the Safien Church books. II. 7, Anna Gredig, married Nov. 21, 1713: a native of Safien "a valley in which haemophilia certainly did not exist." She was the second wife of Samuel Walther. II. 8, Ursula, born March 3, 1678, sister of II. 6 (*vide* I. 5). In the Church books of Safien there is mention of an Ursula Walther who died unmarried in 1757 at the age of 80. There are no other Walthers at Safien. This person is undoubtedly II. 8. II. 9, Anna, born Feb. 27, 1681, died Feb. 10, 1686. II. 10, Ursula Gartmann, born 1695. II. 11, Felix Gartmann, a bleeder, born 1697, died 1702. "Felix Gartmann starb 12 Juli 1702, 5 Jahr und 3 Monat alt, hat sich rasch zu Todt blutet"—entered by Pfarrer Johannes Doentz of Zuz (in Ober-Engadin on main road between Zernez and Ponte). II. 12, Albrecht Gartmann, a bleeder, born 1699, married 1726, died 1730. The Church books give "Albrecht Gartmann, des Hans Gartmann sel. ehelicher Sohn (geb. 11 Juni 1699) ist, nachdem das Blut alles von ihm geflossen, im Alter von 31 Jahre anno 1730 den 21 September gestorben"—entered by Pfarrer Pedolin, of Splügen (*vide* II. 6). II. 13, Magdalena Tester, a widow. The Church books are here incomplete, which accounts for the narrow link between these two and the present day. II. 14, Philip, twin with II. 15, Barbla Gartmann, born 1703. II. 16, Hans Bueler, married 1726 to II. 15. II. 17, Hans Gartmann, a bleeder "geb. 22 Juli 1705, ges. den 8 März 1711 nachdem das Blut ihm alles ausgelofen, ist es in Gott entschlafen"—entered by Pfarrer Martin Schucan. II. 18, Trina, born 1706, died 1773. II. 19, Anna, born 1713. II. 20, Margreth, born 1715. II. 21, Philipp, born 1717, died 1722.

Third generation. III. 1, Marie Walther, born Nov. 17, 1706, married 1730 to III. 2, Joos Gartmann. III. 3, Ursula Walther, born Sep. 31, 1709, died 1739, married in 1732 to III. 4, Joos Gartmann, clerk and churchwarden, died 1742. After 1740 there is no further mention of these two families. Hoessli, after thorough investigation, thinks they went and lived on Heinzenberg and thence spread over the whole Canton. III. 5, Albrecht, son of Samuel Walther, the bleeder, born April 18, 1714, died 1762. He was married in 1754 to III. 6, Ursula Buchli. Of the descendants of these two Hoessli found 11 still alive and free from any trace of haemophilia. III. 7, Michael Walther, born March 16, 1717, married III. 8, Ursula Oswald. She died without issue. III. 9, Samuel Walther, born June 6, 1718. III. 10, Christen (Christian) Walther, born July 26, 1719, married in 1749 to III. 11, Anna Sutter. III. 12, Barbla Walther, born April 27, 1721. III. 13, Anna Marie Walther, born Aug. 1, 1723, married to III. 14, Joos Buchli, clerk. She died without issue. III. 15, Margreth Walther, born May 31, 1725 or 1726 (both stated), married in 1753 to III. 16, Martin Weibel. III. 15, is the progenitor of one of Vieli's families. The descendants of III. 15 and III. 16 are not mentioned in the Church books at Tenna. The authorities are the Church records of Versam and information from living persons. Hoessli saw no reason to doubt its correctness, but he does not accept responsibility in any way for the descendants of V. 26, about whom he knows nothing. III. 17, Elsa Walther, born Jan. 21, 1727. III. 18. Felix Walther, born 1731, died 1732. III. 19, Hans Gartmann, born 1727, died 1729. III. 20, Hans Gartmann, born 1729, married 1765 to III. 21, Elsbeth Hunger. III. 22, Christian Bueler, born and died 1727. III. 23, Ursula Bueler, born 1727, died 1728. III. 24, Anna Weibel, died 1765, married 1757 as first wife of III. 25, Christian Bueler, born 1729, died 1789. He married secondly in 1768, Anna Bräm, died 1810. Hoessli calls attention to the inexact way in which Vieli recorded the names, as according to him this woman's name was Wilhelmina, a name unknown in the Church records. III. 27, Ursula Bueler, born 1732, died 1733. III. 28, Dorothea Bueler, born and died 1734. III. 29, Ursula Bueler, born 1738, married in 1758 to III. 30, Abraham Bueler, clerk. III. 31, Hans Bueler, born 1717, died 1722. There appears to be some mistake in this entry though it is difficult to attribute it to a printer's error. On Hoessli's chart, III. 31 is printed just under II. 21, the figures in each case are given as the same and are probably mixed up. The mother was not married until 1726, and at that time she was only 23.

Fourth generation. IV. 1, Samuel Gartmann, born Dec. 8, 1732. IV. 2, Matheus Gartmann, born Nov. 4, 1734, died July 14, 1736. IV. 3, Matheus Gartmann, born June 7, 1737. IV. 4, Joos Gartmann, born Aug. 21, 1742. IV. 5, Christina Gartmann, born May 4, 1734. IV. 6, Anna Marie Gartmann, born March 17, 1736. IV. 7, Samuel Gartmann, born May 15, 1738. IV. 8, Samuel Walther, born May 15, 1738, married to IV. 9, Cathrina Brehm. IV. 10, Hans Walther, born May 12, 1758, died 1758. IV. 11, Marie Walther, born Sep. 5, 1760. IV. 12, Samuel Walther, born Jan. 15, 1750, married in 1780 to IV. 13, Barbara Buchli. IV. 14, Ursula Walther, born Sep. 19, 1750, died 1754. IV. 15, Anna Barbla Walther, born Jan. 9, 1754, died 1754. IV. 16, Ursula Walther, born 1755, died 1756. IV. 17, Abraham Walther, born Oct. 15, 1757, died Nov. 29, 1841,

married IV. 18, Barbara Gartmann, in 1786. IV. 19, Ursula Walther, born May 6, 1760, died 1766. IV. 20, Anna Walther, born Apr. 18, 1763, died Nov. 11, 1765. IV. 21, Christine Weibel, born 1754, died 1757. IV. 22, Anna Marie Weibel, born 1755, married IV. 23, Georg Joos of Arāzen, in 1777. IV. 24, Christine Weibel, born 1757, married in 1780 to IV. 25, Philipp Gartmann. IV. 26, Margreth Weibel, born 1760, died 1761. IV. 27, Margreth Weibel, born 1762, died 1786, married IV. 28, Christian Gartmann. IV. 29, Hans Weibel, born 1767, died 1768. IV. 30, Alexander Gartmann, born 1766, married in 1790, IV. 31, Elsbeth Buchli. IV. 32, Hans Bueler, born 1758. IV. 33, Cathrina Bueler, born 1769, married in 1791 to IV. 34, Peter Buchli. IV. 35, Barbla Bueler, born 1773, married to IV. 36, Pfarrer Riz-à-porta, who was in office at Tenna for only a short time, towards the end of the 18th century. Hoessli makes some disparaging remarks as to how he departed from the standard of accuracy which up to his time had obtained in the Tenna Church books so that much of the information about this time is derived from Vieli and the recollection of persons then alive. IV. 37, Michael Buchli, married to IV. 38, Ursula Bueler, who was born in 1775. She afterwards married IV. 39, Christian Buchli, in 1811. IV. 40, Anna Bueler, born 1782, married in 1802 to IV. 41, Joannes Bueler. IV. 42, Hans Bueler, born and died 1759. IV. 43, Hans Bueler, born 1760, died 1780. IV. 44, Ursula, twin with IV, 45, Anna, both born and died 1762. IV. 46, Abraham Bueler, born and died 1764.

Fifth generation. V. 1, Cathrina Walther, born June 13, 1782, married in 1812 to Christian Walther, V. 12. V. 2, Magdalena Tester, married in 1822 as first wife of V. 3, Michel Walther, who was born on March 17, 1785. She died without issue. V. 3, married as second wife V. 4, Anna Pedrett, of Praz, in 1826 and she died without issue. V. 5, Ursula Walther, born Feb. 10, 1789—no information as to subsequent history. V. 6, Margreth Walther, born Jan. 25, 1782, died 1863, married in 1800 to V. 7, Daniel Hunger, but had no issue. V. 8, Anna Walther, twin with V. 9, born and died 1786. V. 10, Anna Prader, first wife of Christian Walther, V. 9, born 1789; she died without issue. He afterwards married Anna Gredig, V. 11, who also died without issue. V. 12, Christian Walther, born Jan. 6, 1788, married V. 1, Cathrina Walther. V. 13, Anna Walther, born 1790, married in 1810 to Abraham Bueler, Statthalter, V. 14. V. 15, Barbla Walther, born Oct. 27, 1793, married in 1815 to V. 38, Peter Buchli, a bleeder. V. 16, Elsa Walther, born 1797, married in 1816 to V. 17, Johann Buchli. V. 18, Georg Joos, a bleeder, born 1782, died 1790. According to Vieli he bled to death in early youth. V. 19, Georg Joos, born 1791, died 1831, a bleeder. He went to America with his wife, V. 20, Ursula Gartmann, of Safien, and family. V. 21, Martin Joos, a bleeder, born 1789, died 1797. V. 22, Margreth Joos, born 1778? married in Arāzen to Lorenz Engi, V. 23. V. 24, Daniel Gartmann, a bleeder, born 1781, died 1784. According to Vieli-Grandidier he bled to death in his 6th year. V. 25, Margreth Gartmann, born 1783, married V. 26, Pfarrer Zadriell in Sehnaus (near Ilanz). V. 27, Anna Gartmann, born 1786, married V. 28, Christian Joos, of Arāzen. V. 29, Daniel Gartmann, born 1796, died 1812, marked by Hoessli as a bleeder—not mentioned by Vieli. V. 30, Daniel Gartmann, born 1787, died 1809, a bleeder. According to Vieli he "bled to death from a small wound at the age of 22." V. 31, Martin Gartmann, born in 1789, a bleeder. Vieli was summoned to see this "Gartmann de Tenna" in December, 1844, as he was bleeding copiously from the mouth. Vieli described him (see Bibl. No. 164) as the richest man in the village ("le plus aisé"), Grandidier presumably mistook "aisé" for "ainé" as he describes him as "der älteste Mann in Tenna." In his second edition, however, he merely says he was not the oldest man but the oldest bleeder. We cannot doubt the date of birth, 1789, therefore, he would be 55 in 1844 when seen by Vieli and not 62 as given both by Vieli and Grandidier. The haemorrhage for which Vieli's services were requisitioned was the second attack and had commenced between the canine and incisor teeth. It had originally started with violent toothache, and a loose tooth had been removed. The flow of blood had been easily controlled by gum arabic and Haller's acid elixir, but at the end of eight days (Grandidier says two days) it started again and continued for four weeks, when Vieli arrived on the scene. He found him with a fair pulse and not looking very ill, in spite of the fact that he had lost a colossal amount of blood altogether. To prevent swallowing the blood he had been sitting in a chair most of the time and had only taken milk and water to assuage his intense thirst. Vieli proceeded to practise heroic measures, including compression of the carotid artery and the application of the "pompe à ventouses," both of which induced syncope and convulsions. Lapis infernalis, sulphuric acid and creosote were tried in turn, but without success. Vieli was not summoned again, but learned that Gartmann had died sixteen days after his visit, apparently, as he naïvely adds, "from lack of help." V. 31 was a tall man of vigorous constitution. He married V. 33. V. 32, Elsbeth Gartmann, born 1791, died 1797. V. 33, Elsbeth Gartmann, born 1797, wife of Martin Gartmann, V. 31, she died childless. V. 34, Hans Peter Gartmann, dead, married in 1826 to Elsbeth Hunger, V. 35. V. 36, Sabina Buchli, born 1792, married 1809 to Amman Joos Buchli, V. 37. V. 38, Peter Buchli, born 1793, a bleeder. According to Vieli he died of an enormous haematoma. V. 39, no name, a bleeder, the son of Pastor Riz-à-Porta and Barbla Bueler. According to Vieli he bled to death. V. 40, nothing known (Vieli), marked in Hoessli's pedigree as a female. V. 41, female, went to America with her husband, V. 42, a certain "Weissgerber Abyss of Chur," and was lost sight of. V. 43, Alexander Buchli, born 1800, died 1803, a bleeder; bled to death. V. 44, Christian Buchli, born 1803, married 1830, died in 1831. According to Hoessli this is the bleeder referred to by Thormann (see Bibl. Nos. 105 and 115) who describes him as 33 years of age, healthy and strong. In Jan. 1829 while in bed he suddenly developed a great swelling of the scrotum. Next day when seen

by Thormann, it was as large as his head and was hot, blue and fluctuating. Three days previously he had been kicked in the groin by a cow, but so slightly that he had felt no pain. Thormann says that Buchli, like all his male relatives, was given to bleeding seriously, even from slight scratches. After a trifling injury a blue lump would appear, which would then burst, and pour out blood till they nearly died. Females are not affected but pass it on. Three years after the haematocele, viz. in 1832, while a long way from home, V. 44 slightly injured himself near the knee joint. The bleeding was so bad for several days that he died before Thormann could visit him. Thormann appears to have attended other members of this family, although unfortunately he published no particulars. V. 44 married Christina Gredig, V. 45. V. 46, Hans Buchli, one of the last bleeders to die at Tenna, born 1812, died 1856 (in Hoessli's pedigree) (1862 in text). He is probably the "Geschworener Hans B." described in Grandidier. He was frequently treated by Vieli, and on one occasion for an enormous swelling of the right thigh, the result of a contusion. It contained thin sanious pus, and broken down blood clots when it burst spontaneously. It is related that V. 46, once cut his finger and had an enormous haemorrhage. In Grandidier it is stated that one of Hans B.'s brothers died at the age of 13 "nachdem Wassersucht der Blutung gefolgt war, ein anderer Bruder 38 Jahre alt an Verblutung." This remark may refer to Hans Buchli's two half brothers. V. 46, married Cathrina Walther, VI. 3. V. 47, Anna Buchli, born 1815, married Joos Hunger, of Tenna. V. 48, Anna Buchli, was well known to Hoessli, who frequently visited her, and told him about the family. She alleged that V. 46 was a bleeder, and that her mother was of a bleeder family. She was very anxious about her own boys, but rejoiced that "es hat ihnen allen nichts gethan." She was 66 years of age, and bent with arthritis deformans. Otherwise she appeared to be healthy, although Hoessli was unable to examine her. V. 49, Sabina Buchli, was weakly and died of albuminuria and dropsy soon after her first confinement (Grandidier). V. 50, Peter Bueler, born 1804, died 1808, a bleeder, bled to death early. V. 51, Anna Bueler, born 1815, was subject to epilepsy, married V. 52, Joannes Otto Ambühl, of Masein, in 1835, and went with him to America (Wisconsin). V. 53, Cillia Bueler (Cäcilia), married in 1836 to V. 54, Peter Feltscher, of Masein, Land-Ammann.

Sixth generation. VI. 1, Barbara Walther, born 1813, married 1835 to VI. 2, Michel Buchli, Ammann. VI. 3, Cathrina Walther, born 1820, married in 1840 to V. 46, Hans Buchli, a bleeder. She died in 1861. VI. 4, Barbara Bueler, born 1813; dead. VI. 5, Abraham Bueler, born 1816, married in 1841 to VI. 16, Barbara Buchli. VI. 6, Hans Bueler, born 1819, died 1831. VI. 7, Peter Buchli, same person as VI. 24, born 1816, married Anna Margreth Buchli, VI. 23. VI. 8, Johann Math. Feltscher, of Masein, married VI. 9, Barbara Buchli, born 1819. VI. 10, Cathrina Buchli, born 1823. VI. 11, Cathrina Buchli, born 1824, married VI. 12, Nicolas Gartmann, of Vallendas. VI. 13, Abraham Buchli, born 1829, married to Elzbeth Buchli, of Versam, VI. 14. VI. 15, Anna Buchli, born 1831, died 1835. VI. 16, Barbara Buchli. VI. 17, Peter Feltscher, of Masein, married in 1844 to VI. 18, Sabina Buchli. By him she had no issue. She subsequently married in 1858 Christian de Simon Mark, of Trans (Domleschg), VI. 19. VI. 20, Elsbeth Buchli, born and died 1823. VI. 21, Elsbeth Buchli, born 1825, married VI. 22, Christian Wieland, Ammann. VI. 23, Anna Margreth Buchli, born 1830, married in 1847 to Peter Buchli. VI. 24 (VI. 7). VI. 25, Johann Martin Joos. VI. 26, Martin Joos. VI. 27, George Joos. VI. 25, VI. 26, VI. 27 are all in America—no information. VI. 28, Anna Maria Engi. Her children are all scattered. Hoessli does not think that any of them had haemophilia. Most of them are alive (Vieli represents one, VII. 49, as a bleeder). She married VI. 29, Georg Engi. VI. 30, Barbara Engi, married VI. 31, Johann Martin Buchli, of Versam. With regard to her children the same remarks apply as to VI. 28. VI. 32, sex not stated by Hoessli—a male "bled to death" (Grandidier). VI. 33, sex not stated—a male according to Vieli; "still alive" (Grandidier). VI. 34, sex not stated by Hoessli; Grandidier (1st edit.) gives her as a female married in "Pitasch im Lugazerthal." In his 2nd edit. he calls it Cugazerthal—no doubt it refers to Pitasch in the Lugnetzthal, about 3½ miles south of Ilanz. VI. 35, not mentioned by name. VI. 36, VI. 37, VI. 38, sex not given by Hoessli. In Vieli-Grandidier they are given as females; "no information" (Grandidier). VI. 39, Philipp Joos, marked by Hoessli as a bleeder. According to Vieli there are two persons here, (1) a son who died of haemorrhage after being wounded by a stone; and (2) a son who had six daughters. Vieli also gives the "history of a bleeder in Arāzen" as follows. Philip J. attended by Vieli in 1854. He was 43 and had six daughters, and is living. This may be the second person mentioned above. His age would fit in well with that of his mother. But this man is alleged to be a bleeder, whereas No. 2 above is not. It cannot refer to No. 1 because he is said to be dead. There are thus points of similarity but they cannot be reconciled. As a matter of fact the history of "Philipp J." as given by Vieli is no evidence of haemophilia. He first showed signs of the disease at 10, but the first marked occasion was at 23. At 32 and 39 he had other attacks. They were as follows. He sustained a swelling of the whole leg from hip to ankle. The leg went black. He drinks vast quantities of cold water. He has no fear of bleeding or tendency to bleed. In the worst attacks he has bad headaches, this continues till a slight epistaxis heralds the crisis when he rapidly recovers. The veins of the right leg are varicose. There are no signs of scurvy (Grandidier). The account is far from lucid. VI. 39 was married to Anna Buchli, VI. 40. VI. 41, Maria Barbara Joos, died of *post partum* haemorrhage after the birth of her third child (Grandidier). She married VI. 42, Christian Joos, of Arāzen. VI. 43, Christine Joos, died in childbed (Grandidier) after having married VI. 44, Johann Peter Carisch, of Schnaus. He afterwards married VI. 45, his deceased wife's sister.

She is not mentioned by Hoessli. Grandidier says that after her first child she miscarried four times, with severe bleeding. In her next pregnancy a rich diet and ergot was ordered, when she bore a healthy child. VI. 46, Elsbeth Gartmann, born 1827, married to VI. 47, Hans Rageth Buchli. This couple had no issue. VI. 48, Maria Gartmann, born 1828, married VI. 49, Christian Hunger. VI. 50 (VI. 69), Anna Gartmann, born 1829. VI. 51, Alexander Gartmann, born 1832, died 1835. VI. 52, Hans Peter Gartmann, born 1833, married VI. 53, Anna Christina Hunger. VI. 54 (VI. 71), Sabina Gartmann, dead. VI. 55, Alexander Gartmann, married VI. 56, Christina Hunger. VI. 57, Valentin Gartmann, dead. VI. 58, Valentin Gartmann, married VI. 59, Ursula Buchli. VI. 60, Joos Buchli, a bleeder, born 1810, died 1837, shot himself ("died early of bleeding," Grandidier). VI. 61, Cathrina Buchli, born 1812, small, old and decrepit, lives with her brother, Michel, at Tenna. VI. 62, Peter Buchli, a bleeder, born 1815, died 1862; "bleeder living," Grandidier. He was apparently a typical bleeder, and although a strong man had countless slight or severe manifestations of the disease. Hoessli was not in a position to give clinical histories, but it was said on the country side that this family was the most seriously smitten up to that time. VI. 63, Anna Buchli, born 1817, married VI. 64, Dominic Veraguth, of Ilanz, but died without issue. VI. 65, Michel Buchli, born 1817. This is the man who is quoted as being the only male in the family who failed to show signs of the disease. He was 62, and was well known to Hoessli. He had never been ill. While his brothers were frequently fighting with death he was ever free, though occasions for severe bleeding frequently arose. His arms and hands were covered with scars, and latterly had three teeth removed at one sitting in Thusis. He is now somewhat decrepit and suffers from arthritis deformans. VI. 66, Hans Buchli, died 1857—a typical and severe bleeder, like his brother, Peter, VI. 62. VI. 67, Abraham Buchli, born 1822, dead: a severe bleeder like his brothers. VI. 68, Sabina Buchli, born 1827, died 1877. VI. 69 (VI. 50), Anna Gartmann, married in 1852 to Christian Buchli, VI. 70, born in 1831. After her death, VI. 70 married her sister, VI. 71 (VI. 54), Sabina Gartmann, in 1858. VI. 70 was President and "Civilstandsbeamter" of Tenna, healthy, not a bleeder. VI. 72, Valentin Hunger, born 1840, married VI. 73, Christine Barbara Jehli, of Arāzen, in 1865. VI. 74, Christian Hunger, born 1842, married Anna Maria Engi, of Arāzen, VI. 75. VI. 76, Jodokus Hunger, born 1844. He had a swelling of the knee (Grandidier). Hoessli says his knee was ankylosed; probably tuberculous. He married Marie Deuther, of Ilanz, in 1871. VI. 78, Johann Hunger, born 1846, married VI. 79, Sabina Sutter, of Arāzen. VI. 80, Alexander Hunger, married VI. 81, Barbara Blumer, of Schwanden, Glarus. VI. 82, Leonhardt Hunger, born 1851, seen by Hoessli. Like all his brothers he was tall and broad-shouldered. Was not a bleeder. He was excused from military service on account of myopia. VI. 84, Johann Peter Hunger, born 1854, married 1879. Thoroughly examined by Hoessli. He was never ill; no swellings of joints or tendency to bleed. He was a fine strong man—a soldier. He married VI. 85, Ottilie Hunger, of Hof, Safien (near Platz). VI. 86, Anna Ambühl. VI. 87, John Ambühl. VI. 88, Andreas Ambühl—in America: no information. VI. 89, Anna Feltscher, born 1837, known to Hoessli. Either she or VI. 94 was "ganz beschränkt" (a lunatic?). VI. 90, Christian Feltscher, born 1840, in Messina, Sicily, married VI. 91, Iphigenia Huber. VI. 92, Johannes Feltscher, born 1842, died 1857, was kicked on the jaw by a cow, and although the injury was a trivial one he bled to death. VI. 93, Peter Feltscher, born 1844, died 1874 Dr Buol attended this man who died after four or five days' uncontrollable bleeding from the bowel, for which there was no obvious cause—no autopsy. VI. 94, Maria Feltscher, born 1846, known to Hoessli (see VI. 89). VI. 95, Cäcilia Feltscher, born 1848, known to Hoessli. VI. 96, Martin Gartmann, married in 1876 to VI. 95.

Seventh generation. VII. 1, Ursula Zinsli, born 1837, married VII. 2, Wieland Buchli. VII. 3, Christian Buchli, born 1842. VII. 4, Cathrina Buchli, born 1839 (1849?), married in 1868 to VII. 5, Christian Buchli. VII. 6, Christian Buchli, born 1841, died 1862. VII. 7, Ursula Bueler, dead, married 1870 to VII. 8, Johannes Buchli, born 1847. He married secondly VII. 9, Fida Tester. VII. 10 (VII. 48), Alexander Buchli, born 1851, married VII. 11 (VII. 47) Barbara Buchli—stated here to have had two children, VIII. 10 and 11 (cf. VII. 48). VII. 12, Michel Buchli. VII. 13, Menga Feltscher, born 1847, married VII. 14, Andreas Decahausjāri, of Scharans. VII. 15, Barbara Feltcher, married VII. 16, Joh. Gartmann, of Masein. VII. 17, Peter Feltscher, born and died 1856. VII. 18, Peter Feltscher, born 1859. VII. 19, Barbara Gartmann, born and died 1846. The information about this Gartmann family was derived from Dr Christian Walther, of Ilanz. He knew them well and attended them. VII. 20, Martin Gartmann, born 1847, died 1873. From childhood he showed the typical swellings after trivial injuries. He cut his hand with an axe and bled, during intervals of three to four days, for three months, and died of lockjaw. VII. 21, Peter Gartmann, born 1848, died 1849. VII. 22, Anna Gartmann, born 1849, died 1850. VII. 23, Anna Barbara Gartmann, born 1851, died 1852. VII. 24, Anna Barbara Gartmann, born 1852, died 1874 of acute rheumatism. VII. 25, Peter Gartmann, born 1855. VII. 26, Alexander Gartmann, born 1857, died 1865, after eight days' haemorrhage from a slight wound in the popliteal space. VII. 27, Cathrina Gartmann, born and died 1860. VII. 28, Cathrina Gartmann, born 1861, died 1862. VII. 29, Cathrina Barbara Gartmann, born 1863. VII. 30, Abraham Gartmann, born 1865. VII. 31, Alexander Gartmann, born 1869. VII. 32, Joh. Valentin Buchli, born 1856. VII. 33, Peter Buchli, born 1860. VII. 34, Sabina Barbara Buchli, born 1868. VII. 35, Elsbeth Bueler, born 1842, died 1853. VII. 36, Joannes Bueler, born 1854. VII. 37, Abraham Bueler, born 1858, dead.

VII. 38, Abraham Bueler. VII. 39, Marie de Simon Mark, born 1859. VII 40, Elsbeth de Simon Mark, born 1860. VII. 41, Simeon de Simon Mark, born 1864. VII. 42, Elsbeth Wieland, born 1846, married in 1864 to VII. 43, Christian Joos, of Arāzen. VII. 44, Christian Wieland, born 1850, died 1874. VII. 45, Cathrina Wieland, born 1852. VII. 46, Johann Buchli, born 1851. VII. 47 (VII. 11), Barbara Buchli, born 1854, married in 1876 to VII. 48 (VII. 10), Alexander Buchli. They had no children. VII. 49, Lorenz Engi, a bleeder (Grandidier). VII. 50, Margreth Mani, wife of VII. 49. VII. 51, Anna Margreth Engi, married to VII. 52, Valentin Lötscher. According to Grandidier the marriage was childless. VII. 53, Ursula Engi, married VII. 54, Michel Oswald. VII. 55, Lorenz Buchli, dead. VII. 56, Lorenz Buchli, born 1830. VII. 57, Johann Martin Buchli, married Ursula Buchli, VII. 58. VII. 59, died of bleeding (Grandidier). VII. 63, Christina Barbara Joos. VII. 64, Marie Barbara Joos. VII. 65, Johann Nicolaus Joos. VII. 66, — Veraguth of Pitasch. VII. 67, Anna Joos, married to VII. 68, George Walther, of Vallendas. VII. 69, Margreth Joos. VII. 70, Ursula Joos. VII. 71, Ursula Joos, married to VII. 72, Joannes Jehli. VII. 73, Christian Joos. VII. 74, Anna Joos, married to VII. 75, Christian Gredig, of Versam. VII. 76, living still, a bleeder (Grandidier). VII. 77, bled to death in early childhood (Grandidier). VII. 78, 79, 80, 81, four miscarriages, according to Grandidier. VII. 82, a healthy child (Grandidier). VII. 83, Johann Peter Hunger. VII. 84, Joh. Peter Gartmann. VII. 85, Valentin Gartmann. VII. 86, A. Christina Gartmann. VII. 87, Anna Christina Gartmann. VII. 88, Elsbeth Gartmann. VII. 89, Joh. Peter Gartmann. VII. 90, Anna Gartmann. VII. 91, Maria Gartmann. VII. 92, Valentin Gartmann. VII. 93, Johann Peter Buchli, born 1853, dead. VII. 94, Anna Christina Buchli, born 1855. VII. 95, Elsbeth Buchli. VII. 96, Christian Buchli, not a bleeder. VII. 97, Joos Hunger, born 1867. VII. 98, Anna Hunger. VII. 99, Anna Hunger. VII. 100, Josias Hunger. VII. 101, Anna Hunger. VII. 102, Maria Hunger. VII. 103, Elsbeth Hunger. VII. 104, Josias Hunger. VII. 105, Johann Hunger. VII. 106, Anna Hunger. VII. 107, Anna Hunger, born 1880. VII. 108, no information. VII. 109, Cäcilia Gartmann.

Eighth generation. VIII. 1, Michel Buchli, born 1871. VIII. 2, Thomas Buchli, born 1873. VIII. 3, Wieland Buchli, born 1877. VIII. 4, Michel Buchli, born 1868. VIII. 5, Johann Buchli, born 1871. VIII. 6, Anna Barbara Buchli, born 1878. VIII. 7, Elsbeth Buchli. VIII. 8, Johann Buchli. VIII. 9, Marie Buchli. VIII. 10, Peter Buchli. VIII. 11, Cathrina Buchli. VIII. 12, boy. VIII. 13, Samuel Gartmann, born 1873. VIII. 14, Johann Mathias Gartmann, born 1875. VIII. 15, Barbara Dorothea Gartmann, born 1877. VIII. 16, Christian Joos. VIII. 17, Johannes Joos. VIII. 18, Georg Joos. VIII. 19—VIII. 24, healthy (Grandidier). VIII. 19, Anna Marie Engi. VIII. 20, Cathrina Engi. VIII. 21, Ursula Engi. VIII. 22, Georg Engi. VIII. 23, Daniel Engi. VIII. 24, Daniel Engi. VIII. 25—32, four dead, no bleeders. VIII. 33, Christian Buchli. VIII. 34, Lorenz Buchli. VIII. 35, Joh. Martin Buchli. VIII. 36, no information. VIII. 37, Cathrina Jeli. VIII. 38, Samuel Jeli. VIII. 39, Christian Jeli. VIII. 40, Georg Jeli. VIII. 41, Georg Gredig. VIII. 42, Marie Barbara Gredig. VIII. 43, Christian Gredig. VIII. 44, Johann Jakob Gredig.

To complete the history of the bleeders of Tenna we may also refer to several isolated instances of haemophilia in persons connected with the neighbourhood of Tenna.

Fig. 374. *Cantani's Case.* I. 1, living, aged 85, robust. I. 2, "haemophilic by descent from her mother's side," dead, aged 43. II. 1, premature. II. 2—5, "haemophilic." II. 9, Christian O., aged 39, born at Chur and resident at Naples, where he was under observation by Cantani. His first haemorrhage occurred from the gums at the age of 4, and up to the age of 6 or 7 he constantly became bruised or bled slightly from small blows received in the games by which "Swiss children harden themselves and foster their courage." At this latter age he bled after a normal tooth extraction for three weeks, the blood being finally arrested by digital compression maintained by his sister for 72 hours. At the age of 10, a similar haemorrhage was occasioned by the extraction of a loose tooth by the boy himself, and at 11, some slight haemorrhage occurred on removing a plaster from a cut finger. With the exception of two haemorrhages from tooth extraction, the patient remained free till the age of 28, when he was living in Naples. On this occasion he experienced an haematuria, spontaneous in onset, which lasted 30 days. This was followed a year later by haemorrhage from the bowels and again haematuria, and since his sister was then suffering from menorrhagia, they removed from their house on the Petraia to one in a lower part of Naples. He remained well till the age of 37, when, during a visit to Germany, he cut his finger. In spite of accurate bandaging, the haemorrhage persisted during the whole journey from Munich to Chur and from Chur to Naples. Cantani saw him in 1872 when he was 39, suffering from a wound on the occiput. III. 1, "haemophilic." III. 2, unaffected.

Though it is not possible to assign a place for this man in the Tenna family detailed above, there can be no doubt that he was a member of it. In connection with his name "Christian O." it may be noticed as suggestive that the name "Oswald" occurs twice in the main Tenna family (Pedigree No. 373), e.g. III. 8 and VII. 54.

Fig. 375. *Vieli's Case* 1. I. 1, Regierungsrath Janett. I. 2, Frau Janett. Her parents came from Sarn (on the slope of the Heinzenberg). According to Vieli (Grandidier, 1st edit. p. 27) she came

"aus einer der Bluterfamilien in Tenna." It is possible that she was descended from III. 1, 2, 3 or 4 (Pedigree No. 373), but Hoessli was unable to find any authority for this in the Church books at Sarn. II. 1, died at the age of 8 (Grandidier, 1st edit.), or 3 (2nd edit.), from haematuria. II. 2, died at the age of 10 from a slight wound of the gums occasioned by a fall. II. 3, living, no children. II. 4, living, healthy—classmate of Hoessli's at the gymnasium. Hoessli admits that II. 1, and II. 2, were bleeders.

Fig. 376. *Vieli's Case* 2. In Reams "einen hoch im Gebirge und nahe bei Tenna gelegenen Dorfe kennt Vieli eine Bluterfamilie deren Vater noch lebt; seine 4 Söhne verbluteten sich sämmtlich, der älteste wurde 20, der jüngste 10 Jahre" (Grandidier, 1st edit., p. 27).

Further remarks are added by Vieli of cases in Raffna (Roffna?). In this village lived a boy, Stephan P., aged 19, whose parents and relatives on both sides were healthy. At the age of 17, he was seen by Vieli as he was then suffering from epistaxis. In the intervals of these attacks he developed extravasations of blood. Later his joints were affected. The haemorrhages following phlebotomy and cupping were difficult to arrest. Reams is not, as stated by Grandidier, "nahe bei Tenna," but lies in the Oberhalbstein Thal on the road from Thusis to the Engadine. Roffna is a village in the same valley beyond Tinzen. Hoessli investigated these cases, but could find no evidence of their existence, the villagers and doctors having never heard of them. He remarks that they had no connection with the Tenna families as they were of an entirely different race and religion. Lastly we may refer to a case at Tenna described by Grandidier (1st edit. p. 23). After describing the history of Hans Buchli the bleeder, V. 46, he goes on to refer to Johann B. "aus derselben Familie zu Tenna, Soldat." We are unable to identify this person. He was attended at the age of 20 by Vieli. While marching during some manœuvres he suddenly sustained an enormously large and painful swelling of his R. thigh, showing a play of colours like a bruise. He had frequently had similar attacks, especially after getting wet, and they lasted about nine days. He was in the hospital at Chur last spring with one of these attacks.

A recent echo of the Tenna family appears (1909) under the unusual title *Weibliche Helden*, by Ludwig Pincus, whose contribution was the result of a newspaper paragraph circulating the statement that the maidens of the Canton Graubünden had forsworn marriage on account of the increase of haemophilia. Pincus caused inquiries to be made, and found that there was absolutely no foundation for the statement except in the mind of the writer of the paragraph. On the contrary, a communication was made by Dr Köhl, physician to the hospital in Chur, who, in 22 years' hospital practice there, knew of only two cases, one of whom died in the hands of the doctor, the other being still alive in the Domleschg. In Tenna itself for the last 30 years there have been no cases of haemophilia at all, the isolated examples above mentioned denying any connection with the Tenna families. (See Bibl. Nos. 105, 115, 164, 214, 325, 371, 493 and 901.)

Fig. 377. *Ripke's Case.* An admirable account, referred to once only in the literature, was published as a thesis at Freiburg i. B. by Ripke in 1889. The history is unique in containing a diary of the sufferings of a bleeder boy, from year to year, extracted out of the casebook of the author. The family, V. 21—32, was apparently Russian and very well to do, if not wealthy. They lived in Y., a dry fruitful district: hot in summer and often snowless in winter. Epidemics of the acute exanthemata, including even smallpox, were of almost yearly occurrence. Five bleeders only are described—three boys in one family, their maternal uncle and cousin. II. 1, 3, and 5, died in old age; all three were married and two had numerous children (III. 1 and 2). They are stated to have been healthy and free from haemophilia. II. 7, Reinhold W., of medium build, was in his youth healthy, but latterly a little feeble. He died at 82 of dropsy. His wife, II. 8, Agnes K., had four sisters. II. 9, suffered from haemorrhoids and died of dropsy. II. 11, also had haemorrhoids and some stomach trouble. She died in old age. II. 13, died at an advanced age of influenza. II. 14, at death, had also reached old age. She had rheumatism and an affection of the lungs. II. 19, represents the three sibs of Constantin X., II. 20. They died unmarried at an advanced age. II. 20, himself had rheumatism, morbus cordis and haemorrhoids. He died of dropsy in old age. His wife, Jenny B., II. 21, was apparently the first wife: she died after delivery. Her four step-brothers lived to old age. II. 32, Leo M., "Marine officier," was healthy, but died of a chill at the age of 35. Therese V., II. 33, his wife, the great grandmother through females of the bleeders in generation V., was healthy except for jaundice in her later years. There is no information about her menstruation. She died at 50 after a chill. III. 1, and 2, numerous children free from haemophilia. III. 3—6, died young of scarlet fever. III. 7, Veronica, menstruated from the age of 12 to 42. She was healthy but latterly rheumatic. While she was pregnant of IV. 61, she was much worried, and during the seventh month was jaundiced. The birth was easy. III. 8, Dorothea, menstruated from 13 to 40. After her first delivery she had pain in the abdomen. She was of a neurotic temperament and suffered from headaches. III. 9, Julie, had pneumonia frequently and suffered with her kidneys. These three sisters, it will be noticed, married three brothers. III. 10, Anna, had heart disease. She married III. 11, Rudolph J. III. 12, Raimund, suffered from asthma. He died at an advanced age of dropsy. III. 14, Julius. III. 19—22, died young. Ida, III. 23, died of haemorrhage, to which she was much addicted. Her husband, Ernst L., III. 18, married his deceased wife's sister,

Clara, III. 24. She was 35 years old and deformed. III. 25, Hugo, was corpulent, and died in old age of pneumonia. III. 26, also died well on in years of "inflammation of the liver." III. 27, Anton, was somewhat scrofulous as a boy, but became corpulent and rheumatic with an affection of the heart. He had bad haemorrhoids. III. 28, Victor, was stout and strong. As a boy he had epistaxis. Later he had an affection of the heart and was liable to faint. He died at 70 of pneumonia. III. 29, Carl, had syphilis with heart and kidney trouble. He died at an advanced age. III. 30, Moritz, a soldier, and later a "Beamter." In his youth he probably had syphilis, and later on in life he had chronic nephritis with cardiac failure. He died in 1883 at about 60 of uraemia. His wife, Eva M., III. 31, was the mother of the first bleeder, IV. 67. Though corpulent she was active and of a plethoric tendency. She had palpitations, rheumatism and haemorrhoids. Her menstruation started at 10 and recurred normally. She was married at 17 and, while carrying the bleeder, James, was much disturbed by a thunderstorm. III. 32, Harald, was weakly and troubled with his stomach. He died at 50. III. 34, Emma, though deformed, was healthy. She died unmarried at 66. Her sister, Bertha, III. 35, was also deformed and suffered from her liver. She died at 48. III. 37, Henrietta, was born of another mother. She had leucorrhoea and was married twice. III. 39, and 40, numerous children. III. 41, was feeble minded, the result of a fright in childhood. IV. 1, as a child was scrofulous. She had neurotic pains in the back and died of diarrhoea. Her menses appeared late. IV. 2, was scrofulous as a child and had eczema. She was chlorotic and menses appeared late. IV. 4, was also of a scrofulous disposition. She had pains in her abdomen during menstruation, which was late in onset, and nervous dyspepsia and headache. IV. 5, suffered from epistaxis and a tendency to faint—menstruation late. IV. 6, had some heart trouble. IV. 8, died of phthisis. IV. 10, was scrofulous, and had a chronic disease of the bladder and uterus. IV. 15, suffered from stone. IV. 16, had chest trouble and haemorrhoids. IV. 18, was weakly and troubled with his chest. IV. 19, healthy. IV. 21, chlorotic. IV. 22, deformed. IV. 23, died young. IV. 24, and 25, were both stout. IV. 26, had chest trouble and haemoptysis. IV. 32, weakly and affected with morbus cordis. IV. 34, healthy. IV. 36—50, all fairly normal. IV. 38, was deaf and scrofulous, with eruptions and ulcers on the ears. She married her uncle (from the context, her paternal uncle). IV. 39, was liable to fainting and suppression of the menses. IV. 40, died of paralysis, the result of a wound obtained in war. IV. 44, was for a short time mentally unhinged after the death of his child and wife in childbed. IV. 46, suffered from his liver and committed suicide at the age of 20. IV. 51, had abdominal pains. IV. 52, was aged 34 at the time of her marriage. IV. 54, at the age of 10 had spinal disease. IV. 61, died at the age of 2 of croup IV. 62, Johann X., was born in 1844 and was healthy, with the exception of haemorrhoids, since the age of 20, and gout. He was a cousin of his wife Martha, IV. 64, who was born in 1850. This woman became extremely corpulent (nearly 20 stone). About puberty she was attacked with epistaxis: later, she complained that she bruised very easily, but the author never chanced to see this. Menses first appeared at the age of 11. In 1868, when scarcely 18, she was married, and bled for some days after defloration. In 1869 she aborted without complications. She never suffered from metrorrhagia, but menstruated while suckling V. 21, and 22. At the birth of V. 28, the amount of blood lost was abnormal. With advancing corpulence she became extremely neurotic, and was continually in the hands of gynaecological specialists for some chronic condition of the uterus. "Heart attacks," dyspnoea, and similar manifestations, convinced her of the presence of organic disease, which, however, was not discovered in spite of examinations by numerous practitioners. IV. 63, Emilie, died at the age of 4 of "scrofula." IV. 65, Friederike, born 1854, was pale and thin. In her youth she had scrofulous eczema and otorrhoea. Her menses were irregular: she married in 1885. IV. 66, was healthy and not related to his wife. IV. 67, James, a bleeder, was born in 1855, and died in 1874. At his birth, though no injury occurred, there was severe umbilical haemorrhage. His teeth appeared normally, but early became carious. At the age of 3 he had his first joint swelling. This recurred from time to time, sometimes with pain and sometimes without. Purpuric spots and bruises appeared spontaneously or as the result of slight knocks. Haemorrhages would occur internally or externally, and were very severe after injury. His chief bleedings were from the nose, ear and gums. The subcutaneous injection of morphia produced no untoward symptoms. His death followed an operation for a tumour on the face. There was no autopsy. IV. 68, Georg, born 1858, was scrofulous as a child but showed no haemophilic symptoms. IV. 69, Mignon, was born in 1861. As a child she had eczema, otorrhoea and ulcers on the arm. At puberty epistaxis occurred. Menstruation, starting at 17, was profuse and painful. IV. 70, Hedwig, born 1867, was scrofulous in her youth. Menstruation started at 12—13 and latterly became profuse. IV. 71, Erich, died at 1½ years of wasting. IV. 73, died in war. IV. 74, was nervous and scrofulous. V. 1, had epistaxis, and V. 2, scrofulous eruptions. V. 4, and 5, were both scrofulous. V. 6, 7, 8, 9, 11, 12, 13, 14, were normal. V. 17, died at birth. V. 18, normal. V. 19, died at the age of 1 month of a scrofulous ulcer. V. 20, normal. V. 21, Gertrude, was healthy at birth (1869). In infancy she had a naevus on the knee. At the age of 11, she had epistaxis of no importance. At 12 she first showed signs of chronic bronchitis and otitis media. Menses appeared first at 13 and for three years were at times irregular. Slight epistaxis and otorrhoea continued, but had stopped at the age of 20. V. 22, Adelheid, was born in 1870 and was suckled eight months. She was unhealthy and scrofulous. She had a tendency to haemorrhages

difficult to arrest. Up to the age of 19, when the account stops, she had had for years swelling of the knees, especially the left, which was in a condition of hydrops; glands also were swollen, and catarrh was noticed at the apices of the lungs. Her toes were all "hammered." Menstruation started at 13, and was irregular and profuse. She is described finally as being healthy and suffering from chilblains. V. 23, Conrad, was born in 1872. He was suckled 5 months and was quite healthy. V. 24, Eduard, born in 1873, was the first bleeder in this family. At the age of 8 months he had his first serious haemorrhage from a small ulcer on the ear. He chiefly bled from the nose and gums, and died at the age of 3 of epistaxis following a fall. His teeth early became carious. He was never without bruises and purpuric spots. He had haemorrhages internal and external. Joint lesions occurred with or without pain. His glands were swollen. V. 25, Wilhelmine, born 1874, had in infancy a small naevus on the forehead. She was healthy. V. 26, Ludwig, born 1876, a bleeder, died of internal haemorrhage at the age of 3. The account of this boy shows symptoms identical to those of V. 24. V. 27, Robert, born 1878, a bleeder, was under the personal care of the author from 1880, when he was nearly 2 years old, to 1886, and for 3 years after, he obtained information from other sources. The author states that he himself lived over three miles away, and was certainly not called for every triviality. Robert's tendency to bleeding did not stand at a constant level, but fluctuated above and below an average. The description is given in the shape of extracts from the author's notebooks, and this method may be followed here in outline to show the almost continual state of wretchedness to which these children are reduced. 11/5/1880. Was called to Robert, aged 1 year and 10 months, for epistaxis. Desired to plug the anterior and posterior nares, but was resisted by the parents, who said the haemorrhage invariably lasted some days. Robert lay absolutely quiet, as if he recognised the danger of his condition. The blood slowly dropped from clots about his nares. The haemorrhage stopped spontaneously in 5 days. 13/6/80. Robert is a bright strong boy, though spare. His skin is thin and transparent. Purpura had been noticed within a few weeks of birth. Cuts or knocks are immediately followed by uncontrollable haemorrhage; there is no "latent period." His joints are not affected. 17/6/80. He has slight epistaxis, and is covered with haematomata and bruises. 13/12/80. Slight seborrhoea of eyelids and enlarged glands. 22/12/80. Bronchitis. 25/1/1881. Blood in stools. 7/3/81. Bronchitis. 19/3/81. Slight injury to lip, producing moderate bleeding, which soon stopped. 23/3/81. Vaccinated: normal course. 11/5/81. Injured his uvula with a stick. The bleeding was slight at first, and for 2 days was controlled by the parents' styptic, but then became uncontrollable. After cleaning out the mouth, the blood was found oozing from the palate at no particular place. It was arrested, but broke out again next day and continued all night. Blood was vomited and appeared in the stools; increasing anaemia and restlessness. Examination not permitted. 15/5/81. Great anaemia, but boy conscious. 16/5/81. Sudden cry—clonic spasms—syncope—pulse uncountable. 17/5/81. Fever—brain clearer—no sleep—bad dreams—gradual recovery. 20/5/81. Up. 20/6/81. Well. 26/6/81. Right ankle swollen—recovered in 2 weeks. 28/7/81. Submaxillary and cervical glands enlarged. 4/8/81. Right knee swollen. 8/8/81. Glands more swollen. Eczema around mouth. 16/9/81. Recovered. 25/11/81. Bronchitis. 24/12/81. Ankle swollen. 26/1/1882. Still swollen. 31/1/82. Recovered. 8/2/82. Otorrhoea. 18/2/82. Boy won't walk—parents think this a bad sign. This continued till 3/4/82 when knee swelled and became very painful. 7/4/82. Spontaneous hard haematoma the size of a hand on his right forearm. Ankle swollen and stiff. Very lively. 23/5/82. Slight scratch on cheek—slight bleeding—toothache. 3/6/82. Face swollen from tooth. 2/7/82. Knee and ankle never quite recovered since 3/4/82. Now worse: no crepitation. 30/8/82. Painful swelling of right wrist—no fever. 1/9/82. Right elbow swollen and very painful—tense and white. 8/10/82. Condition continued with remissions and exacerbations. 20/10/82. Almost well. 2/11/82. Toothache, and knee again swollen. 10/11/82. Right cheek much swollen—abscess discharged in connection with a tooth. Pus and blood washed out. 15/1/1883. Sleepless from pain in the knee—bronchitis. 28/1/83. Knee continued bad: recurrence of toothache. 1/4/83. Conjunctivitis. 20/4/83. Slight otorrhoea. 2/5/83. Right ankle white and swollen. 30/5/83. Great pain in unerupted molar. 26/7/83. Elbow and knee again involved. 31/8/83. Epistaxis. 2/9/83. Still continued. 5/10/83. Left knee swollen. 22/11/83. Attack of vomiting after eating a plum. No blood. 6/2/1884. All joints in healthy condition. 21/3/84. Pain in L. ankle. No swelling. 25/3/84. Swelling of L. foot and R. elbow—great pain. 2/4/84. Pain diminishing. 8/4/84. Unable to walk yet. 11/5/84. Well, but ankle still swollen. 12/5/84. Extreme pain in knee, calf, and foot. 16/6/84. Up for the first time. Joints still swollen. No crepitation. 6/7/84. R. hip painful. 20/7/84. Severe pain in R. knee and calf. 7/9/84. Very bad cramp-like pain in the whole left leg. 10/9/84. Continued. 15/10/84. Better, but still in bed. 1/11/84. Reduced in health, but better. 22/11/84. A slight cut of the ear from the barber's scissors bled very little. 29/11/84. Great pain in the right ankle. 1/12/84. Better. 6/12/84. Mild attack of chicken pox: normal course. 13/2/1885. Haematoma on the right buttock. No pain. 21/2/85. Getting smaller. 5/3/85. Typical "tumor albus" of right wrist. 8/3/85. L. forearm and hand swollen and bruised. 12/3/85. Slight pain in R. knee. No swelling. 1/4/85. Right knee caused him to limp. 15/4/85. Abscess and swelling connected with a molar. 17/4/85. Pus and considerable quantity of blood discharged. 23/5/85. Bleeding lasted 3 days. Loss of active

movement in R. leg; passive movements normal. 9/6/85. Condition unchanged. 18/8/85. Pains in right knee. No swelling. 11/9/85. Legs perfect—Robert very lively. 13/10/85. Few days ago, fell from sofa; now an haematoma of left biceps. 17/10/85. Small cut on finger did not bleed much. Vaccinated: normal course. 4/11/85. R. knee again swollen as usual. Tense and white. 2/1/1886. Up to now had limped. 10/2/86. Epistaxis after a fall, stopped by ice. 24/2/86. Left knee affected again. 19/3/86. Palpitation of heart. 20/3/86. Right ankle swollen and painful. 31/3/86. Pain in heart. 4/4/86. Left ankle swollen and stiff. Summer 1886. Fairly well with slight joint attacks. Nov. 1886. Severe joint attack. May—Nov. 1887. Continued as usual. 1888. One leg permanently affected. July 1888. Very bad joint attack. May 1889. On the whole less affected. V. 28, Christine was born in 1879 and lived only 6 months. She had eczema and died of meningitis. V. 29, Ferdinand, born in 1881, was brought up by a wet nurse; apart from eczema and glands, he was healthy and free from haemophilia. V. 30, Eugenie, born in 1885, also had a wet nurse. A few days after birth a number of telangiectases were found. At the age of 4 these had nearly all disappeared: she was quite healthy. V. 31, Christian, was born in 1886, and was quite unaffected, as also was the last child, a girl, Auguste, V. 32, born in 1888. V. 33, Amalie, aged 3, was quite healthy, but a little run down from travelling. V. 34, Erica, aged 2, was in a similar condition. Her mother said she frequently fell down and had epistaxis. V. 35, Paul, aged 5 months, was breastfed for some time and then by bottle. He was strong and healthy until an attack of diarrhoea, occurring while travelling, pulled him down. On the back of his hand was a spontaneous and painless haematoma, the size of a shilling. A similar one had appeared once before on his head, and previously on one occasion there was a little blood in his stools. (See Bibl. No. 548.)

Fig. 378. *Sahli's Case I: Loosli-Riser Family.* This is the history of a bleeder family living in Bern, one member, Hans Ruch, IV. 3, being Sahli's patient. On comparing our pedigree with Sahli's publication certain differences manifest themselves in generations IV. and V. These differences are the result of a correspondence with Professor Sahli, who has sent the corrections embodied in our pedigree. In Sahli's original paper, it will be found that IV. 4 is marked with a ?, from which is derived a generation of six daughters, two of whom had a single daughter, gen. VI. The six daughters on Sahli's generation V. should be sisters of Hans Ruch, IV. 3 and in addition to the two daughters in Sahli's sixth generation (our V. 7 and V. 8) we are able to add a normal family of 6, viz. V. 1—6, and a second family of 3, viz. one girl, V. 9, and two male bleeders, V. 10 and V. 11 discovered since Sahli's publication (letter from Prof. Sahli, Aug. 2, 1909). I. 1, Andreas Loosli, married to a distant relation, also Loosli by name, I. 2; both I. 1 and I. 2 were healthy, and died in old age. I. 3, Joh. Riser, died in old age. Concerning his wife I. 4, there is no information. II. 1 and 2 healthy, II. 3—8 healthy. It is not stated which were married. Their families, III. I, are however stated to be healthy. II. 9, Joh. Loosli, healthy, married to a distant relative, II. 10, Marie Riser, who also was healthy. II. 11 and 12, healthy. III. 3, Frau Elizabeth Ruch-Loosli, aged 61, healthy. She had two brothers and six sisters, the latter of whom are stated to have been healthy apart from excessive menstrual flow. One of the two brothers, III. 20, bled to death. III. 6, Marie Sommer-Loosli, aged 60. III. 8, Kath. Zaugg-Loosli, aged 56. III. 9, male aged 54, healthy. III. 12, Barb. Ruch-Loosli, healthy, bled to death during confinement, no details as to cause. III. 14, Rosa Nyffeler-Loosli. III. 16, Frau Tanner, married first to III. 15, and secondly to III. 17, Minder, by name. By each husband she bore one male bleeder. III. 18, Frau Mosimann-Loosli, died of tubercle at the age of 44. III. 20, male. He wounded his cheek slightly with a piece of elder-tube, and bled to death on the third day. III. 21, and 22, healthy. Of the six daughters of II. 9, and II. 10, four bore male bleeders (fourth generation). IV. 1 frequently suffered from epistaxis and subcutaneous haemorrhages, received a slight injury on the forehead and bled to death, being then two (7, in pedigree) years old. IV. 2, male, bleeder, had symptoms similar to those in his brother, IV. 1; at the age of 5 he injured the frenum linguae with a piece of candy sugar and bled to death. IV. 3, Sahli's patient, Karl Ruch. He began to manifest the signs of haemophilia at the age of two. These consisted in frequent and violent epistaxis coming on spontaneously, or as a result of very trifling injuries. Even eating and drinking would bring it on. This continued till he was 15, when it began to get rarer. Spontaneous painful swellings of the joints now made their appearance. For the relief of one of these a leech was applied and he bled from the bite for three or four days until he was in danger of his life. The bleeding ceased spontaneously, but only when he was exsanguine. On another occasion he nearly bled to death from a cut on the finger, and at the age of 12 after having a tooth extracted. On various occasions he had haematuria; five years ago a haemarthrosis left his elbow almost completely ankylosed in a right angled position. In May, 1904, his knee was involved and this crippled him completely. On the day of his admission he had to hobble on his crutches for some distance, the result being that his right ankle swelled. In the clinic, effusions were diagnosed in his joints; he also suffered from epistaxis, for which no reason could be assigned (rhinoscopic examination). He struck his elbow on the iron bedstead and sustained a large bloody extravasation at the site of injury. He also showed large subcutaneous haemorrhages coming on spontaneously. Sahli carried out interesting observations on his blood and demonstrated a great delay in the coagulation time. Even after his finger was pricked for a drop

of blood, the haemorrhage continued, and lasted as long as five hours. IV. 4, 6, 8, 10, 12, 13, six females all healthy. IV. 14, 15, 16, 17, four male bleeders, all died of haemorrhage when young, no details. IV. 18, healthy. IV. 20—23, four healthy females, ranging from 30 to 18 years. IV. 24—27, four healthy males, aged 22 to 15 years. IV. 30—42, four males and nine females all healthy. IV. 43, and IV. 44, two males, died of croup while young. IV. 45, and IV. 46, males, both very pronounced bleeders, aged 6 and 5 respectively. IV. 47, bled to death at the age of 3. IV. 48, bled to death at the age of 5. IV. 49, IV. 50, two male bleeders, no details. In Sahli's pedigree they are stated to be 71 and 81 years old respectively, but this is evidently a misprint. IV. 51—58, healthy. IV. 59, male bleeder, died of haemorrhage at the age of 5. IV. 60, healthy male, aged 20. IV. 61, and IV. 62, two healthy females, aged 18 and 14 respectively. IV. 63, bleeder, bled to death at the age of 4. IV. 64, healthy female, aged 4. IV. 65—68, four healthy females. V. 1—9, two boys and seven girls, healthy. V. 10, and V. 11, not in Sahli's pedigree, two male bleeders, both 3 years of age, no details. V. 12, and V. 13, two healthy females. V. 14, and 15, boys and girls, number not stated, healthy children of IV. 51, and IV. 52. (See Bibl. No. 782.)

Fig. 388. *Sahli's Case II. Lüdi-Maibach Family.* This contained nine bleeders in three generations. I. 1, healthy, married to I. 2, Frau Raymond-Raymond, who was an only daughter and was healthy. I. 1, and I. 2, had two healthy daughters, II. 2, and II. 4, and one son, II. 5, Aug. Raymond, who was a bleeder, and died in his 30th year. III. 1—III. 4, healthy. III. 6, Frau Lüdi, healthy, mother of Sahli's patient, IV. 13. III. 7, Raymond, a bleeder, bled to death, no details. III. 8, female, died after a confinement, no details. III. 10, male, healthy, now living in America, no other data. III. 14, healthy female, an only child, mother of Sahli's patient, August Maibach, IV. 27. IV. 1, and 2, healthy. IV. 3, and IV. 5, healthy. IV. 4, a bleeder, no details. IV. 6, died of pneumonia. IV. 11, healthy. IV. 12, a bleeder, was weak at birth and suffered from epistaxis and haemarthroses, bled to death at the age of 7. IV. 13, Alexis Lüdi, a bleeder. He early showed symptoms of haemophilia. Every tooth which made its appearance was associated with great haemorrhage. Injuries and extractions of teeth were succeeded by haemorrhage lasting three to four weeks. Profuse epistaxis was common, and from early life onwards he had frequent painful swellings in his joints, occurring either spontaneously, or after slight injuries. As he grew up the tendency to bleeding became less so that he was able to gain employment as a barber—a dangerous occupation for him. One day, when he was nineteen, he received a considerable cut while stropping a razor on his hand. Violent bleeding ensued, and many remedies were tried with a view to arrest it. After bleeding for 13 days he was conveyed (13/8/03) to the hospital, under Sahli's care. Blood was found trickling from a wound of his finger. The wound itself which was about 1 cm. long was covered with a clot the size of a hazel nut. Limitations of movements of the left knee and elbow were also noted. Blood counts showed 7400 leucocytes and 3,776,000 red corpuscles per cubic millimetre. Haemoglobin 60 %. There was also some haematuria. He remained eleven days in the clinic, the haemorrhage being arrested by gauze saturated with a 2 % solution of gelatine. IV. 15, male bleeder, aged 16—affected less severely than IV. 13. IV. 18—IV. 22, five healthy females. IV. 23, and IV. 24, two healthy males. IV. 25, and IV. 26, stated to have been bleeders; no details. IV. 27, Aug. Maibach, a bleeder. Since his 3—4 year he suffered from haemarthroses and large subcutaneous extravasations of blood, with discoloration of the skin. At 13 had a violent haemorrhage from a cut on his finger, and again two years later. He was always liable to considerable haemorrhage from slight injuries. From a tooth extraction he bled eight days, and had to be cauterised. Epistaxis was also common. In 1904, when 21 years of age, his right knee joint swelled up without ascertainable cause, and on June 6, 1904 he was admitted under Sahli, who found an effusion in the joint. On June 9th a blood count showed 4,800,000 red corpuscles and 3600 leucocytes with 45·2 % neutrophiles, 7·5 % eosinophiles, 2·7 % transitional forms, 44 % lymphocytes. Blood platelets numbered 170,000. There was a marked prolongation of the coagulation time. (See Bibl. No. 782.)

Fig. 380. *Sahli's Case III. Tschanz-Jakob Family.* I. 1, and I. 2, no information. II. 1, male, J. Jost, healthy. II. 2, alcoholic. II. 3, Frau Elizabeth Tschanz-Jost, healthy, died of apoplexy. II. 4—7, four sisters, of whom it is stated that two were childless, whereas nothing is known of the other two. III. 1, Gottfried Tschanz, healthy, unmarried, died of enteric fever at the age of 35. III. 2, Jakob, alcoholic. III. 3, Frau Jakob Tschanz, healthy. III. 4, Ferdinand Tschanz, died of intestinal haemorrhage, at the age of 25. In Sahli's pedigree he is figured as a bleeder, although it is stated that he showed no other symptoms of the disease. III. 5, Adolf Tschanz, a bleeder, showed the symptoms of the disease early, and was affected with epistaxis and haemarthroses. He bled to death from the bowel when 9 years of age. IV. 1, no information. IV. 2, male bleeder, aged 25, suffered from frequent epistaxis and joint swellings. IV. 3, and IV. 5, females, no information. IV. 7, A. Jakob, male bleeder, 24 years old. IV. 8, IV. 9, two healthy females. IV. 10, Fritz Jakob, Sahli's patient, now aged 17, a carpenter. Began to show symptoms of haemophilia when 11 months old, when repeated traumatic or spontaneous haemorrhages made their appearance on divers parts of his body. He also had haemarthroses, copious epistaxis, and haemorrhages from mouth and nose following whooping cough.

He bled severely from cuts. As a result of an injury he had haematuria. Since 1897 his R. knee has been affected, and he has been repeatedly admitted into the hospital with this joint lesion. On April 28, 1904, he fell and broke his thigh just above the knee joint. Great swelling set in and he was admitted into the clinic on May 10, 1904. The fracture was set, and he was able to leave on July 16, 1904. Sahli carried out a number of important observations on his blood, both during the period of anaemia following the injury and also after he had left the hospital. The first examination (22/5/04) showed haemoglobin 55%, red blood corpuscles 2,480,000; leucocytes 6700, of which 54·6% were neutrophiles, 2·4% eosinophiles, 42·4% lymphocytes. The second examination (16/8/04) showed neutrophiles 56%, eosinophiles 4%, lymphocytes 39·5%. The coagulation time was greatly prolonged. IV. 11, 12, 13, regarded by Sahli as bleeders. They all died of intestinal haemorrhage shortly after birth, no other details. V. 1—3, three normal females. V. 5, died of haematemesis shortly after birth, considered by Sahli to be a bleeder. (See Bibl. No. 782.)

Fig. 379. *Kurz's Case.* Forty-four year old barber affected with haemophilia since his earliest years; bleeding from gums, epistaxis. In the war of 1877 he was wounded, and after the removal of the bullet bled furiously for five days. Some years later bit his tongue and had copious haemorrhage for 12 days. Haematuria frequent. Of his 23 brothers and sisters (11 brothers, 12 sisters) only one brother was haemophilic. The sisters were healthy, but suffered from profuse menorrhagia; four brothers, it may he remarked, lost their lives on the battle field. Of 12 children of the patient only one, a daughter, is stated to be a bleeder. The grand-parents, parents, and other relations of the barber were not bleeders. Examination of the blood showed 7,000,000 red corpuscles per cub. mm., later when the bleeding stopped, it was even greater. The spleen was not enlarged. (See Bibl. Nos. 504 and 517.)

Fig. 381. *Gröschner's Case.* Weakly and indigent family living in Schlochau (West Preussen). Generation I. stated to have been unaffected. II. 2, not a bleeder, died of gangrene following a contused wound. II. 3, female, not known to Gröschner. She was said to have been ailing and insane, and died of inanition. II. 2, and II. 3, had a family of two girls and three boys. III. 1, girl, aged 27, healthy. III. 2, male bleeder, suffered with swellings of his joints and haemorrhages from early youth. He died of "exinanitio virium" following epistaxis at the age of 19. III. 3, aged 15, weakly and small for his age. From his first year great swellings of knees and elbows. These swellings were extremely painful, and recurred especially during spring and autumn. The region below the knee was livid, but no bruises were to be seen on other parts of his body. He also suffered from severe epistaxis. III. 4, female, aged 11, healthy. III. 5, male, died during teething when one year old. During life he sustained only one severe haemorrhage (site ?), but showed livid spots on his body. No other details. The account of Gröschner is of little permanent value. Indeed the diagnosis of haemophilia is doubtful. (See Bibl. No. 70.)

Fig. 382. *Weitz's Case.* Incomplete account of a case of fatal haemorrhage in a male, III. 3. Haemophilia alleged in several other members of the family. I. 1, and I. 2, healthy. I. 3, said to have been a bleeder, no details. II. 2, healthy. II. 3, wife of II. 2, healthy. II. 4, was a bleeder, no details. III. 1, and III. 2, two girls presumably healthy. III. 3, 4, 5, three males, all of whom bled easily and freely from slight injuries. III. 3, case described by Weitz, male, aged 26. On July 4th, 1899, received a contused wound one inch long over the R. parietal region. At first the bleeding was slight, but later on, severe and uncontrollable and hemiplegia set in. Compression was tried by applying a silver dollar over the seat of injury and keeping it in position by an elastic bandage. On July 6th, however, patient had to be conveyed to the hospital, where all local measures failed to stop the haemorrhage. Inflammation, gangrene, and sloughing set in, and he died on July 10th, six days after having received the injury; no autopsy. III. 4, aged 11, had alarming epistaxis and swollen knee joints, which resulted in deformity. He also showed spots on the skin "simulating peliosis rheumatica." III. 5, bled easily and freely from slight injuries. The case of III. 3, as described above, would not in the absence of other details justify the diagnosis of haemophilia, but we have considered him to be a bleeder from the history given of III. 4, and for the same reason III. 5 was also probably affected. Our abstract is from a communication of Dr A. W. Broyton, editor of the journal in which Weitz's case appeared. (See Bibl. No. 712.)

Fig. 383. *Woelky's Case.* History of two brothers, III. 2, and III. 3. I. 1—4, normal. I. 3, and I. 4, died about 40 of unknown cause. II. 2, robust man aged 48, never ill. II. 3, wife of II. 2, aged 53, slender, small and somewhat weakly woman, although she had never been ill. Her menstruation was not abnormal. She had eight brothers and sisters, of whom four, II. 12, were dead. II. 4, and II. 6, two healthy brothers, both married and both with healthy families, III. 4, and III. 5. II. 8, healthy, married but childless. II. 10, healthy, married and had five children, III. 6—10. III. 1, a bleeder, began to show the symptoms when 11 months old. He often bled profusely from slight injuries and suffered much from epistaxis. When 4 years and 8 months old he died, having bled for six weeks. III. 2, Ferdinand K., bleeder, aged 17. He was perfectly well up to the age of 3, when epistaxis set in and lasted three days. At 5 he had profuse bleeding from a cut on his R. hand; repeated attacks of epistaxis and bleeding from the gums. At 8½ he bled for 11 days from a loose tooth. Swellings of his joints and other parts of his body were also observed. With one attack of joint swelling of knee he was in the hospital in 1861. In 1867 he had swelling of left knee and arm, and

a colossal bleeding from his head. He also had a severe attack of haematuria. The tendency to bleed had however gradually become less. III. 3, aged 13. At one year of age severe epistaxis lasting for several days. At 3 swellings of elbows and fingers. At 5 a great haemorrhage from a falling tooth. In his eighth year he sustained a great swelling of his knee as the result of a fall, and was in the hospital for ten months. In 1867 frequent epistaxis. III. 6—8, three healthy girls. III. 9, and 10, two male bleeders affected as severely as III. 2, and III. 3, but no details given. (See Bibl. No. 302.)

Fig. 384. *Lommel's Case.* In 1908 Lommel published a case with short references to 4 other bleeders in the same family. II. 2, died of apoplexy. II. 4, and 5, were healthy. II. 6, was the youngest son. He is said to have had frequent joint bleedings and haematuria. He still suffered at the age of 65. III. 7, was a bleeder and the second son. He died of haemorrhage at the age of $7\frac{1}{2}$. III. 8, died young. III. 9, died young of a "blood swelling." III. 10, was the youngest son, a bleeder. He died at 26. IV. 1. In his first year he sustained ecchymoses on the slightest injury. At the age of 4 he slightly injured his hard palate with the mouthpiece of a toy trumpet. When Lommel saw him two days after, he was very anaemic, and pale blood was welling from a point the size of a pin's head. Under anaesthesia the thermocautery was applied and the haemorrhage stopped. Later, in the course of whooping cough, he had dangerous epistaxis. For more than one reason local applications were impossible, and therefore serum was given subcutaneously: 20 c.c. of antistreptococcic serum. The epistaxis stopped for nine days, but started again during a bad fit of coughing. Serum snuffled up the nose arrested it, and a subcutaneous injection was again required for recurrence. IV. 2, and 3, healthy. We consider this case, on the evidence submitted, somewhat doubtful. (See Bibl. No. 861.)

Fig. 385. *Winter's Case.* A short and incomplete account of a boy, IV. 1, aged 12, with fractured leg. History of haemophilia in other members of the family. II. 3, said to have been a bleeder and to have died before the marital age. No details. II. 2, and II. 4, healthy females. III. 2, not a bleeder. III. 3, died of cholera. III. 4, was a bleeder from the age of 6 onwards, and bled to death from an abscess which was incised. IV. 1, James S. in South Staffordshire Hospital with broken R. leg. Epistaxis. Five weeks after discharge he had a haematoma on left thigh. Along with his four brothers he had always bled easily and suffered repeatedly from epistaxis and swelling of joints. IV. 6—9, healthy. (See Bibl. No. 425.)

Fig. 386. *Gocht's Case.* After a general account of the disease, of interest especially from the point of view of joint lesions, Gocht describes two families, the first of which is identical with and adds nothing to the family of Hirsch (see Bibl. No. 628). The second family is represented in a pedigree as consisting of four generations, with a number of persons including one female, marked as bleeders. We reproduce this without comment. IV. 4, Hans M., born 1887 at B., suffered at the age of 3 from scarlet fever, which was followed by a sudden, severe haemarthrosis of the L. knee. He was very easily bruised and bled severely after slight wounds. Every month at night time he had attacks of restlessness, followed by swelling of his joints with pain and fever. The hand, foot, and knee were most affected. After two or three days the pain had gone but the swelling remained. At the age of 4 a subdural haemorrhage was diagnosed: it was associated with severe cerebral symptoms. The joint lesions continued. Next year he had an attack of severe pain in the abdomen with vomiting and fever. This lasted three weeks. At 6 he nearly succumbed to an injury and the joint lesions were severe. Four years later haematuria lasted for weeks. On examination, numerous bruises were found. The various joints are fully described. IV. 5, Edwin M., born 1891, bled easily from slight injury. At 2 his right knee became suddenly swollen, and on numerous occasions since: ankylosis and contracture present in the left knee. The attacks occurred 45 times in the right knee, which appeared more or less normal, but the disorganised left knee had been attacked only 39 times. In both boys the joint would become suddenly and enormously swollen within the space of ten minutes. Flexion might or might not be present. The same applied to pain. The joint might remain free or be held immobile. Photographs and X-rays of both boys are inserted. These two boys were demonstrated by Gocht a year later at Würzburg. (See Bibl. Nos. 698 and 720.)

Fig. 387. *Morris' Case.* Notes on a Jewish bleeder IV. 1, living in S. Africa, with history of haemophilia in generations I. and III. I. 1, stated to have been a bleeder, no details. Generation II., no data. III. 3, said to have been a bleeder, no data. IV. 1, Morris' patient, aged 13, healthy at birth, no excessive haemorrhage after circumcision; at 5 his left ankle became swollen and painful; at 8 he cut his finger and it bled for three weeks. He had always bruised very easily and without apparent cause, and had frequent epistaxis. When seen by Morris, he was a strongly built well nourished boy, but was nearly exsanguine from haemorrhage from the bowel. Bruises were present on his arms, legs and trunk, including one on the R. buttock (6 in. × 4 in.). Examination of bowel failed to disclose a bleeding point, black blood, however, "was seen oozing down from the sigmoid." (See Bibl. No. 815.)

PLATE XXXV. Fig. 389. *Chelius-Mutzenbecher-Lossen Case. The Mampel family.* This family, one of the finest in the history of haemophilia, has been under observation for 80 years. The first account is by Chelius, in 1827, followed by that of Mutzenbecher, in 1841, and by that of H. Lossen in

1877. In 1905, Prof. H. Lossen republished the available data, and brought the pedigree up to date and into the condition which we follow throughout. The family is first heard of in the latter part of the 18th century living at Kirchheim, a village near Heidelberg. In this village they have lived ever since, being represented on the chart as six generations, including some 250 persons of whom 37 were bleeders. The history opens with Chelius' account of G. M. Mampel, III. 1, who, at the age of 26, came to the surgical clinic of Heidelberg on Jan. 10, 1826, for advice respecting an haemorrhagic swelling of the hand, arm, and shoulder. Chelius also referred to the condition of a younger brother, probably III. 15 (*q.v.*), and another, III. 5 (?), who had bled to death. The sisters were not affected, but Chelius saw a boy, IV. 10 (?), with bruises on his arm and suffering from severe epistaxis. Fourteen years after the publication of Chelius appeared Mutzenbecher's thesis, in which he gave a very complete account of generations III. and IV. in part. In addition to Prof. H. Lossen's two papers Köster described the family in 1903, and Morawitz and J. Lossen investigated the blood of one of the members, VI. 70, in 1908. The originators of this haemophilic stock, as far as is known, were J. P. Mampel, II. 2, and Katharina Andreas, II. 3. In their ascendants no instance of the disease has been found. Of their 11 children three were bleeders, III. 1, III. 5, III. 15, while two daughters, III. 4 and III. 8, were the originators of two great haemophilic stocks. III. 4, had 13 children, of whom five boys were bleeders, and three girls, IV. 9, IV. 21, and IV. 24, transmitted the disease to seven of her grandchildren. One of her female grandchildren, V. 19, passed on the disease to her great-grandchild, VI. 17. III. 8, the originator of the second stock, bore 19 children, and of these eight boys were bleeders. Of the eight bleeders three married, IV. 26, IV. 34, IV. 46, but did not transmit the disease. Three of the girls also married, and two of them, IV. 36, and IV. 42, are known to have passed the taint on to their sons, V. 52, V. 72, V. 77, V. 79, and V. 86. IV. 36, had five married daughters, and three of these had haemophilic sons. Of the daughters of IV. 42, one alone married, and had two bleeder boys, VI. 80 and VI. 84. I. 3, Christoph Andreas, "a verger" at Bruchausen near Kirchheim, who married I. 4, Katharina Christ. Raab, of Eppstein, Pfalz. Enquiries from the priest and oldest inhabitants of Eppstein failed to elicit any information about them. Mutzenbecher says that J. P. Mampel affirmed that none of his family were affected, nor his wife's, so far as he knew. His brothers and sisters, and his wife's brothers and sisters, were likewise healthy and strong. It is particularly stated that they had no joint troubles. II. 2, Johann Peter Mampel (called Michael, in Lossen's first publication), was strong and healthy, though alcoholic. He married Katharina Andreas, II. 3, in 1798. Of her it is related by Mutzenbecher that she was gay and garrulous. She ascribed the peculiar diathesis of her son, Georg Michael, III. 1, to having been frightened by a soldier who had tried to rape her while she was suckling the boy. She fainted, and asserted that from that moment the boy lost his health, and showed bruises and haemorrhages. In Lossen's first paper, II. 2 and II. 3 are credited with one daughter less, but in his second he specifies six sons and five daughters. Mutzenbecher moreover gives the number of the children as eleven. III. 1, Georg Michael Mampel, 1798—1853, was seen by Chelius in 1826 when he was 27 years old. His parents stated that he first showed signs of haemophilia at the age of 9 months. When put to bed in the evening he would be quite well, but in the morning his mother might find him covered with bruises. Three times he had had severe epistaxis and had also suffered from bleeding from the gums. At the age of 8 he developed arthritis of the knees. From trivial injuries he bled copiously: on one occasion several towels were soaked with blood from a slight wound on his finger. He was a saddler by trade, and married in 1825. Ten weeks after his marriage (Jan. 10th, 1826) he came to the clinic in Heidelberg with a large extravasation of blood on his upper arm and shoulder. This had developed without any cause. His subsequent history is related by Mutzenbecher, who says he repeatedly came to the clinic, and that as he got older he enjoyed relatively good health. His haemorrhagic tendency had diminished so much at the age of 37 that he was able to apply leeches for the relief of his joint pains, and, contrary to his former experience, no great haemorrhage followed. At the age of 40 he developed piles, but this was considered by Mutzenbecher of no moment. He was a clever, intelligent man; blonde, with blue eyes and transparent skin. He died at 55, after begetting seven children, who were all free from the haemorrhagic taint. Philipp Jacob Teutsch, III. 3, was an ordinary man of phlegmatic temperament. His wife, Elizabeth Mampel, III. 4, was 40 at the time of Mutzenbecher's publication. She was a strong and wiry woman with black hair and grey eyes. Her complexion was dark and temperament phlegmatic. Her movements were slow and torpid, and it was difficult to carry on a conversation with her. She had never been seriously ill, and had shown no trace of an haemorrhagic tendency. Her menstruation was normal. III. 5, Johann Peter Mampel, 1802—1807, early showed symptoms of haemophilia. He is probably the boy referred to before as having been mentioned by Chelius. He fell on his face and received a slight cut, from which he bled to death with blood pouring from mouth and nose. Mutzenbecher says he died of epistaxis. Anna Catharina Mampel, III. 8, was born about 1811, and was the mother of a great haemophilic stock. Mutzenbecher describes her as being the exact opposite to her sister, Elizabeth, as a pleasant, jovial person with a simple mind. She was blonde with fair complexion; always healthy, and, though she had 19 children, all were easy births

uncomplicated by haemorrhage. Her menstruation was at first normal, but later she was compelled to remain in bed a week. She also exhibited the peculiarity of menstruating throughout pregnancy. Eight of her sons were affected with haemophilia. Her husband, Christoph Wendling, III. 9, was presumably normal. III. 10, 11, 12, all died in the first six months of life. III. 13, David Mampel is stated by Mutzenbecher to have been healthy and not a bleeder. III. 15, Adam Mampel, 1822—1837, was a severe bleeder. According to Chelius, he fell on a stone at the age of 5 and received a very trivial wound, of which he nearly bled to death. While young he was left a cripple by a traumatic arthritis of the left knee. At the age of 14½ he fell backwards while jumping over the trunk of a tree, and at the time complained of severe pain in his back and limbs. Swellings appeared on his chest and arm of the right side, and after great suffering he died on the fifth day. An autopsy revealed vast haemorrhage into the subcutaneous tissues and muscles, and the substance of the lung. The rest of the body was exsanguine. The blood vessels were normal. IV. 1—6, are stated to have been healthy, but IV. 7 died in infancy of a cause unknown. IV. 8, Joh. Georg Schneider, husband of IV. 9, Katharina Teutsch, and father of the bleeder V. 5. IV. 9, was described by Mutzenbecher as being 20 years old. A rustic girl with dark complexion and brown eyes. She never showed any tendency to haemorrhage or to abnormalities of menstruation. IV. 10, Peter Teutsch, 1821—1856, was a pronounced bleeder. Mutzenbecher describes him as of medium size, and suffering from severe arthritis and a tendency to bleed. Like his uncle, he was a saddler, and came frequently to the clinic to report himself. He bled copiously from trivial abrasions, and his joint affections ended in stiffness. As he grew older his haemorrhagic tendency decreased, but Lossen adds that while drunk he fell over a heap of stones and injured his cheek and nose, from which, after 8 days bleeding, he died. IV. 11, is probably the girl, Barbara, mentioned by Mutzenbecher, who died the day after her birth. IV. 12, Philipp Jacob Teutsch, was born in 1824, and is said in Lossen's two accounts to have died both in 1830 and 1836. In Lossen's second pedigree, however, he is stated to have died at the age of 6. He early showed signs of haemophilia. On the final occasion he bled for one week from the fauces, while a large hard tumour developed in his neck. At the autopsy this tumour was found to be a vast haematoma. IV. 13, who died, according to Lossen, in her first year, may well be the Lisetta Teutsch, mentioned by Mutzenbecher as having died in infancy. Again, in Mutzenbecher's account, we find a Stephan Teutsch, who died at the age of 11. He was not a bleeder, but had scarlet fever with subsequent alopecia, which took 12 months to disappear. He may be IV. 14. IV. 15, Georg Teutsch, 1830—1853, was a moderate case, his chief symptoms being joint pains and epistaxis. After drinking new wine, he had an attack of haematemesis and epistaxis, and died. Of IV. 16, Philip Teutsch I, nothing is known. IV. 17, his wife, was not named by Lossen. We suggest that she may be Ephrosyne Teutsch, stated by Mutzenbecher to have been aged 8 in 1841. She is otherwise not to be accounted for. IV. 18, Christoph Teutsch, was a severe case. He was born in 1833 and died in 1840. He had joint pains, and on one occasion developed a vast haematoma from a knock on the head. At the age of 7 he bled to death from an injury received in a fall. IV. 19, Carl Teutsch, died at the age of 11 weeks. Bruises were noticed early, but the cause of death is not stated. Both IV. 20, and IV. 21, Philip Koch, and Maria Katharina Teutsch, were unaffected. IV. 22, may be a second Stephan Teutsch, mentioned by Mutzenbecher. He also was free from the disease, and was alive and well in 1841. His hair was yellow and his eyes blue. IV. 23, is mentioned as Georg Teutsch in Lossen's second pedigree. IV. 24, is Barbara Teutsch. IV. 25, *née* Windisch, was presumably healthy. Philipp Wendling, 1830—1890, IV. 26, is described as a slight case. He was born before his mother's marriage, whether by her husband or not is not stated. As a boy he sometimes bled from the gums, and was dismissed from the army for haemoptysis. It is stated that his lungs did not appear quite healthy. However, after this occasion, he remained quite well with an occasional bleeding from the gums. IV. 27, is stated by Lossen to have died in infancy, and is probably the Peter Wendling stated by Mutzenbecher to have died three days after birth. IV. 28, Mutzenbecher speaks of Christiana Wendling, who died at the age of 1, and Margaret Wendling, another child, who died at 4. These two must occupy positions IV. 28 and IV. 31. The scanty information about IV. 29, and 30, is as follows. IV. 29, Johannes Wendling I, born 1833, died 10 days later. Bruises were noticed, but he did not die from bleeding. IV. 30, Christoph Wendling, born 1834, died 1837. He bled very much from a slight wound and died from quinsy not long after. IV. 31, *vide* IV. 28. There can be little doubt that the unnamed female, IV. 32, with the family of seven is the girl, Catharine Wendling, seen by Mutzenbecher at the age of 3, alive and well. The name of IV. 33 was Ludwig Clauer. IV. 34, Joh. Jacob Wendling, 1839—1873, was a bleeder. At the age of 2 he bled so much from an injury to his upper lip that Mutzenbecher was forced to make three applications of the actual cautery. Later his chief trouble was haematuria, which once gave rise to sudden anuria. On this occasion his bladder was washed out with iced water, and there was much haemorrhage. It is stated that the blood soon coagulated. He married in America in 1873, and died three weeks later of haemoptysis. His daughter, V. 51, is not mentioned in Lossen's pedigree. IV. 36, Elizabeth Wendling. IV. 37, Wilhelm Sickmüller. IV. 38, Johannes Wendling II, 1841—1848, while playing, injured his hard palate with a wooden cigar holder, and died

from haemorrhage lasting several days. IV. 39, Georg Michael Wendling, born 1843, bled to death at the age of 1½ after falling with his mouth against the edge of a small wooden door. The boy, IV. 41, died in infancy. IV. 42, Magdalene Wendling. IV. 43, her husband, Bernhard Rehm. IV. 45, Georg Wendling, 1853—1854, is described as a bleeder because he early exhibited small bruises. He died while "teething" at the age of 14 months. IV. 46, Carl Philipp Wendling, was born in 1854. Though stated not to bleed from the skin or into the internal cavities, he is described as a bleeder on the strength of having once bled from the gums, in 1876, and his complaints of dyspnoea and palpitations. Both IV. 48, and IV. 49, died before attaining the age of 12 months. IV. 50, in the text of Lossen's first publication a normal married son is mentioned in generation III. with one daughter. This can only be David Mampel, III. 13. In the pedigree, however, which accompanies this text, this daughter, IV. 50, is credited with the two children, V. 95 and V. 96. These last two children are omitted in Lossen's second publication, but included in ours. In generation V. we find 26 children dying in infancy. We dismiss them in the following list, death taking place *before* the date given. V. 1, birth: V. 2, 6 months: V. 9, 6 months: V. 10, birth: V. 11, 6 months: V. 12, 12 months: V. 13, birth: V. 14, 6 months: V. 15, 6 months: V. 16, birth: V. 17, 6 months: V. 32, birth: V. 34, 6 months: V. 35, 6 months: V. 38, 12 months: V. 41, 42, 43, 45, 6 months: V. 46, 12 months: V. 48, 6 months: V. 50, 12 months: V. 55, 6 months: V. 68, 6 months: V. 78, 12 months: V. 91, 6 months. V. 5, Philipp Schneider, born 1856, was married twice. He showed no haemophilic tendency until he was undergoing military service in the Dragoons. He then had a severe attack of haemoptysis while trotting. Lossen directs comparison between this man and IV. 26, who was also a "slight bleeder." V. 18, Peter Koch, born in 1861, bled immoderately after wounds; suffered from epistaxis and palpitations, and further vomited blood without apparent cause. Susanna Koch, V. 19, though in the position of a "conductor," married her bleeder cousin, Philipp Teutsch II, V. 25, in 1888, and had by him a bleeder son, VI. 17. Her husband, Philipp, was born in 1864, and was often affected with epistaxis. He once bled without cause from the mouth and suffered from palpitations. V. 21, Jacob Koch, born 1872, is another case of palpitation and epistaxis. V. 23, Ludwig Rostock, married into the family. V. 26, Philipp Jacob Teutsch II, born in 1866, bled once spontaneously from the mouth, and suffered from epistaxis. He seldom exhibited bruises. In 1891 he married Margaret Ziegler, V. 27. V. 29, is Martin Spiess. V. 30, Adam Teutsch, born 1872, married 1896. At the age of 7 he bled from two places in the hard palate without any obvious lesion being present. His brother, Johannes, V. 33, was born in 1877, and died at the age of 25 of pneumonia, complicated by excessive haemoptysis and gangrene of the lungs. At the age of 1, he bled from the rectum, probably as the consequence of scybala. The haemorrhage ceased spontaneously. Subcutaneous extravasations of blood were of frequent occurrence. At the age of 2 there were instances of epistaxis and bleeding from the lip. At 5 his knee became bruised and swollen as the result of a knock. V. 36, Georg Michael Wendling, not affected. V. 40, Michael Weiss. V. 52, Philip Sickmüller, born 1861, was a mild case of haemophilia. In his youth he was easily bruised and very prone to epistaxis. At the age of 19 he was kicked by a horse on the foot and sustained a large blood extravasation. The only data we have of the following are their names. V. 57, Philipp Kettenmann: V. 58, Ad. Kühni: V. 59, Eva Rosina Sickmüller: V. 60, Heinrich Klingmann: V. 61, Veronica Sickmüller: V. 62, Jacob Schweppler: V. 64, Georg Sickmüller: V. 66, Elizabeth Sickmüller: V. 67, Peter Jäger: V. 70, Adam Puttler. In V. 72, we have another bleeder, Bernhard Sickmüller, 1880—1888. At the age of 10 months he fell and bled badly from the upper lip. On another occasion from a cut finger. Subsequently he bled from time to time from the upper lip, the haemorrhage ceasing spontaneously. He died of haemorrhage. Judging from Morawitz and J. Lossen's paper this fatal haemorrhage may have been occasioned by tooth extraction, but their paper is not without discrepancies. V. 74, is Katharina Rehm, wife of V. 75, Georg Heinrich Sauter. Carl Philipp Rehm, V. 77, born 1873, is the youngest bleeder in Lossen's first paper. In early childhood he bled badly from a leech bite, and blood extravasations were caused by the mere rubbing of his trousers. At the age of 2 he bled badly from two small wounds in the hard palate. At 5, he injured the fraenum of the upper lip and bled with intervals for 12 days. At 7, his knee became swollen without any injury, and further, at the age of 31, we are told he suffered from haematuria. V. 79, Wilhelm Rehm II, born in 1877, had little opportunity to demonstrate his condition, although at the age of 1 year he bled from the gums. At 3 he was run over by an empty waggon. The wheels passed over his pelvis and thighs, but there was no fracture. When seen three days later he was blanched and pulseless. Vast extravasations extended all over his thighs, and both knee joints were distended with blood. He died the next day. His brother, V. 86, Georg Rehm, was born in 1895. When 15 months old he bled from the gums, between the two incisor teeth, and also from the fraenum of the upper lip. The haemorrhage was stopped by many applications of the cautery, but the child died. With reference to the boy, V. 91, who has been mentioned before as having died before the age of 6 months, we are told that before death he "went all blue"; but the doctor in attendance stated that this was due to convulsions, and that there were no extravasations. With reference to V. 95, and V. 96, *vide* IV. 50. In the next generation VI., there is also a high rate of infant mortality. VI. 3, died before the age

of 6 months: VI. 7, at 12 months: VI. 12, at 12 months: VI. 16, at 6 months: VI. 21, 22, 24, 28, 30, 38, 42, 43, 44, and 52, at 6 months: VI. 25, 45, and 49, at 12 months: VI. 58, at 6 months: VI. 61, at 12 months: VI. 66, at 6 months: VI. 73, at birth: VI. 75, 76, 77, 78, at 6 months. With reference to VI. 1—7, Lossen's text gives five daughters and two sons, not six daughters and one son, as in the chart. Again, with regard to VI. 12—18, the text once says five daughters and one son, and in another place six daughters and one son. The chart shows six daughters and one son. VI. 17, Adam Teutsch II, was born in 1898, the offspring of cousins both tainted with haemophilia. He exhibited bruises and bled badly from the nose. On one occasion he bled badly from a slight injury to the tip of his tongue. Both knee joints were swollen. Peter Klingmann, VI. 55, born 1893, is the first instance in the family of umbilical haemorrhage, of which he died on his 15th day. His brother, Ludwig, VI. 56, born 1895, is the second instance, bleeding therefrom for three days. At the age of 15 months he had a large blood extravasation all over the L. leg. Finally, shortly before the age of 3, he fell and hurt his lip, and died from the bleeding. Another brother, VI. 57, Jacob, was born in 1897, and lived only 9 months. After a fall against a coal scuttle he bled from the gums. He also bled in numerous places under the skin and died of it. VI. 60, Georg Schweppler, born 1891, bled from the upper lip. His knees and elbows very easily became swollen, especially his left knee, at the age of 3. VI. 68, Georg Michael Jäger, was born in 1895. He once bled greatly from a cut on the tip of his nose, so that the cautery was frequently required to stop it. On another occasion he bled from a wounded lower lip. He died of haemorrhage at the age of 2. VI. 70, Philipp Jäger, born 1898, was, as the late Prof. H. Lossen informs us in a private communication, the boy selected by Morawitz and J. Lossen for blood investigations. This boy bled from the umbilicus at birth, and at the age of 3 from the mucous membrane under the tongue. He also bled profusely from a small incision into an abscess on the left side of the neck under the jaw. He was in the Heidelberg clinic in 1908 bleeding from the gums. VI. 74, and 75, twins. VI. 80, Carl Philipp Sauter, born 1895, was always easily bruised and frequently bled from the gums. At the age of $8\frac{3}{4}$ he fell off his chair on to the back of his head At the time he felt nothing; he played about as usual and slept well. Nearly 24 hours later he complained of headache and shortly died in convulsions. With Hermann Bernhard Sauter, VI. 84, we reach the last of the bleeders. He was born in 1902, and died in his first month of syncope (Herzschlag). He is marked in Lossen's chart as a bleeder, but not as having died therefrom. In a personal communication from the late Hofrat Prof. H. Lossen it is stated that his parents considered this boy to be a bleeder, and that he had subcutaneous extravasations.

Having stated shortly, but somewhat exhaustively, the facts collected with so much labour by Chelius, Mutzenbecher and Prof. Lossen, we find it necessary to add some words of criticism. This family has been held up as the finest instance of the disease under consideration. Throughout, it follows the type. Males only affected: transmission through unaffected females. No transmission through the male. In the interests then of a clear conception of the nature of the disease, it is necessary that its typical illustration should be beyond reproach. There is no person, male or female, not marked as a bleeder in this pedigree who has any claim to such title: of the persons marked as bleeders we may take some exception to IV. 19, IV. 26, IV. 29, IV. 45, IV. 46, V. 5, V. 21, and VI. 55. Of these IV. 19, IV. 29, IV. 45, died in infancy and had exhibited bruising. Considering then the stock they were born into we see no reason to doubt that they were bleeders. VI. 55, also died in infancy of umbilical haemorrhage, which, as a sign of haemophilia, we have been led to regard with suspicion. IV. 26, and V. 5, are suggestive of phthisis; while palpitation of the heart in the cases of IV. 46, and V. 21, has not been often cited as an haemophilic manifestation. Those, however, who read the original descriptions of these cases will be convinced that the facts, as given here, do not represent a fraction of the calamities that have fallen upon this extraordinary family. If every case of haemorrhage were known and inserted, the account would tend to become repetitive. We venture to add that few cases of haemophilia have come to light under auspices comparable to those of this family. Its future history will not, we think, be allowed to go unrecorded. (See Bibl. Nos. 48, 123, 375, 763, 791, and 864.)

Fig. 390. *Rieken's Case.* This is an excellent and minute description of a family of bleeders, living at Nohfelden, a small town in the south-west of Birkenfeld—a principality belonging to the Duchy of Oldenburg. Rieken's work, which is one of the best in the whole literature of haemophilia, appears to be very inaccessible, as many of the standard writers mention that they have been unable to consult it first hand. Our pedigree and account are taken from the original publication, a copy of which was sent to us through the generosity of the late Hofrath Professor Hermann Lossen in Heidelberg, from his private library. Rieken expressly states that the family, described by him, was not related to other bleeder families published up to that time (1829). I. 1, 2, 3, 4, no specific information, but it is stated that they were not bleeders. I. 5, 6, no information. I. 7, Matthias F., suffered from severe joint pains from his 76—78th year, when he died. I. 8, his wife, no information. II. 1, and 2, no information. II. 4, was always healthy, and lived to the age of 82. II. 5, also healthy, died in old age. II. 6, Georg Wilhelm L., healthy, not a bleeder. II. 8, his wife, healthy. Of her brothers and sisters, II. 10, Christian, the fifth brother, is said to have suffered in slight degree from gout, but was not a bleeder. A sister, Sophie, II. 11, aged 70, married to Heinrich H., II. 12, was still living at the time of Rieken's publication. All

the others, II. 9, had died in youth or in early life. The numerous descendants, III. 10, of the brothers and sisters of II. 8, were all free from the tendency to bleeding. II. 7, was a brother of II. 6. II. 3, his wife, both lived to be old. III. 4, 5, 6, 7, not bleeders. III. 6, includes nine members of the family of whom it is stated that some died early of variola or other epidemic diseases, others living to a great age. III. 7, Ernst P., father of bleeders, a husbandman and carpenter, alive at time of Rieken's publication, and 86 years of age; of medium size, with blue eyes and grey hair. At no period of his life had he shown an haemorrhagic tendency. With the exception of smallpox, and an attack of miliary fever, he had always been well. This was justified by his appearance when seen by Rieken, for, at the age of 85, he repaired a bridge at Nohfelden, and in doing so had to stand in cold water up to his knees for 8—10 hours during almost a whole week. In 1771, at the age of 28, he married Sophie Marie L., aged 17, III. 8, who at that time had already shown symptoms of pulmonary phthisis. At the end of 11 months, 19 days after the birth of a son, IV. 5, she died. During her pregnancy she had complained of great pain in her hand, but its exact nature was never cleared up. Eighteen months after the death of III. 8, Ernst P. married her sister, Elizabeth Maria, aged 17, III. 9, who became the mother of three haemophilic boys and one girl, alleged to be haemophilic. III. 9, was of small stature, and after her 30th year suffered from pains in the joints and kyphosis, associated with asthmatic attacks. After much suffering she became dropsical, and died in 1821, aged 66. According to her husband she was not a bleeder. Her menstruation was regular and lasted 7—8 days. She was subject to congestion and flushings, but never had epistaxis. III. 3, Andreas L., died of asthma, aged 54. IV. 2, was a daughter of the older brother, III. 4, of Ernst P., III. 7, seen by Rieken at age of 58. She is said to have had rather profuse menstruation, but did not suffer from haemorrhoids or arthritis, and was not a bleeder. IV. 5, only child of first marriage of III. 7, weakly child, who died of convulsions at the age of 15 weeks. IV. 6—17, twelve children of second marriage of III. 7, by sister of III. 8. IV. 6, Wilhelm Ludwig. Up to age of 14 years was healthy, but at that time had serious epistaxis, lasting eight days, and finally requiring plugging of nose with "Zunderschwamm" (Polyporus fomentarius). This epistaxis was only a preliminary to further troubles, for, two days after it was arrested, he complained of a feeling of tightness in the praecordium, followed by vomiting of fluid black blood which lasted a week, at the end of which time he died completely exhausted. Rieken's account was apparently obtained from the father. IV. 7, Marie Elizabeth, born 1776, died at age of 5 of smallpox. IV. 8, Marie Margarete Wilhelmine, born 1778, a weakling, unable to suck properly on account of tongue-tie. While the father was absent on the fourth day after her birth the nurse relieved the tongue-tie with some instrument and furious bleeding from the fraenulum set in, and she died the following evening of convulsions and haemorrhage, a frequent result of unskilled operations for the relief of tongue-tie. This account was confirmed by the pastor in Nohfelden. IV. 4, Jacob P., not a bleeder, cousin of IV. 9, Wilhelmine, whom he married. IV. 9, menstruated regularly but profusely, the blood being usually dark and thin. She was troubled with plethora during pregnancy, and had to be bled, but she was not a bleeder; alive and well. IV. 3, Philip P., brother of IV. 4, a healthy man, married his cousin, Juliane Margarethe, IV. 10. Like her sister, IV. 9, she was regular if somewhat profuse. At the age of 30 had swelling of the knee. Alive and well at time of Rieken's publication. IV. 11, Ludwig Paulus, died of smallpox at age of 4. IV. 12, Johann Christian Wilhelm, born 1786, a bleeder. During his first year he developed livid spots, which recurred periodically for the next eleven years. In his tenth year he developed great pains in his legs and a hard tumour on his left knee, which left him lame. A year and a half later he had violent toothache which necessitated an extraction. Very great haemorrhage ensued and could not be arrested, and on the eighth day, having become exsanguine, he died. The blood was thin and watery. IV. 13, Henriette, born 1787, died of convulsions, aged 3 days. IV. 14, Johann Wilhelm Jacob, born 1789, died of variola in his third year. IV. 15, Elizabeth Magdalene, born 1791, died of variola, aged 4. IV. 16, Philip Heinrich, born 1795, a bleeder. Livid spots were observed on his back during first year. When 17 months old he vomited a large quantity of fluid blood and died of complete inanition. IV. 17, Louise Catharine Jacobine, born 1796, 32 years of age at the time of Rieken's publication and seen by him. She was of small stature, and with an unusually fine and white skin: not a bleeder. Menstruation began in the thirteenth year, and was regular but profuse. She had variola at 6 or 7, but recovered without complications. At age of 10 toothache occurred, and all her teeth became carious. Pains in extremities developed at the age of 21. Severe *ischias nervosa Cotunni*, for which she was treated by Rieken in 1822. Fever and pain in abdomen after her last confinement. Had been bled for plethora, and it was noticed by the surgeon that the blood did not coagulate so quickly as usual and was unusually dark. She married in her eighteenth year Jacob L., V. 1, a miller, aged 19, who had no tendency to bleeding, although he suffered from congenital deafness. IV. 17, and V. 1, had six children, three of whom were bleeders. V. 2, V. 3, V. 4, three sisters of V. 1, not bleeders. V. 4, aged 40, suffered from indigestion and pains in limbs, married V. 5, Carl A., a healthy man. V. 6, Philippine, not a bleeder, married V. 16. V. 2, and V. 3, said to have had many children, both male and female. It is not stated however whether they were all married or only two of them. V. 7, brother of V. 1, healthy, married V. 8, who died without issue. V. 9, first wife of V. 10. V. 11, second wife of V. 10. She died without issue. V. 12, N. N., first wife of V. 13, died

at age of 24. V. 14, second wife of V. 13. V. 15, unmarried, Elizabeth, aged 26. At 20 she had pain in left thigh. V. 16, Philip Jacob P., aged 27, of pale complexion with blue eyes and brown hair: not a bleeder, but had neuralgia of face and toothache leading to empyaema of antrum of Highmore. V. 16, married V. 6. V. 17, Christian, aged 22, suffered from pain in limbs and a tendency to phthisis: unmarried. V. 18, Elizabeth, aged 19, menstruated very freely and suffered from pains in her neck; married V. 22, her first cousin. V. 19, female, died of variola, aged 4 years. V. 20, Ernst Jacob, died of convulsions, aged 15 months. V. 21, Elizabeth, died of croup, aged 4. V. 22, Jacob, aged 21, had suffered since the age of 12 from pains in limbs, especially with changes in weather. In 1821 was confined to bed for six months with pains in legs, ending with osteitis in his right forearm and left foot. Ulcers formed and exfoliation of bone. Leeches were frequently applied for pain, but no unusual haemorrhage was seen. V. 23, Louise, aged 16, poorly developed. Menstruation came on early and was abundant. VI. 7, Caroline Wilhelmine, born 1818, had blue eyes, blonde hair, good teeth; no symptoms of haemorrhagic diathesis. Cuts on lips and fingers not followed by unusual haemorrhage. VI. 10, Elizabeth Frederike, born 1825, died of fits at 9 months: not a bleeder. VI. 8, VI. 9, VI. 11, VI. 12, all born healthy: no umbilical haemorrhage. Skin fine and white with veins showing. VI. 8, VI. 11, VI. 12, had raven black hair, whereas VI. 9 was blonde. VI. 8, VI. 9, suckled by mother. VI. 11 by a healthy nurse, on the advice of Rieken, who thought that the haemorrhagic tendency seen in VI. 8, and VI. 9, might be prevented by the milk of a normal woman. In VI. 8, and VI. 9, teeth appeared at usual time, whereas in VI. 11 they were late. All were vaccinated without any trouble in the way of bleeding. All had stinking aural discharges. VI. 8, Philip Jacob L., born 1821. In the seventh month he began to manifest tendency to bleeding, and this continued till his death in 1825. From the most trivial injuries he developed livid spots on his buttocks, arms, sides, face, scrotum. Spots varied in size from that of an "8 groschen-stück" to that of half a hand. Sometimes, the whole body was covered with them. Rieken once counted thirty of them at one time. Hard tumours, probably due to extravasated blood, were also constant. A bite of tip of tongue at one year caused great bleeding for four days, and its arrest defied treatment. At $1\frac{1}{2}$ years haemorrhage from nose which nearly proved fatal, as he developed convulsions and coma. Up to age of $4\frac{1}{2}$ he had profuse spontaneous haemorrhages from nose every three months, and lasting 4—10 days at a time. Blood at first deep red, later, a dirty pale colour, and without tendency to coagulate. Blood stopped spontaneously, but only when patient was completely exhausted. During his fourth year pains in thighs developed and became very severe, ending with a white swelling of knee, involving the bones but not the soft parts. By degrees the swelling disappeared, but he died of enteritis shortly after a fresh attack of haemorrhage. The parents fearing an autopsy, reported his death to Rieken only eight days after his burial. VI. 9, Jacob, born 1825. In the fourteenth week after birth he showed large livid spots. At the age of 10 months a furuncle appeared in R. axilla. It was most carefully opened by a superficial cut, but after the evacuation of the pus it bled profusely for three days. Nine months later, he almost bled to death from small wound of fraenulum of lower lip. Ultimately the actual cautery had to be resorted to. For the next $2\frac{1}{2}$ years he remained well, but plethoric symptoms began to show themselves in 1827. Rieken found him in a high fever with all the symptoms of pneumonia, and blistered him with success. On the fourth day violent epistaxis set in and defied all treatment. He became exsanguine and unconscious on the third day after, and then the bleeding stopped. Towards the end the blood was quite watery. In the beginning of 1828 he developed severe pains in his limbs, especially legs, and these settled in his left knee where a swelling developed. At the end of the same year he fell and cut his tongue against an incisor tooth. Blood gushed from his mouth, and the cautery was applied thrice without effect, the bleeding continuing for seven days till he was like a wax doll in appearance. Within a fortnight after it ultimately stopped he had a copious epistaxis, and died with convulsions on the third day. In spite of entreaties Rieken was not permitted to make an autopsy. VI. 11, Zacharias, born 1826. At the age of 3 months a drunken barber ruptured the fraenulum of the tongue for tongue-tie, and violent haemorrhage ensued and lasted three days in spite of repeated cauterisation. In the sixth month spots appeared. Death took place from haemorrhage from wound of tongue caused by a tooth. No p. m. VI. 12, Jacob Carl, born July 14, 1828. At age of 2 months developed a chronic eruption of face. A livid spot was observed on the inner side of arm at age of 5 months, and accompanied by a hard tumour as in the case of VI. 8. He was treated with Glauber's salts, but without beneficial effect, fresh ecchymoses making their appearance. Believing that the child was a bleeder, and as the parents were most anxious that he should not succumb as his brothers had done, Dr Rieken, early in January 1829, put him on a course of *ol. jecoris Aselli*, which proved most beneficial. When it was stopped livid spots again made their appearance. On resuming the oil they disappeared. His aural discharge also ceased. About the age of 10 months he cut his ring-finger with a knife deep into the cutis, but the haemorrhage was not more severe than in a normal person. An incision into a boil caused evacuation of pus without haemorrhage. Another cut on the finger did not bleed profusely. At the time of Rieken's publication this child was 1 year old, and, although he expressed the intention to publish its subsequent history, he does not appear to have done so. VI. 3, male, aged 3 months, affected with *crusta lactea* (some skin disease). VI. 6,

died of hectic fever at age of 3, after suffering from rickets and scrofulous ulcers. VI. 4, Wilhelmine, aged 8, suffered from a weeping eruption of face (*crusta lactea*) and from ulcers of both legs, which were apt to bleed. The eruption and the ulcers disappeared for the most part in summer and winter, to break out again in spring and autumn: thin and weakly. VI. 5, Jacob, aged 3, had eruption on face, and was thin and weakly. (See Bibl. No. 60.)

Fig. 391. *Sadler's Case.* Sadler has given a very careful account of a typical bleeder family of the name of Wibberley living for the last hundred years in Salt Alley, Ashbourne, Derbyshire. A thorough revision of the history of this family with numerous additions and details has been personally furnished for this paper by Dr Sadler to whom we desire to express our great indebtedness. This family, now known in five generations, has shown sixteen male bleeders of the classical haemophilic type. No female has so far shown the tendency. The head of the family was Mrs Wibberley, III. 11, who furnished Dr Sadler with many details of the ancestry, in which however no undoubted case of haemophilia could be traced. I. 1, 2, 3, 4, no information. I. 5, and 6, were long lived and had 14 children. I. 7, and 8, lived to old age and had 16 children. II. 2, Wibberley sen., died of rheumatic gout at the age of 78, having at no time manifested any tendency to haemorrhage. II. 3, his wife, died of dropsy at the age of 71; not a bleeder. II. 5, and II. 8, 13 and 15 children respectively the histories of which can unfortunately no longer be traced. II. 6, the father of Mrs Wibberley, died at the age of 46 from bleeding "from the person." As Dr Sadler remarks, this is, however, indicative of local trouble and does not point to the existence of the haemorrhagic diathesis. II. 7, died in childbed at the age of 26. The cause of death is reported to have been "shortness of blood" but not haemorrhage. Transfusion was employed but unsuccessfully. III. 1, George Wibberley, not a bleeder, died aged 68, of senile decay. III. 2, 3, 4, 5, 7, 9, no information. III. 6, Fanny, died of dropsy, III. 8, Frances, died in infancy. III. 10, six children, all of whom died in early life—no details. III. 11, Mrs Wibberley, died 1910, aged 70, not abnormal in any way; married III. 1, and by him had 20 children including seven miscarriages. III. 14, William, died 1910, aged 68, not a bleeder. III. 13, Hannah, died of tuberculosis. III. 12, Reuben, died when two weeks old. IV. 1, Edward, a bleeder. First showed the haemorrhagic tendency when $3\frac{1}{4}$ years old, at which date he fell and bit his tongue and bled to death. IV. 2, a miscarriage. IV. 3, Charles. From the age of $1\frac{1}{2}$ years, when he commenced to walk, he was noticed to bruise badly whenever he fell or received a blow. He frequently developed large haematomata which took weeks to subside. His first attack of external haemorrhage was from the gum when he was cutting a tooth at the age of $6\frac{1}{3}$ years. The bleeding could not be arrested and he died completely exhausted in a week. IV. 5, John, not a bleeder—married and had six children, four males and two females all healthy. IV. 7, William, not a bleeder; was formerly a soldier; married and had four healthy children without trace of haemorrhagic diathesis. IV. 9, Hannah, married to Clowes, IV. 10. She had always enjoyed good health and had normal menstrual periods. She did not lose excessive quantities of blood during confinements but had three boys who were typical bleeders. IV. 11, Harriet, married to Warrington, IV. 12. She had not excessive losses of blood during confinements and had normal menstrual periods until the last few months when she suffered from menorrhagia, the result of endometritis. Three of her boys were typical bleeders. IV. 13, a miscarriage. IV. 14, Fanny, not a bleeder. Menstrual periods normal. Confinements not associated with excessive haemorrhage. Married to Dale, IV. 15, and by him had four bleeder sons. IV. 17, Edward 11, unmarried, aged 35, suffered for a year from haemorrhage from the bowel. No other symptoms of the diathesis—very doubtful case of haemophilia. IV. 18, Sarah, always had profuse periods and suffered from menorrhagia lasting three weeks at a time and so severe as to cause fainting. At the time of Sadler's publication (1898) she was about to be married against the wishes of her mother and sisters on account of the haemophilia in their own families. She married, however, and had two bleeder boys in a family of three boys and one girl. IV. 20, Albert, died at the age of six months from "sunstroke." He had not exhibited the haemophilic tendency. IV. 21, Julia, had normal periods and no excessive bleeding during confinements. Married to Brown, IV. 22, and by him had one son a bleeder and a healthy daughter. IV. 23, Frank, not a bleeder. Married to IV. 24, and had one normal son. IV. 25, Harry, aged 21, a bleeder. He bruised easily and bled freely from cuts or scratches. Epistaxis, but had no joints affected. IV. 26, three miscarriages, their exact position in order of birth of the family not being ascertainable. V. 1—10, not haemophilic. V. 11, Charles Clowes, a typical bleeder. At the age of 11 months he fell and cut the fraenum of his lip. The bleeding stopped only when the exhaustion was extreme. No remedy appeared to have the slightest effect upon it. He had several other haemorrhages from the same part and they were arrested only after lasting for days. Blood oozed from the gums beside the teeth on and off since he was five. The oozing came on almost every week and lasted for a few days. From the time he was 18 months he was hardly ever free from bruises. He had haemarthroses in his knees, ankles and elbows. At the age of 11 he cut his thumb and bled to death on the tenth day after all remedies had proved ineffectual. V. 12, Sarah, not a bleeder, died of croup and inflammation at the age of 3 years and 9 months, this being her first illness. V. 13, George, early showed bruises and haematomata, and this continued down to the present time. Ever since he was 4 or 5 oozing of blood from the gums had been frequent. After the extraction

of a tooth he bled for a week. A prick from a fishbone bled to such an extent that he almost became exsanguine. A cut on the head bled for a week in spite of the many remedies which were tried. His joints began to be involved about the age of 3, an injury causing one of his ankles suddenly to swell up. Later, his knee became affected and for the last four or five years he was unable to walk properly. He was 14 and with increasing age there appeared to be a proportionate diminution in the tendency to bleed. V. 14, and V. 15, twin boys. V. 14, Herbert, died of broncho-pneumonia at the age of 2 years, having up to that time exhibited no haemorrhagic tendency. The other twin V. 15, John, showed bruising early, but, chiefly on account of the great care taken of him, did not bleed externally till the age of 11, when he bled to death from a cut on the head. V. 16, Frank, 17, Emily, 18, Amos, aged 6, 4, and 2½ years respectively; alive and well, not bleeders. V. 19, William, died aged 24 hours. V. 20, Edward, "wasted away," aged 3 months. V. 21, Harry, died of convulsions, aged 1 year. Up to that time had shown no tendency to haemorrhage. V. 22, John, bled to death from wound of fraenum of lip, being then 16 months old. It was his first attack and remedies were powerless to arrest the haemorrhage. V. 23, George, bruised whenever he was touched. Even the marks of hands showed when he was lifted up. If he fell, a blood tumour would develope. He always bled from the gums when cutting a tooth. He was very carefully watched to avoid cuts or falls but developed haemarthrosis of the knee, at the age of 4½, from a slight knock. At the age of 6 he bled to death from a new tooth which pushed out an old one. On account of the danger, extraction had been carefully avoided. According to the testimony of the father nothing would cause the blood to clot and in spite of such remedies as perchloride of iron, adrenaline and calcium salts, he died exhausted after five days of continuous haemorrhage. V. 24, Edward, a bleeder, 18 months old. He bruised badly in spite of the fact that exceptional care was taken of him. V. 25, John Dale, a bleeder. As soon as he began to walk he was noticed to bruise easily and extensively from trivial falls. At the age of 18 months he bled for 14 days after biting his tongue. At 2½ years he cut his finger and bled for a fortnight. At 3 he contracted whooping cough and in the third week of the disease epistaxis set in accompanied by haemorrhage from mouth and ears and death ensued on the fifth day. This was in the Burton Infirmary where he had been on several other occasions on account of bleeding from the gums, mouth and fraenum of the lip. He had suffered from haemarthroses. V. 26, Frances, born prematurely, was always a weakling and died at the age of 10 months. V. 27, Walter, a typical bleeder. Like his brother he was several times in the Burton Infirmary bleeding from the gums. At the age of 4 months he bruised his hands from striking them on a table. His joints had been and were still (1909) affected. The greatest care had to be taken of him; aged 12. V. 28, Sidney, 9 years old, a bleeder. He bruised easily and had painful joint affections. Had been an in-patient in Burton Infirmary bleeding from the gums. Four months before he caught his knee on the corner of a form at school and had been laid up since (1909). V. 29, Harry, was attacked for the first time at the age of 15 months and bled to death from the gums. V. 30, James, 2½ years old. So far had shown no traces of the family malady. V. 31, William Edward, died of convulsions at the age of 6 weeks, having shown no tendency to bruising or haemorrhage. V. 32, George, exhibited the haemorrhagic tendency twice. He scratched his wrist and it was with difficulty that the bleeding could be arrested. Three weeks later, being then 2 years old, he fell and bit his tongue, and bled to death in six days. V. 33, Elsie, 4½ years, strong and healthy. V. 34, Tommy, aet. 2½, a bruiser. A few weeks before haematuria began and lasted for a week, recurring again after some days and lasting a like time. V. 35, Fred, bled to death into the peritoneal cavity when 2 years old. He had previously shown a marked tendency to bruise and had bled copiously from trivial scratches. Haemarthroses set in some months before his death and he was never able to walk on account of it. V. 36, Eva, healthy and well, aged 1½ years. V. 37, Edward, aged 6 months, apparently healthy. (See Bibl. No. 690.)

Fig. 392. *Brown's Case.* A short history of one bleeder with no data of eight other cases which are alleged to have been affected. I. 1, 2, 3, 4, not specifically mentioned. II. 4, died of bleeding from stomach, no other data. II. 3, sister of II. 4, and source of information respecting III. 1—6, of whom it is stated that they all died of bleeding. No other details. III. 7, male aged 13, admitted into Leeds Infirmary suffering from large swelling on outer side of R. thigh. Had been a patient on three previous occasions, viz. in 1894 when he was admitted for haemorrhage from pin prick of roof of mouth, in 1896 with haemorrhage from gums, and in 1897 when he bled into R. knee joint as the result of a fall. The swelling on R. leg above mentioned was opened by a small incision but bled copiously, and was associated with haemorrhage from gums and nose. III. 8—III. 16, healthy females. III. 17, died of haemorrhage aet. 14, no details. IV. 1, and 2, presumably unaffected. (See Bibl. No. 673.)

Fig. 393. *Elsaesser's Case.* In the years 1824 to 1833 Elsaesser of Möhringen near Stuttgart published various accounts of a family of bleeders he had first observed in 1821. The parents of this family had four daughters, II. 1, II. 3, II. 5, and II. 6, of whom one, II. 1, had two bleeder sons. Another daughter, II. 6, had a family of eight including one normal boy, III. 9, and three girls. One of these girls, III. 14, who herself exhibited a slight tendency to haemorrhage, had one bleeder son, IV. 5, and other children with a tendency to bruise. It may be noted that the children of II. 7, by his second marriage with II. 8, are described as being not affected. I. 1, and 2, though frequently ailing, lived to a

fair old age. Neither of them showed any signs of haemophilia. I. 3, and 4, were both healthy though I. 3 suffered severely from gout in her old age. II. 1, Catharina, the mother of two bleeders, married a healthy husband; as did her sister Cydonia, II. 3; II. 5, we have inserted to account for the context. Lorenz Weizenäker, II. 7, of Stetten, about five miles N.E. of Tüttlingen on the Danube in the Black Forest, married firstly Theresia, *née* Weizenäker, II. 6, a near relative and sister of II. 1, the mother of the bleeder boys, and later Juliane, *née* Büstlen, II. 9, of Stetten near Stuttgart. III. 1, represents eight children dying between the ages of 3 months and 2 years of various disorders. The bleeder III. 2, frequently suffered from haemorrhages in his youth and further bruises, joint pains and feebleness of the arms. At the age of 25 he fell off a horse and died soon after of epistaxis. He was older than his brother III. 3. This boy, III. 3, is described as a bleeder on apparently inadequate grounds. He never bled, but suffered from a painful swelling of the right knee joint and spontaneous bruises. He is described as having been weak in the feet. At the age of 14 he was completely crushed by a log (Sägeblock). III. 4, and 5, died of measles between the ages of 3 and 5 without exhibiting any haemorrhagic tendency. The position of III. 6—9 in the family is not stated. The first three died early of smallpox: the fourth was living and not affected. III. 10, the eldest of the daughters, married and emigrated with her family to Russia. III. 14, Lucca, the mother of the bleeder IV. 5, was herself considered by Elsaesser to be of that tendency. In her youth she often suffered from epistaxis and up to the age of 12 showed numerous spontaneous bruises. Her menstruation started at 15 and, though scanty, recurred twice in the month. From her earliest recollection to the age of 19 in summer, especially when she got cold, she used to develop a bright red nettlerash, which soon faded. Her tissues reacted normally to trauma. Her husband, Jacob Sehr, III. 15, age 34, a labourer, was born at Vaihingen auf den Fildern near Möhringen. He lived with his family at Kaltenthal in the neighbourhood. He married in 1816 and his numerous ascendants supplied no instance of bleeding. Lucca's sister Salome, III. 16, was considerably younger than the others. From her youth up, especially while cutting teeth, she exhibited bruises like her sister. This tendency had now stopped. She never bled. III. 17, died young, and the rest of the family, seven or eight persons, were alive and well. In the next generation IV. 2, and 3, died young. Johannes, IV. 4, the eldest brother of the bleeder IV. 5, was born healthy but after the first week became torpid and snuffling. On the fourteenth day the healed navel started to bleed and he died. The bleeder Johann Michael, IV. 5, in whose case Elsaesser was first called in, was born in 1818. When he was 10 weeks old dark spots appeared spontaneously all over his body. These came out in crops and passed through the colours of bruises. At 8 months he bled for eight days after a fall. When he started to walk, he showed great weakness in the arms and feet. When he was 2 years old the haemorrhages were frequent. Grave attacks of epistaxis occurred every six weeks. He nearly died from a cut finger and from epistaxis after a fall. Whooping cough nearly carried him off with epistaxis, while he was prostrated with pains and swellings of ankles, knees, and elbows. On one of these occasions another physician applied six leeches to his knee and produced a grave haemorrhage lasting three days. When just over 3 years old he received a trivial wound on the side of the head, from which his mother saw three drops of blood issue. A scab formed, but 14 days later, after getting hot, he picked it off and started an uncontrollable haemorrhage, of which, with accompaniments of epistaxis and vomiting of clots, he became comatose and died. Elsaesser adds that his blood often smelt like pus. IV. 6, Johann Jacob, was born in 1821. He did not bleed unusually when cut, but exhibited bruises. His muscles were feeble and he had inflamed eyes. Elsaesser finding him suffering from worms and a distended belly, ordered a purge and produced two bright red stools. In his second year he bled profusely from a small but deep wound in the head, but the haemorrhage was readily stopped by plugging. At the end of his second year he died of an acute condition, the symptoms of which are given as diarrhoea, wind, the passage of dark red stinking blood followed by free haemorrhage and convulsions. A post-mortem examination conducted 28 hours later showed the whole large gut to be dark in colour, and with the lower part of the ileum full of red mucus. The bronchial and mesenteric glands were large, hard, and yellow. Of the remaining members of this family IV. 7, Catharina, born July 1824, was healthy, but at 6 months showed a few bruises on the back of her forearms. IV. 8, Christian, born two years later, was healthy except for one bruise on the arm. Another boy, IV. 9, was born on March 2, 1829. He died on the ninth day—with jaundice, trismus, aphthous condition of the mouth and watery red discharge from the navel. Carolina, IV. 10, was born in 1830. In spite of the fact that this family has a tendency to reduce itself to one certain case of haemophilia, we must point out that Elsaesser's energy has the rare merit of having been sustained over a number of years. (See Bibl. Nos. 38, 52 and 76.)

Fig. 394. *Durham's Case.* Case of a boy aet. $3\frac{1}{2}$ cut for stone by Mr Durham; death from uncontrollable haemorrhage six hours after operation. Family history of haemophilia obtained from mother, IV. 2, who said that as far back as her grandmother's, II. 2, generation and from hers downwards, the male children had been subject to bleeding from slight causes. The number and order of birth of these males are not given. We have represented them in the pedigree as II. 3, and III. 3. IV. 3, 4, 5, three brothers of III. 2, died of haemorrhage before they were 4 years old. V. 1, died from haemorrhage aet. 7, from a bite on the tongue. Bleeding lasted six days and could not be arrested. V. 2, died of

haemorrhage from lancet wound (abscess), aged 13 months. V. 3, at age of 3¼ fell and cut his gums and died from uncontrollable bleeding after four days. V. 4, Mr Durham's patient, James Joy, aet. 3½, admitted into Guy's Hospital (9/4/1866) suffering from stone in the bladder. Fair child, clear complexion, light hair, blue eyes. The mother, IV. 2, gave history of V. 1, 2, 3, 4, just related but Mr Durham decided to operate. Lateral lithotomy performed on the straight staff and uric acid calculus easily removed. Operation at 2 P.M. (17/4/66) was followed by comparatively little bleeding which indeed had stopped by 5 P.M., but soon after this oozing commenced and, in spite of every measure, could not be stopped, the child dying at 8.30 the same evening. Autopsy by Dr Moxon, showed an effusion under occipito-frontal fascia. No actual bleeding point could be found on injecting water into the iliac arteries. The number of leucocytes was found to be increased. V. 5, male children (how many not stated) affected with bleeding. Cousins of V. 1, 2, 3, 4. (See Bibl. No. 298.)

Fig. 395. *Wardrop's Case.* Wardrop relates the history of a family occurring in the practice of Mr Ward, Surgeon, at Ewell (? Surrey, near Epsom). The account given by Wardrop is evidently that sent him by Mr Ward. I. 1, and I. 2, not mentioned. II. 1—II. 5, all stated to have had the haemorrhagic tendency. Three died from division of the fraenum linguae and one from the extraction of a tooth. The other had the same disease but died from some other cause. II. 6, 8, not affected. II. 10, presumably not affected. III. 1, 2, 4, 5, stated to have been afflicted with haemophilia, no details. III. 3, 6, 7, not affected. III. 8, many children, presumably unaffected. III. 9, aged 22, bleeder, no details. III. 10, a bleeder aged 8. Haemorrhagic tendency manifested itself at the age of 2 months, when his arm became bruised to an unusual extent as the result of a slight blow. Shortly after, a superficial wound of lip occasioned a haemorrhage which was restrained with difficulty. After a superficial wound in palm, violent haemorrhage ensued, When it had lasted for days, Wardrop noted that 12 ounces of blood oozed away in 12 hours. It was not coagulated. At a later period, when 8 years old, another considerable haemorrhage took place from a wound on the head. (See Bibl. No. 91.)

Fig. 396. *Erdmann's Case.* I. 1, always healthy, died of some acute hepatic trouble at the age of 62. I. 2, died of dropsy at the age of 71. II. 8, Carl Jakob, painter, 47 years old, never affected with excessive bleeding; at the age of 27 married II. 12, Frederike Arland. Two months later, he had haematemesis but not excessive. He also complained of pains in joints especially in spring and autumn. His six brothers and their descendants healthy except a son of the eldest, III. 1, who in youth was troubled with excessive epistaxis. II. 9, of somewhat delicate constitution, hair reddish, menstruation at first irregular. Her parents and brothers and sisters healthy. Their children were not haemophilic. II. 9, II. 11, two sisters of II. 8, suffered from pains in the joints till they were 20. III. 3, healthy descendants of brothers of II. 8. III. 4—10, family of II. 8, and II. 12, viz. 4 females, and 3 males. III. 6, died of eclampsia at the age of 1 year. III. 7, died of diphtheria, aged 2. III. 4, alive, aged 15, suffered from chlorosis and palpitation. III. 5, aged 13, had suffered from rheumatic pains in joints. III. 8, male, like II. 12 in appearance. At first dentition, convulsions, then ecchymoses on back and breast, also bruises. At 16 months acute orchitis for which leeches were applied. Severe haemorrhage followed, necessitating the actual cautery for its arrest. Subsequently severe haemorrhage from small wounds and injuries. Fell from a low stool and cut his tongue against his teeth. Bleeding set in and in spite of a variety of remedies he died on the fifth day. At first, the blood was dark, becoming reddish and serous later on. III. 8, never bled spontaneously but only from wounds. III. 9, male, aged 5, was born healthy and was vaccinated without trouble. At first dentition, severe convulsions followed by ecchymoses often as large as a "taler," on his back, breast, and nates. Fell and cut his lip; bled copiously until he was profoundly anaemic. On the third day, the actual cautery had to be applied and the bleeding stopped. Recovery was very rapid. From this time almost monthly he had a return of the "orgasmus sanguinis," and when no actual haemorrhage took place he had spontaneous ecchymoses. He had a foul stinking aural discharge. After remaining for three months free from haemorrhage he was seized with epigastric pain and vomited blood. He also bled from the finger. III. 10, nearly 2 years old, like his father in appearance, not a bleeder. Wounds did not bleed copiously and stopped spontaneously. (See Bibl. No. 144.)

Fig. 397. *Hamilton's Case.* Four typical bleeders occurring in a Jewish family living in Newark, N.J. I. 1, 2, 3, 4, stated to have been free from any haemorrhagic tendency. II. 2, not a bleeder; a native of Westphalia. II. 3, Mrs M., a native of Bohemia. Is stated to have menstruated freely and to have lost a good deal of blood after confinements. On the ninth day after her first confinement—the day of the circumcision of her child—she was very nervous and lost a quantity of blood from the vagina. Her child had been bleeding from the circumcision wound unknown to her. This was her story to explain the origin of the haemorrhagic tendency which afterwards manifested itself in her boys. II. 4, and II. 5, brothers and sisters of II. 3, number not stated, no details. III. 1, Frank M., born 1875, bled for 24 hours after being circumcised, and became exsanguine from the haemorrhage. When he began to walk he was noted to be easily bruised and large ecchymoses appeared upon him. At the age of 5 a horse trod lightly on his foot and the nail of the big toe was loosened, but no actual wound was produced. In the night the mother found him covered with blood and the haemorrhage continued off and on for seven weeks.

Haemorrhage also occurred with the loss of each tooth and at some times it was alarming. On one occasion only he had epistaxis. Joint symptoms appeared at the age of 10, the knees and elbows becoming involved, and severe joint pains followed. Mentally was unstable and suffered from tachycardia; pulse 120 per minute. III. 2, Julius M., born 1887 (no doubt 1877 is meant, see III. 6), not a bleeder; was circumcised without great loss of blood, never had ecchymoses, died of pneumonia when 8 months old. III. 3, Solomon M., born 1879, bled for three weeks after circumcision, pains in joints and swellings set in at the age of 5. When 9 he struck his head while driving into a barn. There was no wound but he developed an enormous haematoma which took four months to disappear. Pulse 104. III. 4, Jacob M., born 1881; a bleeder. Always had ecchymoses on trifling injuries. Pulse 104. When 3 he fell against an iron safe injuring his head; bleeding ensued and lasted several days. On another occasion he almost lost his life from a wound caused by a bite on the lip when he fell. Pains and swellings of joints at 5. Pulse 112. This is probably the boy referred to in Fry's paper (see Bibl. No. 676). In 1898 he was suffering from severe swelling of elbow. III. 5, Leo M., born 1883. As a result of experiences with the other boys the parents did not have him circumcised. Ecchymoses and swellings of joints but no actual haemorrhage except when his teeth fell out. Swollen knee four inches greater in circumference than on normal side. Large suggillation above knee joint. Pulse 140. III. 6, born Aug. 4, 1887, was not circumcised, never bled profusely but had ecchymoses. No joint affection. Pricked himself with a pin and bled for one hour (doubtful bleeder). III. 7, an infant. Sex not stated, born 1888, never showed any haemorrhagic tendency. III. 8, and III. 9, children of brothers and sisters of II. 3, no information except that they were not bleeders. (See Bibl. Nos. 568 and 676.)

Fig. 398. *Buels' Case.* Account of the Collins family living in Litchfield, Conn., U.S.A. I. 1—4, no evidence could be obtained of the disposition to haemorrhage. II. 1, Rev. Timothy Collins, first pastor of Litchfield, Conn., not a bleeder. II. 2, Mrs Collins. III. 2, Oliver Collins. III. 3, his brothers, III. 4, his sisters, all free from haemorrhagic tendency. III. 1, no information. IV. 1, 2, 3, 4, 5, 7, children of Oliver Collins, viz. four males and two females. All the boys were bleeders in early life and continued so. Three of them bled to death, the fate of the fourth being unknown. One, IV. 1, died in childhood of haemorrhage following a slight laceration of the tongue. Another, IV. 2, although his life was frequently in danger from haemorrhages, lived to grow up but died of bleeding after the extraction of a tooth. The third, IV. 3, bled to death from a slight contusion of the forefinger. IV. 4, history unknown. It is related that these boys exhibited an extreme sensibility in relation to their danger, shuddering at the sight of edge tools and avoiding with the utmost caution all exposure to them. IV. 5, Mrs Baldwin, not a bleeder. IV. 7, her sister, Mrs Gaity, not a bleeder. IV. 9, and 10, families of III. 3, and III. 4, no details. V. 1, Mr Kilburn, no information. V. 2, Mrs Kilburn, not a bleeder. Her brother, Baldwin, affected with haemophilia, like his uncles IV. 1—4. He is said to have died about the age of puberty from haemorrhage set up by the fall of a pewter plate on his foot. V. 5, 6, 7, 8, not bleeders, age and sex not stated. VI. 1—4, children of Mr and Mrs Kilburn—all bleeders. VI. 5, not a bleeder. VI. 1, bled to death while young from a penetrating wound of the foot. VI. 2, died after being rendered exsanguine from epistaxis. The complication of an abscess on his leg may have hastened his death. VI. 3, at the age of 18 months nearly bled to death from rupture of the fraenum of the upper lip. VI. 4, was frequently reduced to exhaustion by haemorrhage from the fraenum of the upper lip and slight wounds in other parts of the body. Had had a swollen knee. The account of the above four boys is very involved in the original. VI. 5, not affected. Only the Kilburn branch of the family came under the direct notice of the Buels. (See Bibl. No. 29.)

Fig. 399. *Eve and Bidwell's Case.* This is the history of a family with five male bleeders in two generations. The authors also consider that two females (II. 9, and II. 10) were affected, but the evidence on which this is based appears to us to be insufficient. I. 1, dead, cause unknown. I. 2, died of heart disease at the age of 55. II. 1—5, five males who died in infancy from unknown causes. II. 6, Thomas B., bled to death in Guy's Hospital when 7 years old. II. 7, aged 22, a bleeder. Cause of death unknown. II. 8, Harry, a bleeder, was several times in the hospital for bleedings; died at the age of 26 of heart disease. II. 9, Margaret, aged 36, bruised easily, alive and unmarried, no other data; regarded by Eve and Bidwell as a bleeder. II. 10, aged 30, not a bleeder, but suffered from flooding after two confinements. II. 11, her husband, healthy. II. 12, and II. 14, two healthy females. II. 16, a male, not a bleeder. III. 1, Joseph B., aged 5, admitted into the Evelina Hospital for children (London) in 1889. He was suffering from swelling of the R. knee. The history elicited from the mother, II. 10, was to the effect that he was well until he began to walk at the age of 18 months. As a result of falls he developed very large ecchymoses, and lumps the size of hens' eggs formed on his buttocks. At two, a fall caused his R. elbow to swell and become discoloured. At 3 his left elbow became similarly affected and this was followed by swelling of a wrist, the left knee, left ankle, and finally the right ankle. He was found to be very easily bruised when pinched, but had not shown any external haemorrhage up to the time of observation. III. 2, bled to death after circumcision performed when he was 18 months old. III. 3, a male, alive, aged 18 months, had not shown any haemophilic tendency. III. 4, and III. 5, healthy families, sex not stated. (See Bibl. No. 540.)

Fig. 400. *Schneider and Cramer's Case.* Schneider and Cramer, doctors practising in Aschersleben on the eastern extremity of the healthy Harz district in Germany, each, within five years, published an account of a family of working people of that town. On comparing the two accounts, there can be no doubt that they deal with the same family and in the following we have combined the two. The parents, I. 1, and I. 2, named Frühling, were healthy working people. In 1835 they had been married 20 years, and had had ten children, including four sons, of whom three were bleeders, and six healthy daughters. Of these latter, one, II. 6, married and had one child, a bleeder boy, III. 1. II. 1, the eldest son, first showed signs of the disease at the age of 2. He started to bleed spontaneously from a small orifice on the inner surface of the upper lip, and after three weeks succumbed. The second son, II. 2, suffered from various spontaneous bleedings. At the age of 9, in consequence of a painful swelling of the right hip, he had five leeches applied. In spite of the presence of more than one doctor, he bled to death in 14 days. The third son, II. 3, died early of convulsions and had shown no signs of haemophilia. The fourth son, II. 4, was alive. He is described as being 15 years old, stunted, with good teeth and gums. Except for a swelling of the left knee he was quite well. He had bled on numerous occasions, chiefly from the inner aspect of the upper lip and from the nose. These attacks were worse in spring and autumn, and were preceded by an outbreak of "black and blue" spots all over the body. Coincident with the haemorrhage occurred painful rheumatic affections. The bleeding did not stop till the patient had fainted from loss of blood. He had been vaccinated without untoward manifestations. The blood of this boy, after an epistaxis lasting eight weeks, was described by Cramer as being thin and dark and little apt to coagulate. He also remarked that none of these boys bled while suckling. At the age of 22 the daughter, II. 6, married a healthy woodman, Kebener, aged 26, II. 5, and had by him one child, III. 1, a bleeder. II. 6, and II. 7, her adult sister, were the oldest of the daughters, at any rate, of those surviving. II. 8, was 12 years old. II. 9—11, all died in infancy; according to Schneider two of these were twins. III. 1, the bleeder boy, when attended by Cramer was 16 months old. He was breast fed, had a few teeth and had been vaccinated. At the age of 3 months a few blue spots were noticed. When one year old he fell out of his perambulator and slightly injured his left eyebrow. Blood started to flow from a minute spot on the inner aspect of the upper lip: this haemorrhage lasted one week, and when it stopped, the blood broke out from the injured eyebrow. In spite of Cramer's efforts, this bleeding lasted 9 days. The child then recovered completely, with, however, a swelling of the right knee. (See Bibl. Nos. 64, 87.)

Fig. 401. *Burger's Case.* Burger has given an account of a large family living in Endingen, Kaiserstuhl (Baden). I. 1, Bürgermeister Heinrich of Hecklingen, living about 1812. By his wife, I. 2, he had four daughters. One of these, II. 2, married in Riegel (Baden), but died without issue. Another, Veronika, II. 4, married II. 3, Matthäus Hämmerle, of Hecklingen, and by him had three daughters and two sons, one of the latter being a bleeder. II. 5, the third daughter of I. 1, and I. 2, married II. 6, Märzweiler, and they emigrated to America. Nothing is stated with regard to their family if any existed. The fourth daughter of I. 1, and I. 2, Katharina Heinrich, II. 7, married Ligibel, of Hecklingen, II. 8. III. 1, Benedict Hämmerle, a bleeder, was a frequent sufferer from epistaxis, and was said to be crippled; died at 20. III. 2, Müller, of Hecklingen, married to Barbara Hämmerle, III. 3, who was large and stout. Between the ages of 30 and 50 she used to vomit blood or pass it by the bowel during the spring and autumn. Throughout her life she showed livid spots from slight contusions, and she became stiff and unable to walk. There was no evidence that she bled in youth. She died at the age of 72. III. 4, Kopp, of Hecklingen, married to III. 5, Cäcilie Hämmerle. III. 7, Theresia. III. 8, Joseph, not haemophilic. III. 11, emigrated to America—no information. III. 16, a male, bleeder. He had numerous manifestations of the disease such as epistaxis, and joint swellings, and ultimately died of it when 10 years old. III. 17, Schindler, married to III. 18, Katharina Ligibel. She is described by Burger as the third daughter, but evidently the fourth is meant. IV. 1, male not affected. IV. 3, Muser, of Bleichheim, married to IV. 4, Barbara Müller. IV. 5, name not given. Stated to have been a bleeder. He had epistaxis, swelling of the joints, ending in lameness, and he ultimately died of haemorrhage from the urinary passage. The three brothers of IV. 5, viz. IV. 6, 7, 9, were not bleeders. IV. 10—18, all healthy. IV. 19, not affected (in the text referred to in the plural). IV. 21, 23, 24, 25, healthy. IV. 27, Josepha Schindler, married in Endingen. IV. 29, Bertha, married to IV. 30, Helbling, of Endingen. V. 1—7, healthy. V. 8, Robert Muser (*vide* also, Lea Gutkin, Bibl. No. 773) aged 28—a bleeder; living. Information regarding him was obtained from his mother, IV. 4, who stated that during her pregnancy she suffered much from epistaxis. The labour was normal and the child was fat and healthy until he was 3 years old. At that time the father, IV. 3, noted the presence in the child's right groin of a swelling as hard as a stone. The child was pale and evidently suffered pain. In the course of time the swelling disappeared and a similar one appeared on the right knee. These swellings passed from one joint to another and still occurred, especially after exercise. Haematomata arose from the slightest knocks, and he was formerly subject to bleedings from the nose and gums. The onset of a haemorrhage was heralded by a period of irritability. He bled continuously for a week as the result of a slight cut with a circular saw. V. 9, brother of V. 8, and like him a bleeder. Healthy till 3, he

suddenly in that year developed a swelling of the R. knee, which left him lame. He bled frequently from nose and gums, and the haemorrhage was very difficult to arrest. At the age of 15 he had severe haematemesis, and, after filling two washing bowls with his blood he died. V. 10—43, all healthy, viz. 12 males and 21 females. V. 44, marked in Burger's pedigree as a bleeder but no data given. V. 45, August Heinrich Helbling, aged 19, a bleeder. Walked at nine months, but as a result of falls, large extravasations of blood developed on his buttocks. At the same time he had severe epistaxis, lasting four or five days and recurring almost every month. Physicians were powerless to cause arrest of the bleeding. It was observed that the blood, when collected in vessels, remained fluid for hours. When once the haemorrhage ceased, recovery from the anaemia was very rapid. By the time he was 15 the tendency to epistaxis had greatly diminished. At the age of 6 his joints became involved, starting first with the right ankle. The swellings passed away except in the case of the left knee, which was permanently damaged, and, in consequence, he was lame. The swellings of the joints were frequently preceded by attacks of epistaxis. Spontaneous bleedings from the gums occurred repeatedly. At the age of 16 he was seized with haematuria. Otherwise he was healthy. V. 48, Ernst Helbling, a bleeder, presented symptoms similar to those in the case of his brother, V. 45, and he bled to death at the age of 8 as a result of a bite on his tongue. V. 49, Rudolf Helbling, another brother, also showed all the signs of the family complaint and bled to death from the gums, being then 3 years old. V. 51, Friedrich Helbling, died at the age of 6 from a gun-shot wound. He was not a bleeder. V. 55, Friedrich Helbling, not a bleeder, died of whooping cough, aged $1\frac{1}{2}$. All the daughters in this family, viz. V. 46, 47, 50, 52, 53, 54, 56 were free from the disease. VI. 1—4, healthy. (See Bibl. Nos. 714 and 773.)

Fig. 402. *Benedict's Case.* History of a Scottish family, MacG., from Dundee, but living in Athens, Ga. The inheritance is stated to have been on the mother's side. The females in the family did not bleed excessively. I. 3, died from haemorrhage following an operation for a fatty tumour. II. 3, sex not stated, but probably a female. II. 4, and II. 5, died from haemorrhage from accidents. III. 1, MacG., a native of Dundee, Scotland. III. 2, Mrs MacG. III. 3, died from a "traumatic dissecting temporal aneurism" (probably a haematoma(?)), which undermined the whole of the scalp and extended into the upper eyelids. The right temporal artery was ligated without causing cessation of the haemorrhage, blood flowing from the cutaneous incision till he died. III. 4, burst a blood vessel in his lung while coughing. He brought up enormous quantities of blood, and died exsanguine. In the absence of other data it is impossible to assert that he was a bleeder. IV. 1, David MacG., aged 14, a bleeder. At 2 fell and cut his upper lip in median line; actual cautery required three times before the haemorrhage ceased. A year later, another cut in the lip bled for many days, and only ceased on the application of the actual cautery. About the same time he fell and cut his forehead. This wound oozed for five weeks without ceasing. At four he cut his finger and bled for three weeks. Continuous pressure had to be exerted for two weeks. Bleeding from the teeth had also been frequent and severe. While still living in Scotland he bled for 81 days from a lower molar and the haemorrhage was then arrested only when pressure had been exerted by a cork for ten days. A tooth was extracted by Benedict, and continuous pressure had to be kept up by means of a cork for a week. IV. 2, Ernest, 11 years of age. At 3 months he fell and cut his forehead, and bled from the wound for weeks. While crossing to America at 7 years of age he cut himself on the head and bled throughout the voyage, and subsequently at Newport from sutures inserted by a physician to stop the haemorrhage. Was treated by Benedict for oozing of blood from gums. Great difficulty was experienced in controlling it. IV. 3, Strickler Gladstone MacG., 4 years of age. When $2\frac{1}{2}$ he fell, biting his tongue and causing a minute wound the size of the point of a pencil. The haemorrhage was so great that he was almost exsanguine. The efforts of two doctors had failed to arrest the bleeding. Cautery and continuous pressure of a very drastic kind ultimately made it stop. When 4, he struck his head on the wheel of a buggy and developed an enormous haematoma of the head and face. The right side of the buccal cavity was occupied entirely by it, and it hung like a bag under his jaw. Ultimately, however, complete absorption took place. Dr Benedict found that in the case of these boys no remedy appeared to have the slightest effect in arresting haemorrhage. (See Bibl. No. 551.)

Fig. 403. *Bowlby's Case I.* Generations I. and II. healthy: not haemophilic. II. 2, alive, aged 70. II. 3, alive, aged 60. III. 1, died of bleeding from mouth, aged 2 years. III. 2, aged 31, a bleeder, great haemorrhage after tooth extraction at 7; nearly bled to death from a cut of the lip at 11. Admitted, into St Bartholomew's Hospital, under Mr Holden, at age of 19, for extravasation of blood in the right calf. Since early childhood subject to sudden swellings of ankles, knees and elbows. III. 3, healthy, not a bleeder. III. 4, first showed himself to be a bleeder when cutting his teeth. Bled furiously from trivial cuts and several times was in danger of his life. Swelling of right knee at 5. Left knee also swollen. III. 5—III. 8, four sisters, not bleeders, unmarried.

Fig. 404. *Bowlby's Case II.* Haemophilia in two brothers. I. 1, and I. 2, not bleeders. II. 1, aged 36, admitted into St Bartholomew's Hospital under Mr Willett in 1885. Had bled furiously from scratches and after tooth extraction. Left knee swelled when he was 7, and he had a similar attack at 8. II. 2, stated to be a bleeder—no details. (See Bibl. No. 552.)

Fig. 405. *Du Bois' Case.* Du Bois relates the history of a family of five males and one female, four of the males being affected with haemophilia. They lived at Auvernier, a small town near Neuchatel, the father, I. 2, being a gardener of that town, while the mother, I. 1, was a native of Nassau. I. 1 was of robust constitution and not affected with haemophilia. She also alleged that of her numerous ancestors none were bleeders, although Du Bois says it is probable she suppressed the truth. She may possibly have been connected with some of the Nassau bleeders. I. 2, healthy. II. 1, died of convulsions when one day old. II. 2—II. 4, died of haemophilia, and II. 5, aged 7, was also a bleeder. II. 6 was healthy. With respect to the bleeders, it is stated that all were born at term, and were nourished by the mother. Within a fortnight after their birth bruises appeared spontaneously or from trivial injuries. Later, in the first to third years, they had epistaxis and bled profusely from trivial wounds. Pressure of the finger caused livid marks to form. Haematemesis and bleeding from the bowel were also seen. The fourth child died from biting his tongue accidentally during play. Painful swellings of joints, ankles, knees and elbow were also seen. Leeches were applied to swollen joints of II. 2, and the haemorrhage was only arrested after three days, and with the aid of sutures. They were all blond and had blue eyes. Dentition normal but very precocious. II. 2, died of epistaxis at $2\frac{1}{2}$ years. II. 3, died of haemorrhage from mucous membranes at 8. II. 4, died of wound of tongue at 20 months. (See Bibl. No. 106.)

PLATE XXXVI. Fig. 406. *Davis' Case.* Theodore Davis, a surgeon of Nailsea, near Bristol, published in 1826 an account of a family of bleeders, which was, no doubt, more extensive than the accompanying table shows. The woman, II. 2, of strumous appearance, had a bleeder brother and bore two bleeder sons. Davis gives the following information supplied by her. "None of the males of her (II. 2) family had during some generations reached man's estate, and they had all of them been subject to profuse haemorrhages from trifling wounds. The females enjoyed good health and were entirely free from the tendency to bleed. But if they married, their sons inherited from them the family curse, whilst their daughters were equally free with themselves from it." Of I. 1, and I. 2, there is no information. II. 3, died after tooth extraction. III. 1, represents several daughters, who appeared strong and healthy. Their brother, III. 2, aged 12 or 13, was subject to haemorrhages which appeared spontaneously. Before the onset he was particularly buoyant. Since writing his notes Davis records that this boy had had renewed attacks of haemorrhage and that his joints were swollen. His brother, III. 3, was of similar constitution. He was four years old, and when Davis was called to see him, was in the throes of an haemophilic attack. A week before, he fell and slightly cut his head. The wound bled for three or four days, and then started to heal, but broke out again. His face was ashy, his lips blanched and eyes glassy. The pulse was hardly perceptible. The haemorrhage continued for a day or two after the arrival of Davis, and then epistaxis started and he gradually sank. The author, who graduated in London in 1823, and lived in Twickenham, a village near Bristol, would probably be unacquainted with the latest American and German literature of that period. His observations on the nature of this disease might therefore be considered to be of greater interest. (See Bibl. No. 46.)

Stahel's Cases. Figs. 407, 413, 414, 415. Hans Stahel has published a very complete account of a number of bleeders occurring in several families in Wald (Canton Zürich), a small town of 7000 inhabitants and situated over 2000 feet above the sea level. The surrounding county is wooded, but the climatic conditions between autumn and spring are rigorous. The poorer sections of the community are engaged in factory work or in spinning at home. The chief dietary of this class is milk, bread and potatoes.

Fig. 407. *Case I. Heusser-Keller Family.* The progenitors of this large family were I. 1, Erhard Heusser and his wife, I. 2, Susanna Keller. No detailed information is now obtainable, but they were probably born between 1750 and 1760. I. 2, bore four daughters, three of whom gave birth to bleeders. II. 1, Jacob Müller, married II. 2, Cleovia Heusser, who was born in 1779. She was a plethoric individual, and thus had often recourse to leeches. At the age of 40 she developed a swelling on the nape of her neck. It opened spontaneously and sanguineous fluid came through numerous apertures. The cause of her death is unknown. II. 3, Kindlimann, a powerful athletic man, died at an advanced age. He married the second daughter of I. 1, and I. 2, namely II. 4, Regula Heusser, who had the same habitus as her sister, II. 2. The third daughter of I. 1, and I. 2, was Anna Heusser, II. 5, but Stahel could gain no information about her. II. 6, Felix Keller, married II. 7, Barbara Heusser, who died of pulmonary complaint, associated with much haemoptysis. She was ill nine days. In the third generation the three sisters Heusser had between them 15 children (excluding four miscarriages), of whom seven were males and eight were females. The seven males were bleeders. III. 1, Felix Müller, son of Cleovia Heusser, a bleeder. Soon after his birth he showed discoloured swellings on various parts of his body. The slightest knock produced great lumps, and from a minute abrasion of the skin he bled to death, being then eighteen months old. III. 2, Jakob Müller, the second son of Cleovia Heusser, was like his brother, III. 1, a bleeder. The symptoms of the disease manifested themselves shortly after he was born, and he bled to death from a trivial cut when he was 2 years old. III. 3, Johann Müller, Cleovia's third boy, was affected in a manner like his two brothers, and bled to death from a cut on the cheek at the age of $2\frac{1}{2}$. Cleovia Heusser had three daughters, viz. III. 5, III. 7, and III. 9 (Anna

Müller), who married respectively III. 4, Kindlimann, III. 6, Honegger, and III. 8, Kunz. III. 5, III. 7, and III. 9, each bore a bleeder boy. III. 9, was alive, aged 58, and supplied the information respecting II. 2, III. 1, III. 2, III. 3. III. 9, had an extraordinarily fine skin, and showed a greenish discoloration on the temple. By the marriage of Barbara Heusser (II. 4) with Kindlimann (II. 3) there were three daughters and two sons, both the latter being bleeders. The first daughter, III. 10, married Weiss, III. 11. The second daughter, Susanna Kindlimann, III. 12, aged 55, of medium size and somewhat corpulent. Her skin was white and remarkably fine. She was healthy. On one occasion while pregnant a lump appeared on her left leg. It increased in size and then opened spontaneously, discharging a "fluid like blood." She died in 1878 of an apoplexy with right hemiplegia and aphasia. III. 12, married III. 13, Heinrich Kindlimann. The third daughter, III. 14, married Bietenholz, III. 15. The eldest son of II. 3, and II. 4 was Rudolf Kindlimann, III. 17, alive, aged 58, a gunsmith. He early showed signs of haemophilia, any injury producing a bruise or a haematoma. At the age of 12 he began to suffer from violent epistaxis, which was very difficult to arrest. The attacks were prone to develope after severe bodily exertion. After he was 21, apparently without cause, his knees and elbows began to swell. During these swellings he often spat blood. Between 20 and 22 he had very copious epistaxis and haemorrhages from the gums. At the age of 40 he had an attack of jaundice, and about this time often spat out clots of blood. The joint swellings were constantly recurring, but not at any definite periods. By degrees almost all his joints became involved. He also passed blood *per rectum* without any ascertainable cause. At 52 he had pneumonia with very profuse sanguineous expectoration, epistaxis and delirium. He was not alcoholic. Latterly he was very bent, with a large knob-like cyanotic nose. Teeth defective and carious: gums swollen, dark red and softened: creakings in joints and limitation of movements. III. 17, married a healthy woman, III. 16. III. 19, Jakob Kindlimann, brother of III. 17, was also a bleeder. He was 56 years old, and married to III. 18, Barbetta Hablützel of Feuerthalen. III. 19. He began to show symptoms of haemophilia at the age of 5 years. Massive discoloured swellings appeared after slight injuries and cuts led to great haemorrhage. About the age of puberty epistaxis was particularly distressing. From eighteen onwards painful swellings of his large joints made their appearance and lasted for weeks. Resolution was not complete, so that he was partially lamed. Some of his night attacks of epistaxis were so bad that he awoke to find himself in a pool of blood. The marriage of Felix Keller, II. 6, and Barbara Heusser, II. 7, was productive of four children, viz. two sons and two daughters. Both the boys were bleeders. II. 7, also miscarried four times (III. 20—III. 23), these being marked female on Stahel's chart. Of the daughters, III. 24, Elizabeth Keller married Jakob Hurlimann (III. 25), who died of cancer of the oesophagus at the age of 60. III. 24, herself was of medium size and very corpulent: she had a fine white skin and red cheeks. She began to menstruate at the age of 14, and was normal. Her confinements were not associated with excessive bleeding. III. 26, the sister of III. 24, married Honegger, III. 27. The two bleeders in the family of II. 6, and II. 7 were Jakob Keller, III. 28, and Johannes Keller, III. 29. III. 28, early showed himself to be a bleeder as he developed discoloured lumps on his body. As he was learning to walk these lumps were of enormous size on the forehead, buttocks and knees. At the age of 3 he injured his tongue with a small piece of wood and bled to death in two days. III. 29 showed exactly the same symptoms as III. 28, and at the age of $2\frac{1}{2}$ bled to death from a most insignificant cut on his finger. In the fourth generation of Heusser-Keller family there were 18 males, including 16 bleeders, and 11 females. With the exception of IV. 27, whose father was also a bleeder (III. 19), all the bleeders of the fourth generation were the product of unaffected females of the third generation. The two unaffected males (IV. 23, and 24) of the fourth generation had a bleeder, III. 17, for their father. IV. 1, Heinrich Kindlimann received a minute superficial abrasion on the finger when 2 years old, and after bleeding for eight days he died. Therapeutic measures to arrest the haemorrhage proved useless. IV. 2, Jacob Honegger, a cousin of IV. 1, was also a bleeder. At the age of six months a hard swelling showed itself in his skin. After falls he developed enormous swellings on his forehead, knees and elbows. At 18 months of age he nearly bled to death from a bite on the tongue, the result of a fall. It was only when he had become exsanguine, that the flow of blood ceased. At the age of $4\frac{1}{2}$ he developed a blister, on his big toe, the result of a mechanical injury. The blister burst and he bled to death. IV. 3, Rudolf Kunz—a bleeder, cousin of the bleeders, IV. 1, and IV. 2. The haemophilic tendency showed itself in his first year when he developed numerous blood lumps often as big as walnuts. These lumps disappeared in 8—10 hours (days?) and were associated with the play of colours seen in haematomata. Great care was taken of the child, but from his 5th to his 10th year he still showed haematomata. From his twelfth year he now became subject to terrific epistaxis, which ceased only when he was in a state of syncope. Between his attacks he was a bright lively red-cheeked boy. In his thirteenth year he injured his finger and sustained a colossal haemorrhage which lasted for 14 days, and defied every means used for its arrest. At the finish when exsanguine and when only serous fluid flowed, arrest occurred. During subsequent years his joints began to be seriously involved, swellings appearing without any cause, and in the end he became a cripple. In his eighteenth year a large swelling made its appearance in his right elbow. It burst spontaneously and discharged a sanguineous sero-purulent fluid. At 20 he died from the effects of a fall on his head. Before this he had shown symptoms of mental defect. IV. 4, Jakob Hess. IV. 5, Anna Kunz, healthy.

IV. 6, Heinrich Weiss, a bleeder, aged 29, married to IV. 7, Verena Bär. In youth IV. 6, had epistaxis lasting three to four days. After slight injuries discoloured lumps appeared. At 18 he sustained a great bleeding after the removal of a tooth. It ceased spontaneously. Three times he passed through a pneumonia with excessive haemorrhagic sputum. Swellings of his joints were regarded as rheumatic. IV. 8, Johannes Weiss, aged 27, married to IV. 9, Elizabetha Schmid. IV. 8, bled freely from the nose at puberty and twice had pneumonia with haemoptysis. Frequent joint affections. The evidence of haemophilia in this case is rather slight. Concerning the two sisters of the bleeders IV. 6, and IV. 8, Stahel gives no information. Susanna Kindlimann, III. 12, and her husband, III. 13, had six children, viz. three boys, all bleeders, and three girls unaffected. IV. 14, Albert, aged 32, unmarried, suffered much from epistaxis at puberty and bled for one week after a tooth extraction. He often complained of rheumatic pains in his joints. IV. 15, Johannes Kindlimann, brother of IV. 14, aged 28, unmarried: in youth profuse epistaxis and severe haemorrhages from trivial injuries: in later years haemoptysis. IV. 16, Jakob Kindlimann, aged 24, more severely affected than either of his brothers, IV. 14, and IV. 15. In youth he bled very copiously from the nose, and after trivial lesions often bled for three days; complained of pains and stiffness of his joints. IV. 17, Babette Müller, wife of IV. 16. IV. 18, and IV. 19, dead. IV. 20, no information. IV. 21, Jakob Bietenholz and IV. 22, Johann Bietenholz, both bleeders, sons of III. 14, and III. 15. IV. 21, early showed ecchymoses. Haematomata appeared after trivial injuries. At the age of ten violent epistaxis, and from his thirteenth year onwards, joints became involved, especially the knee. At the age of 18 he slightly injured his finger with a knife and sustained a grave haemorrhage which lasted for a week. Died of haemoptysis at the age of 21. IV. 22, unmarried, aged 19, showed the same symptoms as IV. 21, ailing and broken down. IV. 23—26, two boys and two girls, all healthy, children of the bleeder Rudolf Kindlimann, III. 17. IV. 27, Jakob Kindlimann, aged 17, son of the bleeder of the same name, III. 19. In early infancy he had great superficial extravasations of blood, especially on his arms. At 10 violent epistaxis; at 12 slightly injured a finger tip and bled copiously for a week. Joints attacked at the age of 13, and this left him a cripple. IV. 29, Jakob Hürlimann, born 1838, died 1877. In youth he had frequent epistaxis and bled severely for days from slight injuries. His joints escaped, but he succumbed to a terrific haemoptysis when 39 years of age. He was very alcoholic. His wife, IV. 30, was Karoline Spörri, of Sternenberg. IV. 31, Johann Hürlimann, brother of IV. 29, was also a bleeder. At six weeks hard subcutaneous haematomata began to make their appearance, and after showing a play of colours they gradually disappeared. Otherwise the child presented a healthy appearance and learned to walk early. No blood was lost when the milk teeth came through the gums. At 2 he injured the skin on the tip of one of his fingers with a knife. The injury was only a small erosion, but terrific haemorrhage set in and could not be controlled in any way, death occurring 36 hours later. According to the parents the blood spurted out and for a time, in spite of the great loss of blood he did not appear to collapse. At the end he died quite quietly. IV. 32, Joh. Hürlimann, brother of IV. 31, was also a healthy baby, but showed haematomata when six weeks old. At the age of 2 he cut his finger on a broken piece of glass and sustained a fearful haemorrhage which lasted 24 hours. Compression proved useless, the cessation of blood being ultimately brought about by prolonged application of "Rothstein," but he died of some cerebral affection, the chief symptoms of which were violent convulsions, deviation of the eyes, and pallor of the face. IV. 33, a female, died at the age of one year. IV. 34, Johannes Honegger, a bleeder. Even in early infancy he showed bruises and haematomata with discoloration of the skin. When $5\frac{1}{2}$ years old he injured the tip of one of his fingers with a knife and bled to death on the third day following. Towards the end the fluid which came from the wound was serous. IV. 35, Albert Honegger, a bleeder, aged 28, nearly bled to death at the age of 5, and had violent epistaxis at 10. Joints involved after his fifteenth year. IV. 36, Marie Honegger, sister of the two bleeders, IV. 34, and IV. 35, married Egli, IV. 37. IV. 36, was a corpulent person with a very fine white skin, and olive tinge on the temples. Her menstrual periods were normal, and her two pregnancies ran a normal course. The fifth generation of the Heusser-Keller family contained only one bleeder, V. 13. V. 1, Jakob Hess, aged 10, suffered from epistaxis, but did not bleed abnormally from small injuries: not a bleeder. V. 2, died 46 weeks old. V. 3, died aged 19 weeks. V. 4, healthy children: not marked on Stahel's pedigree. V. 5, healthy children marked as one male in Stahel's pedigree. V. 6, 7, 8, 9, on pedigree marked healthy, but not mentioned in text. V. 10, healthy children marked male in pedigree. V. 11, male, died at the age of 20 weeks. V. 12, male, died at the age of 9 months, in both cases of unknown cause. V. 13, Albert Egli, a bleeder. After an easy birth, large haematomata appeared on his buttocks, back, shoulders and head: no umbilical haemorrhage. As he began to walk the most insignificant injuries induced extravasations and suggillations. When he was $3\frac{1}{2}$ years old he fell on his face, bit his tongue, and bled to death. V. 14, female, no information.

Fig. 413. *Case II. Schaufelberger-Müller Family. Schaufelberger branch.* In addition to the large Heusser-Keller family above described, Stahel has described other cases of haemophilia, in Wald, which could not definitely be traced to the Heusser-Keller stock. I. 1, Heinrich Schaufelberger suffered for a long time from rheumatism. He was married to I. 2, Barbara Keller. The son of I. 1, and I. 2, Heinrich

Schaufelberger, II. 1, was a typical bleeder. He was observed in 1878 by Stahel. Symptoms of the disease were first noted when he was 1½ years old. After slight injuries, haematomata made their appearance and bruises were frequent without any ascertainable cause. When 4 years old he injured his tongue against a hard piece of bread, and sustained a terrific haemorrhage lasting 14 days. It ultimately ceased spontaneously when only serous fluid flowed from the wound. His joints became involved at the age of 5, the swellings affecting chiefly the knees and elbows and being associated with very severe pains. A stiffness was always left behind. These attacks were so frequent that at times he was unable to leave the house for months. Between-times he was liable to grave epistaxis lasting for three weeks. Prior to attacks of haemorrhage he was unable to eat and suffered from insomnia and palpitation, and not infrequently he became excitable or even maniacal. These prodromata were usually succeeded by sweats and then the haemorrhage started, at first very violently but afterwards the blood flowed more slowly. Present state (15/4/78) three days after having bled for three weeks; medium sized man with black hair and brown eyes, lips slightly cyanotic, bad teeth, and swollen gums receding from the teeth. Muscles of both arms atrophic; defective movements of joints of legs and arms; very lame. Two months after this note he was attacked with very severe pain and swelling in the metacarpo-phalangeal joints.

Müller branch. I. 1, Elisabeth Keller, said to be distantly related to the mother, I. 2, of the bleeder Heinrich Schaufelberger, II. 1. She had suffered for some time from an affection of the liver. Her husband, I. 2, Johannes Müller healthy. II. 1, Rosine Müller, born 1849, no information except that she was married and had two boys. II. 3, Johannes Müller, a bleeder, born 1853, and observed by Stahel in 1878. In infancy spontaneous and traumatic ecchymoses, especially on his forehead and knees. When 1½ years old, he slightly injured his gums with a piece of wood and bled to the point of complete exhaustion for four days. At five, he had haemarthrosis of left knee; later, the right knee and then both elbows. A similar recurrence of haemarthroses rendered him lame before he was 8. Stahel gives an elaborate description of his joints. In his 14th year he cut his arm with a saw and bled for eight days till he was exsanguine. At puberty, epistaxis was particularly severe and he also bled profusely from carious teeth. At 17 he had his first attack of haematuria, associated with great renal pain. The bleeding lasted 14 days. Any severe work caused painful swellings of his arms, and on one occasion his hip was involved. A second attack of haematuria occurred in 1878. II. 4, Jakob Müller, brother of II. 3, was also a bleeder, the symptoms developing in his first year. At 6 his joints also became affected and left him lame. When 12, he cut his finger and sustained a tremendous haemorrhage which lasted till he was exsanguine. In this patient, haemorrhage from the gums was particularly marked, and he bled to death in one of these attacks. II. 5, Albert Müller, born 1859, quite healthy. II. 6, Elisa, born 1861 and II. 7, Anna, born 1862, both healthy.

Fig. 414. *Case III. Kindlimann Family.* The relationship of this family with the other families could not be traced. I. 1, Joh. Zangger, married to I. 2, Anna Vontobel. They had an only son, II. 2, Marx Zangger, who married first, II. 1, Elisabetha Honegger. After her death he married II. 3, Dorothea Hess, who had previously been married to II. 4, Kindlimann, and by him had borne a bleeder, II. 8. III. 1, Barbara, died at 3 months of "Kinderwehen." III. 2, Gottfried, died of an unknown cause at the age of 1 year. III. 3, Anna, was feeble from birth and only lived a year. III. 4, Elise, died aged 2 months. III. 5, Elise, healthy girl, unmarried. III. 6, Heinrich, quite healthy. III. 7, Barbara, died at the age of 2 of a malignant tumour of the eye. III. 8, Jakob Kindlimann, a bleeder. From infancy he suffered from ecchymoses and when he was learning to walk he sustained bruises. At 4 he nearly bled to death from a cut on the finger, the haemorrhage stopping spontaneously only when he was in a state of syncope. His joints, especially his knees, became affected and by the age of 10 he was a cripple. At 16 he had a tooth removed and bled almost to death in the course of the next three weeks. He died at the age of 18 of cerebral haemorrhage, the result of a slight fall on his head.

Fig. 415. *Case IV. Schaufelberger Family.* In this instance, likewise, a connection with the other bleeder families in Wald could not be discovered. I. 1, Joh. Keller, married I. 2, Dorothea Egli. I. 1, and I. 2, had a daughter II. 2, Elisabetha Keller, born 1817. She married II. 1, Hans Heinrich Schaufelberger. II. 1, and II. 2, had two girls and two boys, one of whom, III. 5, was certainly a bleeder. Of the daughters, III. 2, Anna Elisabeth married one Spörri, III. 1, and by him had healthy children, IV. 1. The other daughter, Lina, III. 4, born 1850, was healthy. III. 3, was born and died 1844. He was operated upon, in the hospital, for harelip. Stahel says "it is very probable that he died of haemorrhage." III. 5, Huldreich Schaufelberger, a bleeder, brother of III. 3, born 1851, died 1867. At the age of 3 months his mother alleges that livid spots and lumps appeared upon him. From the play of colours which they exhibited there can be no doubt that they were haemorrhages. He also complained of violent pains in his joints which became swollen. Long continued epistaxis was common about puberty. He suffered much from toothache for the relief of which a tooth was extracted. He bled to the point of death during three weeks and then died three months later of inanition. (See Bibl. No. 420.)

Fig. 408. *Hay's Case. Appleton-Swain Family.* The following Appleton-Swain family is a widely known example of haemophilia chiefly owing to the influence of William Osler who reinvestigated it in 1885. The history of this family extends from the early part of the 18th century to the later

years of the 19th. The members were well-known citizens of Reading, a small town near Boston, Mass., U.S.A., and the infirmity was so notorious, as to be mentioned at some length in Felt's *History of Ipswich* (1834). This account is given verbatim by the editor of the *Boston Medical and Surgical Journal* (1851–2) under the heading "Extraordinary Bleeders," and it states that they lived in Hamilton, once part of Ipswich, Mass. "There are four families in this town called 'Bleeders.' Three of them are immediately, and the other mediately related. The number of individuals so denominated is five. They are thus named from an unusual propensity in their arteries and veins to bleed profusely, even from slight wounds. A cut or other hurt upon them assumes at first the common appearance, but after a week or fortnight the injured part begins and continues for several days to send forth almost a steady stream of blood, until the redness of this disappears and it becomes nearly as colourless as water. A portion of the coagulated blood forms a cone, large or small according to the wound. The bleeding ceases when the cone, which has a minute aperture and is very foetid, falls off. The persons thus constituted dare not submit to the operation of a lancet. They often bleed abundantly at the nose and are subject to severe and premature rheumatism. Some of their predecessors have come to their end by wounds which are not considered by any means dangerous for people in general. This hemorrhage first appeared in the Appleton family who brought it with them from England. None but males are bleeders, whose immediate children are not so, and whose daughters only, have sons thus disposed. As to the precise portion of those who may resemble their grandfathers in bleeding of this kind, past observation furnishes no data; it has been found altogether uncertain." Our knowledge of detail rests upon John Hay's paper published in 1813 and supplemented by Osler in 1885. Hay's account is very involved, and in constructing a pedigree we have been compelled to make a few assumptions. Our figure, however, corresponds to that given by Joseph H. Pratt (see Bibl. No. 869), and we are convinced by a personal communication from Dr Pratt, that this is a true rendering. Hay's paper is of special interest owing to the fact that many of the bleeders were neighbours of his and that his son married a girl of the bleeder stock. In this family then, we find Oliver Appleton, I. 1, an alleged bleeder, begetting three daughters. The first, II. 2, had two bleeder sons, III. 1, and III. 3, each of whom transmitted the disease through their daughters, IV. 4, and IV. 7, to their grandsons, V. 1—5. This first daughter, II. 2, also had two daughters, III. 5, and III. 9, each of whom bore bleeders, one of the latter, IV. 11, reproducing the disease through his normal daughters, V. 6, and 8, in his grandsons, VI. 1—5. III. 9, also had a daughter, IV. 16, whose grandson, VI. 6, and great-grandson, VII. 1, both on the maternal side, were bleeders. The second daughter, II. 3, of Oliver Appleton had a bleeder son, III. 12, but the descendants, if any, of II. 5, are lost sight of. It will be thus observed that in this family it is alleged that four *male* bleeders, I. 1, III. 1, III. 3, and IV. 11, transmitted the disease through their normal daughters: a most unusual occurrence. On examination of the details of these cases, we are struck by the paucity of data adduced, as compared with other well-known cases of haemophilia. Oliver Appleton, I. 1, lived at Ipswich, Mass., about 1713. He was subject from his youth to profuse bleeding from slight causes and died late in life of haemorrhage from bed sores. His daughter, II. 2, married Dr Thomas Swain, of Reading, II. 1. We are told that she pronounced V. 1, 2, and 3, to be bleeders in infancy, "her predictions with regard to her bleeding descendants being always verified." Of II. 4, Benjamin Swain of Reading, there is no information, except that "a former bleeding" is referred to. Dr Oliver Swain, III. 1, grandson of Oliver Appleton, died at the age of 33 in 1770 from the kick of a horse. The bone of the leg was laid bare for three inches. He bled profusely for some hours after the accident, but not again till four days later, when there was a little haemorrhage at the dressing. At two subsequent dressings his leg bled a little, but 11 days after the accident he began to bleed fast, with extreme pain. In the following days, during which the bleeding continued, he was at times delirious, "threatened with fever," unconscious, speechless and distressed for breath. Sixteen days after the accident the tibia was found bare for the size of a cent, "but in a good way." He died 20 days after the accident in the greatest distress of body. His brother, Dr Thomas Swain, jun., III. 3, having received a small cut with a pen-knife, almost died of haemorrhage. Soon after he was well he succumbed at the age of 30 to haemoptysis. III. 6, and III. 10, persons who married granddaughters of Oliver Appleton, were named respectively General Benjamin Brown and John Bachilor. Of III. 12, it is stated that he was a bleeder and died of "throat distemper." III. 14, we identify as a certain Mrs Whipple mentioned by Hay. In generation IV. the three sons of III. 1 are stated to be unaffected and to be living with Hay. Their brother-in-law, Jeremiah Hartshorn, IV. 5, was Hay's next-door neighbour. IV. 8, is Mr Parker of Reading who married a great-granddaughter of Oliver Appleton and was the father of the two bleeders V. 4, and 5. IV. 9, bled to death at the age of 15. IV. 11 is Nathaniel Brown, aged 54, of Reading. He told Hay that at the age of 53 he bruised his right hand and that some time later the blood burst from the bruise and flowed for a fortnight. He supposed he lost two quarts. IV. 13, and 14, were bleeders. They were dead. Their brother, IV. 15, was also a bleeder and bled profusely "last winter," 1810. (It appears from internal evidence that Hay's account was prepared in 1811.) IV. 16, a daughter of III. 10, John Bachilor, was added by Osler and spelled by him "Bacheller." She married one Norton, IV. 17. IV. 18, the son of Mrs Whipple, is mentioned and was presumably not affected. V. 1, when 9 months old bled

for nine days from a scratch, although everything was tried to stop it. His brother, V. 2, "has also bled from time to time." The third brother, V. 3, was about 8 years old. A year before, he had bled for four days from one of two small wounds on the fingers. Of the cousins of these three boys, V. 4 died of haemorrhage a year before, while V. 5 "bled profusely last year." IV. 11, Nathaniel Brown, told Hay that he thought his daughters V. 6, 8, and 10, "to be in a proper line for bleeders." The husbands of two of these girls, V. 6, and 8, were each named Norton. V. 11, and 12, were mentioned by Osler; V. 11 is Daniel Hart who married a Norton. VI. 1—5, are stated to be bleeders: one of them, VI. 4, bled to death; another, VI. 5, was drowned. Of VI. 6, Osler states that he nearly died three or four times from trivial wounds and that he finally died of haemorrhage from a slight scalp wound. VI. 7, was a Mrs Coburn of Reading, whose son, VII. 1, Warren Coburn, shared his uncle's fate. He died about 1873 at the age of 17. Osler states that he was a bleeder. Hay further states that "a person by the name of Appleton married a Smith of Haverhill, supposed to be the Mrs Smith mentioned by Dr Otto" (*vide* Otto's case). At the end of his account he mentions that his eldest son, Jonathan P. Hay, "married a descendant of Mr Appleton." They had eight children including three sons, of whom the youngest "has the complexion of the bleeders, but as yet has not bled more than common." History is silent as to the condition of this family during the last 25 years. Dr F. F. Brown, of Reading, who corresponded with Osler in 1885, stated that no Appleton or Swain families existed, in Reading, which then contained bleeder members, while Pratt states in Osler's "System" (Bibl. No. 869), and confirms in a private communication, that none of the physicians of Reading knew of any bleeders living there during the past 25 years. With regard to this well-known family, we do not feel justified in tampering with the diagnosis. We must, however, point out that in the case of I. 1, haemorrhage from bed sores late in life is no evidence of haemophilia: that III. 1, and III. 3, are not stated to have bled before the ages of 33 and 30 respectively, and that the fatal instances in these two cases are clearly explicable without assuming the presence of haemophilia. Further, V. 1 was presumably about 10 years old at the time of the account; thus only one instance of haemorrhage in ten years, is mentioned. For the rest, we must remain content with the mere statement, always excepting VI. 6. On the other hand it must be remarked that this family was notorious and that Hay's account bears the stamp of accurate observation. Hay was immediately acquainted with many of the cases and was even related to them. Under these circumstances it is highly probable that he forbore to multiply instances. (See Bibl. Nos. 25, 199, 499 and 869.)

Fig. 409. *Wachsmuth's Case.* Account of cases in the author's own family. I. 1, and I. 2, no information. II. 1, sex not stated. II. 5, and 6, suffered from gout. II. 8, Wachsmuth's paternal grandmother, alive and well, aged 92. III. 4, first wife of III. 5, Major v. G. She, III. 4, had by him ten children all healthy. III. 6, second wife of III. 5, he being also her second husband. By him she had one son, a bleeder, one normal son and one daughter, Frau Dr Wachsmuth, IV. 8. III. 5, suffered from gout and cataract but was otherwise a big strong man. III. 6, strong and healthy, aged 69. In early life she had suffered from chlorosis, disorders of menstruation and rheumatism. III. 7, first husband of III. 6, by whom she had one daughter, IV. 10, who died shortly after birth. III. 10, Wachsmuth's mother, a lively clever woman, died of hepatitis. III. 11, Wachsmuth's father, a severe sufferer from gout, died of marasmus at the age of 54. IV. 1, and IV. 2, "two daughters of a female cousin (paternal side) of my (Wachsmuth's) father-in-law." Of them it is stated that they showed suggillations and bled dangerously from wounds although they never had spontaneous haemorrhage. The one at the age of 21 bled to death from rupture of the hymen on the bridal night. The other died about the same time of haemorrhage; no data. Wachsmuth, a very careful observer, does not appear to write at first hand on these two cases which have frequently been referred to as female bleeders. We do not consider the evidence sufficient to justify the diagnosis of haemophilia. IV. 3, ten healthy children. Sex not stated. IV. 5, Carl, born in 1816, bled from umbilical cord at birth; copious haemorrhage from a boil on the cheek when four months old; extravasations of blood after injuries; profuse haemorrhage after tooth extraction when 6 years old; was leeched in that year for pneumonia and nearly bled to death. Between his 6th and 7th year he was leeched for rheumatism of hip and again nearly bled to death. Between his 8th and 11th years was in a Cadet's Academy but was discharged for some disease of his arm said to have been rheumatism. Later, spontaneous haemorrhages came on frequently. After puberty, bleedings and rheumatism became less frequent, the suggillations having ceased even before this. A phlebotomy for pneumonia at 20 years of age gave no serious after-bleeding. Was first treated by Wachsmuth, when he was 26 years of age, for a painful affection of the right hip. About this time leeches and venesection were not succeeded by severe haemorrhage. In his 27th year, however, a spontaneous haemorrhage occurred from the mouth and in three days he lost an enormous quantity of blood. Two years later he married but in the following year he ran a splinter of wood under the nail of the thumb which accident ended in gangrene and death. IV. 6, Hermann, not a bleeder but suffers from piles. IV. 7, his wife, healthy but in youth scrofulous. IV. 8, Frau Dr Wachsmuth, tall, slender and apparently strong. Hair blond; eyes blue grey; skin very fine and white. Her nervous system somewhat unstable but her intellectual faculties well developed. As a child she suffered a great deal from scrofula. Menstruation began at 17 years after a long chlorosis. The periods were not excessive and at no time did she show the haemophilic diathesis. IV. 9, an abortion.

IV. 10, died soon after birth. IV. 11, five healthy children. IV. 13, Dr Wachsmuth, the author, a man of medium size and strong. Black hair and beard, skin white, eyes grey. Frequently had epistaxis from right nostril about the time of puberty. At 17 years developed piles but these disappeared. V. 1, six years old, highly scrofulous—a bleeder. Began to show symptoms of the diathesis about two years ago although the haemorrhages were not very severe. Joints affected but not very badly. Bruises and ecchymoses frequent on hairy scalp. V. 2, Otto Wachsmuth, born 1844. There was bad bleeding when the cord was cut but it was arrested by a double ligature. In his 4th month leeches were applied for pneumonia and he nearly bled to death. The haemorrhage stopped only after he was unconscious and after the actual cautery had been applied to the bites. At 9 months the cautery had again to be requisitioned to stop a haemorrhage from the fraenum of the upper lip caused by a spoon. Blood spots appeared early and were usually large if rarely numerous. No bleeding occurred at his vaccination. At a later period during an attack of diarrhoea spontaneous haemorrhages occurred. Three times he developed joint lesions, once in the shoulder and twice in the right ankle. He also developed a stinking discharge from his ear. Some time ago he fell and badly cut his chin but no great bleeding followed. After the wound was completely healed spontaneous haemorrhage occurred from the mouth and it was permitted to go on for 24 hours, as was Wachsmuth's latest practice before proceeding to the exhibition of drugs. Otto Wachsmuth early cut his teeth and they early decayed. He had blond hair, blue eyes, and a fine white skin with the veins showing through. He was quick and bright but with a great tendency to angry outbursts. V. 3, died of diarrhoea when 4 months old. Up to that time he had shown no evidence of haemophilia. V. 4, Benno Wachsmuth. From the umbilical cord, which was wound twice round his neck, blood flowed in considerable quantity. Bruises and ecchymoses appeared early and before the end of the first year, spontaneous haemorrhages. No joint lesions up to this age. No bruises after the age of 6 months and no excessive haemorrhage after injuries. Considered to be a bleeder but if so, be must be regarded as one of slight degree. V. 5, born 1847, died of convulsions when 3 months old. V. 6, born 1848, strong healthy boy. Wachsmuth's account is full of detail but frequently somewhat vague. The evidence on which V. 1, and V. 4, are regarded as bleeders appears inadequate. Moreover as pointed out, IV. 1, and IV. 2, are far from definite Very little could have been known about them to Wachsmuth, otherwise the nature of the fatal haemorrhage would have been stated in so detailed an account. We have marked IV. 1, and IV. 2, as being, in our opinion, unaffected. (See Bibl. No. 181.)

Fig. 410. *W. H. B. Brook's Case.* A short account of one genuine bleeder, IV. 1, and an indefinite account of haemophilia in three other male members of the family. II. 3, died at the age of 11, after tooth extraction. III. 1, died from gangrene following haemorrhage into the knee joint. III. 2—4, alive and well. III. 5, informant, lost much blood at confinements. III. 7, 9, 11, 13, well. IV. 1, born 1891, suffered repeatedly from haemarthroses of knee, elbow and ankle. During his second dentition he was several times in danger of his life. Had haematuria on two occasions for four and two weeks respectively. At birth he bled from the navel. IV. 2, born 1894, bled from the umbilicus at birth and showed marked bruising at the age of 3 months. When 1 year old he died from what was diagnosed as cerebral haemorrhage. To prevent the mother from having more bleeders, Brook treated her during her third pregnancy with calcium chloride. The third child, IV. 3, was not affected with the disease. IV. 4—7, "all male children are well." (See Bibl. No. 738.)

Fig. 411. *Demme's Case.* A male bleeder observed by Demme in the Jenner Kinderspital in Bern. I. 1, and I. 2, II. 1, II. 2, II. 3, II. 4, no information. II. 5, died at the age of 5 of constantly reeurring epistaxis: marked in our pedigree as not with certainty a haemophilic. III. 4, bled to death from an injury, no details. III. 5, bled to death from the bowel, during an attack of enteric fever. IV. 1, Demme's patient, a bleeder, son of poor parents. Admitted into the hospital suffering from epistaxis which required plugging. On his left temple there was also a haematoma the size of a goose's egg. A blood examination showed the red blood corpuscles to be 3,580,000 per mm.^{3}, the ratio of white to red being 1 : 80. In the following year he again had haematomata. In a communication from Professor Stooss of Bern it is stated that IV. 1, ultimately died of the disease. IV. 2—IV. 7, six healthy girls. IV. 8, and IV. 9, both died of haemorrhage in the course of enteric fever. This in itself would be no evidence of haemophilia but it is stated in addition that from early youth IV. 8, and IV. 9, were in constant danger of their lives from uncontrollable haemorrhage following cuts and wounds. (See Bibl. Nos. 566, 583.)

Fig. 412. *Abderhalden's Case.* A short account of a family in which two males who were cousins married two sisters. In both the resulting families, haemophilia appeared. Abderhalden cousiders that the study of this family negatives with certainty the idea that haemophilia can have as its basis an abnormal constitution of the blood inasmuch as wounds of the skin behaved as they do in normal people, whereas the haemorrhages from the mucous membranes were very severe. This should point to some anomaly in the structure of the small vessels (veins) and capillaries in the mucous membranes rather than of the blood fluids themselves. The account of the bleeders given by Abderhalden is very short and apart from the general statement given above no detailed reference is made to any skin injuries which could be compared to the effects of bleeding from the mucous membranes. III. 1, and III. 4, two cousins in whose

ascent no trace of haemophilia could be discovered. They married two sisters in whose family likewise no history of haemophilia existed. IV. 1, aged 47, a male, "showed haemophilia." As a child he had long standing and severe epistaxis. It was scarcely possible to arrest haemorrhage after tooth extraction. At 42 he nearly bled to death after an operation on a small dental ulcer. IV. 9, aged 26, bled for 8—14 days after having had a tooth removed. He bled for 10 days after biting his tongue. He never had epistaxis, but on three occasions he bled into his elbow joint. IV. 12, male, aged 13, suffered from epistaxis. After a slight injury he bled into a knee joint and on another occasion into his hip joint; absorption of the blood in the second instance took three months. V. 1, ♀, aged 7. V. 2, ♂, aged 19. V. 3, ♂, aged 13. V. 4, ♂, "showed long standing haemorrhage after tooth extraction." V. 5, ♀, aged 4. V. 6, ♂, aged 1. All healthy. (See Bibl. No. 764.)

Fig. 416. *H. Fischer's Case.* Fischer of Biberach has described three cases of haemophilia in one of which there was evidence that it was a family disease. Fischer's observations were published in two separate journals. (Bibl. Nos. 622 and 623.) An account also appears in a paper by Linser with the slight modification that II. 3, is stated to be a brother of II. 2, whereas Fischer refers to II. 3, as a male relation (Geschwisterkind). In a letter from Linser to us (7/5/09) he says that the relationship of II. 3, as given by Fischer, is the correct one. II. 1, and II. 2, healthy. II. 2, was an only child. II. 3, a male relation of II. 2 (cf. *supra*). It was reported to Fischer that he bled to death after tooth extraction. III. 2, female, repeatedly suffered from epistaxis and bleeding from the gums. III. 3, and III. 4, healthy, as also III. 5, and III. 7. III. 8, and III. 10, also were free of any signs of haemophilia. IV. 1, a severe bleeder. He had livid spots and haemorrhages into his joints. IV. 2, healthy boy. IV. 3, girls, healthy, number not stated. IV. 4, Fischer's patient, a blond, blue-eyed boy of $7\frac{1}{2}$ years. During his first two years he was healthy but after this time he was scarcely ever seen to be without bruises. His grandmother had kept a diary in which 43 of his severe attacks of bleeding had been chronicled. His shoulder-joints, elbows, hands, hips, feet, toes and especially his knees, were frequently affected. The right knee was contracted. In his second year he bled profusely from a cut on the lip. At the age of 3 he was jerked out of his perambulator and was unconscious for 36 hours, probably due to cerebral haemorrhage. IV. 5, brother of IV. 4, aged 3 years, also affected with livid spots and swellings of his joints. IV. 6, girl, 5 years old, healthy. IV. 7, a bleeder, no details. IV. 8, 9, 10, healthy. Of Fischer's other cases one was a 7 year old boy whose parents and sisters were healthy. Nothing was known of the disease in the family. The boy himself constantly had livid spots and extravasations of blood. One day he was pulled by the hair of the head and a large raised up fluctuating tumour developed which on being incised by Fischer discharged dark thin sanguineous fluid. The other case was a man aged 32 who broke his bones at the elbow joint. Prior to this he had not shown symptoms of haemophilia except that at the age of 4 after a cut on the scalp he bled so copiously that he almost died. On three occasions when teeth were extracted the bleeding could scarcely be arrested. On one occasion when 18 years old he had a tooth extracted and at first the haemorrhage was not great. About six hours afterwards, however, violent haemorrhage set in. He awoke in a pool of blood and it was only after two weeks when it was feared that he would bleed to death that the haemorrhage at last ceased. He had lost 19 lbs. in weight during this crisis. The injury to his arm above described was the result of being pitched out of a cart. After the fracture was set great swelling ensued with enormous extravasation of blood so that his whole arm was $2\frac{1}{3}$ times the size of that on the normal side. The skin burst and blood welled forth and it was feared that the limb would have to be amputated, but ultimately he recovered without the loss of his arm. Of his family it is said that his sisters were feared by dentists as they were regarded as bleeders. The children of these sisters were not bleeders. The patient's own children, two daughters, were not haemophilic. He was unable to furnish any information of his mother, her brothers or her parents. (See Bibl. Nos. 622, 623 and 645.)

PLATE XXXVII. Fig. 417. *Nordberg's Case.* History of a family of bleeders living in Finland where according to Nordberg the disease is rare. No female was affected and no healthy male transmitted the disease. The mother of Nordberg's patient was IV. 12, and she asserted that her grandmother, II. 3, and her great-grandparents, I. 1—I. 4, were not afflicted with haemophilia. II. 2, Daniel S., a farmer, married at the age of 17 to Beda J., II. 3, then aged 15. He died at the age of 50, she at the age of 82. Neither was a bleeder. They had three daughters and two sons, one of the latter dying from haemorrhage. Two of the daughters had sons who were bleeders and all three had grandchildren who were bleeders. III. 5, a bleeder; at 3 had a haematoma on his head. This was incised and he bled to death. III. 6, died from some trauma at the age of 5 but the statements about him are uncertain. There is no positive evidence that he was a bleeder. III. 2, Anna Kajsa, married III. 1, Heikki M. from Keitele County (Finland) and by him had two boys and three girls. One of the boys was a bleeder; the girls were healthy. III. 4, Beda Greta married III. 3, Matti M., from Pihtipudas County. Both were healthy. III. 7, healthy, married to Salomon N., a farmer, III. 8. IV. 1, a bleeder, died from the disease at the age of 4. IV. 3, healthy, and had healthy children. IV. 4, Anna Maria, healthy, married IV. 5, Antti N., also healthy. Of their two boys one was a bleeder. IV. 6, Margareta, not a bleeder, married IV. 7, P. K., also healthy, but by him had a son a bleeder. IV. 8, died at the age of 20, cause of death unknown. IV. 9,

Vilhelmina, not a bleeder, married IV. 10, Juho R., and by him had nine children, one of whom was a bleeder. Her sister, IV. 12, married a healthy man, Petter S., IV. 11, and by him had ten children, three of whom were bleeders. IV. 14, healthy, not a bleeder. IV. 15, Frederika Maria, healthy, married F. N., IV. 16, and by him had had three boys, bleeders. IV. 17, a bleeder, died at the age of 6 from a penetrating wound of the finger. IV. 18, a bleeder, died at the age of 14 of haemorrhage from the urethra, the result of an injury. IV. 19, IV. 20, IV. 21, died of some pulmonary complaint, probably tuberculosis. IV. 22, healthy, unmarried. IV. 23, healthy, and had a healthy child figured as a male in Nordberg's pedigree, described as a girl in the text. V. 1—9, healthy (6 ♂, 3 ♀). V. 10, a bleeder, died of haemorrhage. V. 11, his brother, healthy. V. 15, a bleeder, died of haemorrhage at the age of 5. V. 16, figured and described by Nordberg as a bleeder, on account of death from umbilical haemorrhage. We consider this a very doubtful case and figure it as such in the pedigree. V. 17, a bleeder, bled to death from a trivial wound on the finger when $1\frac{1}{4}$ years old. V. 28, 29, 30, three male bleeders. V. 28, bled to death at 1 year old from a wound on the finger. V. 29, bled to death when 1 year old from a wound on the chin. V. 30, bled to death at the age of 2 years from a wound on the tongue. V. 31, 32, 33, 35, 36, 38, 39, not affected with haemophilia. Three of them were dead, cause of death not stated. V. 41, healthy. V. 42, aged 14, healthy. V. 43, bled to death from a cut on the finger when 5 years old. V. 44, bled to death from a slight cut on the gums when 4 years old. V. 45, had nearly died from haemorrhage on more than one occasion. Nordberg states that the disease first manifested itself among those affected at about the age of 6 months. No haemophilic troubles were observed after vaccination. Of the 13 bleeders in the family 12 died from the disease before the age of puberty. There was no evidence of haemophilia in the sixth generation of this family. We are indebted for an accurate translation of Nordberg's paper to Mr Palmaer, a student of the London Hospital. (See Bibl. No. 633.)

Fig. 418. *Albers' Case.* Family of bleeders living at Andernach am Rhein. I. 1, Johann Zimmermann, died aged 92, not a bleeder. I. 2, his wife, *née* Caecilia Steiner, also healthy, died in old age. II. 1, and II. 2, no information. II. 3, Anton Zimmermann, not a bleeder, died aged 60. II. 4, Johann Joseph Z. died aged 75, not a bleeder. II. 5, his wife, *née* Christine Selzer, not a bleeder, and so far as is known not descended from a bleeder family. III. 1, III. 2, III. 3, died respectively at 60, 86, and 79 years; no haemophilia. III. 5, Johann Herzmann, not a bleeder, died at age of 60. III. 6, Severin Z. died aged 58, not a bleeder. III. 7, wife of III. 5, *née* Gertrud Zimmermann, died of apoplexy aged 55: not haemophilic. III. 9, Matthias Zimmermann, living in Heister near Erpel am Rhein. Had haematemesis but no other symptom of haemophilia: married first Adelheid Eich, III. 8, who died shortly after of phthisis. By her he had two girls and a boy. He then married Maria Dung, III. 10, and by her had also three children: all healthy. III. 11, Joseph Z., not a bleeder, died of tetanus aged 7. III. 12, Anna Maria, not a bleeder, died aged 64. She married Schopp, III. 13, a roof thatcher and had by him ten children all free from haemophilia. III. 14, Elizabeth Z., married Lindlohr, III. 15, in Heister. She was alive, aged 62 and had borne 13 children; all free from haemophilia. III. 16, Johann Z., alive, aged 60, living with his wife, III. 17, *née* Verborg, in Honnef am Rhein. When younger he had one attack of epistaxis lasting one day. He had eight children, all free from haemophilia. III. 18, Joseph Z., died aged 1 year and III. 19, died aged 2 weeks, neither being bleeders. IV. 1, and IV. 2, twins. IV. 1, died aged $4\frac{1}{2}$ of a cerebral inflammation. IV. 2, died at 2 from unknown causes. IV. 3, Josephine Herzmann, aged 41, always healthy but suffering from time to time from excessive menstrual flow. IV. 4, her husband, Franz Busch, not a bleeder. IV. 5, Anna Maria Herzmann, died of inflammation of brain at age of 1 year. IV. 6, Johann H., a bleeder. At an early period he had severe epistaxis: bled to death (aged 8) from a wound of the leg caused by a fall. IV. 7, Anna Maria H., died of convulsions aged 7. IV. 8, Matthias H., died in 13th year as result of an accident; not a bleeder. IV. 9, Elizabeth H., aged 33, married a watchmaker, Reisdorff, IV. 10, and bore him two children, both healthy. IV. 11, Johann Joseph H., a bleeder; bled to death at age of 2 from a trivial wound of gum. IV. 12, Michael H., died of convulsions at the age of 1 year. IV. 13, Elizabeth Zimmermann, died on 14th day; cause unknown. IV. 14, Kasimir Z., roof thatcher, died aged 20 from an accident. IV. 15, Anna Z., died of some pulmonary trouble aged 15 months. IV. 16, Anna Z., died of cerebral disease, aged 2 years. IV. 17, Maria Z., died of pulmonary disease aged 14 years. IV. 18, ♂, died aged $11\frac{1}{2}$ years; pulmonary disease. IV. 19, Maria Elizabeth Schopp, died aged $5\frac{1}{2}$. IV. 20, died aged 2 years. IV. 21, stillborn. IV. 22, died aged 1 year. IV. 23, died aged 5 days. IV. 24, five other children: no details. IV. 25—IV. 33, 13 children, all free from haemophilia. IV. 25, IV. 26, twins, sex not stated, premature. IV. 27, IV. 28, also premature. IV. 29, stillborn. IV. 30, Gertrude Lindlohr, died of some infection at age of 17 months. IV. 31, Johann L., died of whooping cough aged 7 months. IV. 32, Gertrude, alive, aged 32, an epileptic. IV. 33, five other children; no details. IV. 34—IV. 41, eight children, all free from haemophilia. IV. 34, died of scarlet fever, aged 7. IV. 35, died of diphtheria, aged 4. IV. 36, died young, cause unknown. V. 1, Franz Busch, died of convulsions aged 1 month. V. 2, Heinrich B., a bleeder; cut his hand when 3 years old and almost died of haemorrhage; even before this suffered from ecchymoses as a result of slight pressure. V. 3, Franz B., aged 14, quite well; not a bleeder. V. 4, Peter B., aged 12, a bleeder. Eyes blue; texture of skin extremely fine. Haemophilia manifested itself

at age of $1\frac{1}{2}$ years, when dark blue spots appeared at the site of any trauma. Violent and protracted epistaxis, frequent haemorrhages from gums coming on after eating. Bleeding lasted three weeks and then stopped spontaneously. He early showed swollen joints which were extremely painful, ending with stiffness, and making him very lame. In the Klinik in Bonn to which he was brought it was observed that the slightest pressure on any part of the body made him "black and blue." The most minute abrasions or cuts bled uninterruptedly for hours or days. Ecchymosis on dependent parts. V. 5, Hans B., died of convulsions aged $2\frac{1}{2}$ years. V. 6, Stefan B., aged 6. V. 7, Ferdinand Anton Joseph, aged 4; both free from haemophilia. (See Bibl. No. 802.)

Fig. 419. *G. Taub's Case.* This contains the history of a Hungarian bleeder. Our account is taken from an abstract furnished by Professor Pertik, of Budapest, who adds that haemophilia is a rare disease in Hungary. In the records of the Stephanie Kinderspital in Budapest between the years 1873—1903, 355,842 children were treated, and of these only 58 (0·16 per cent.) were bleeders. The 58 included 18 female cases. I. 1—I. 4, no information. II. 2, and II. 3, normal. II. 4, II. 5, II. 6, three male bleeders; no details. III. 1, R. Z., observed from his 10—17th year—a male bleeder, haematomata, haemorrhages, pains in the joints. (See Bibl. No. 576.)

Fig. 420. *S. Tomka's Case.* History of a Hungarian bleeder who had haemorrhage for eight days following paracentesis of the drum of the ear. Our account is from an abstract kindly furnished by Professor Pertik of Budapest. I. 1—4, no information. II. 3, presumably healthy. II. 4, bled to death after circumcision. II. 5, "other brothers of the mother" stated to be bleeders, no data. III. 1, almost bled to death after tooth extraction. III. 2, Tomka's patient, aged $2\frac{1}{2}$, was suffering from otitis media. Paracentesis was performed and a week later violent haemorrhage ensued and lasted for eight days. Both III. 1, and III. 2, suffered from haemorrhages from various parts of their bodies—a somewhat doubtful case of haemophilia. (See Bibl. Nos. 747 and 748.)

Fig. 421. *P. Linser's Case.* History of a case, IV. 1, of haemophilia in the surgical clinic of von Bruns in Tübingen. II. 1, male, not mentioned in Linser's text but figured as a bleeder in his pedigree. III. 1—6, six male bleeders, no details. III. 7—9, presumably not affected. IV. 1, Bernard Christian, $14\frac{1}{2}$ years of age, patient of von Bruns and Linser. At the age of 7 he was in the clinic suffering from swelling of the hand, the result of a blow. During his stay in the hospital he developed bruises and copious epistaxis. He likewise passed blood clots by the bowel. Haematomata of knee and legs and repeated swellings of ankles. After his discharge from the clinic he continued to have swollen knees, elbows, feet and hands, and on one occasion his hip was involved. Linser gives a photograph of this boy who was manifestly a typical bleeder. IV. 3, a younger brother of IV. 1, died of haemorrhage. Prior to this he had swollen joints. Two other brothers, IV. 9, and IV. 10, marked in Linser's pedigree as bleeders. In Linser's pedigree the order of birth of all the individuals in the fourth generation is reversed but as he describes our IV. 1 as the eldest son we have transposed the whole generation. Linser describes two other cases and gives a pedigree identical with that of H. Fischer, Pedigree 416. (See Bibl. No. 645.)

Fig. 422. *G. Faludi's Case.* Faludi has described a case of haemophilia from Budapest. II. 1, healthy. II. 2, suffered from epistaxis. II. 3, suffered from excessive bleedings and bled to death after the extraction of a tooth. III. 1, and III. 4, two females, both healthy. III. 2, Eugen L., aged $6\frac{1}{2}$ years, a patient in the Stephanie Kinderspital in Budapest. When he was circumcised the haemorrhage which ensued was difficult to arrest. At 4 months old a blood spot first made its appearance on his chest and since that time similar spots had occurred with or without any trauma. He suffered from epistaxis and bleedings from the gums. During the eruption of his teeth considerable haemorrhage ensued. He was in hospital at the age of 2, suffering from haemarthrosis of right knee. In 1903, after injuring his R. arm, the whole limb swelled up, and he showed ecchymoses, epistaxis and swellings of his joints. About this time he fell off a stool and injured himself over the R. popliteal space. The whole leg swelled and gangrene of the foot set in and spread upwards. When the line of demarcation had extended to about the middle of the shin, amputation was performed, calcium chloride being injected per rectum. No untoward bleeding occurred, but the flaps became gangrenous and he died. The autopsy showed adhesions round the upper lobe of the right lung. Chronic perisplenitis and perihepatitis. No changes in the joints, no evidence of tubercle or lues. III. 3, died of epistaxis when 13 days old. In this family III. 2 is without doubt a bleeder, and in view of this, II. 3, and III. 3, may be admitted although the evidence is slight. (See Bibl. Nos. 762 and 766.)

Fig. 423. *Assmann's Case.* History of a family living in Strausberg near Berlin. I. 1, Gustav Gericke, not affected, dead. II. 1, Adolf Gericke, died unmarried at the age of 53. Throughout his life he had suffered from dangerous epistaxis and vomiting of blood, from an attack of which he ultimately died. From early life his joints, especially his knees and elbows, became so seriously involved that by the age of 30 he was crippled. II. 3, Pfeiffer. III. 2, Frau Schneider, III. 4, Frau Loeben, III. 6, Frau Haase, three sisters, all alive and well. III. 7, Fritz Pfeiffer, a bleeder. In his first year he had severe epistaxis and his joints became affected. During his second dentition he pulled out one of his incisors and bled continuously for twelve days and nights. On an average he bled ten times a year and at the age of 20 bled to death. IV. 1, Carl Loeben, dead, not a bleeder. IV. 2, considered by Assmann to have been

a bleeder, the evidence being that at 4 months old he bled to death from the umbilicus although it had been completely healed up. No actual wound or lesion could be detected. The haemorrhage lasted five days. IV. 3, Wilhelm Loeben, aged 30, a bleeder. The diathesis showed itself in his first year, when he bled seriously on the slightest provocation. It was alleged that after a prick on the hand with a needle he bled six months. In 1868, when 29 years old, he bled for 14 days and then the wound became purulent. His large joints were all swollen but he was not lame. IV. 4, Rudolf Loeben, aged 28—a bleeder. At the age of 3 he bled for four weeks as the result of a cut on the back of his head. He was a frequent sufferer from epistaxis. He asserted that at the age of 9 he bled for six weeks after having had a tooth extracted. Two years later he bled for 14 days from a small wound on his finger and had a similar bleeding when 12 years old. At 15 his body was covered with swellings about the size of a "two thaler piece." These swellings lasted about a year and suppurated or discharged blood. At 18 he had swollen and painful knees and elbows, and contrary to his wish a physician placed nine leeches on one of the affected joints. The result was a bleeding which lasted four weeks. At the age of 27 his left leg, up to the level of the knee, became swollen, and a discharge of blood took place and lasted for three or four months. Latterly he also bled from the gums. IV. 5—13, nine children unaffected, age, sex and order of birth not stated. IV. 14, Wilhelmina Haase. IV. 15, Pauline Luedecke, *née* Haase. IV. 17, Fritz Haase, aged 26, not affected. IV. 18, Gustave Haase, aged 25—a bleeder—continually under Assmann's observation. At the age of 3 he bled profusely from a trifling wound on his finger. At 7 he very nearly bled to death, and similar events occurred on more than one occasion up to the age of 24. In 1868 a very painful swelling developed spontaneously on his left thigh and extended from the knee to the hip. In the course of some weeks the discharge of a sanguineous purulent fluid took place just below the patella. It was over a year before the discharge ceased. The ultimate result was that the leg was rendered useless. Shortly afterwards a large haematoma appeared, involving the left calf and popliteal space. It was eventually punctured, but the skin became gangrenous and bloody pus was discharged from three apertures. Long continued suppuration followed. IV. 19, Wilhelm Haase, a bleeder; when 10 years old he bled for 11 days after having a tooth removed. At 14, painful swellings of both knees and elbows set in and this lasted till his death, at eighteen years, from the removal of a tooth. (In Assmann's chart his death is given at the age of 15.) IV. 20, Rudolf Haase, aged 17, a bleeder. When 15 years old he bled continuously for three weeks after an injury to his big toe. At 16 he bled, to the state of exsanguinity, from a minute hole at the back of his head. His elbows were early involved but it was observed that the joint swellings tended to decrease during the course of a free external haemorrhage. (See Bibl. No. 304.)

Fig. 424. *Benavente's Case.* Account of a Spanish bleeder, III. 1, with history of haemophilia on the maternal side. I. 1, Marcos de la Peña. In youth he cut his finger slightly with a table knife and bled 12 days in spite of the application of styptics. Ultimately, strong ligatures were applied but led to gangrene of the finger. He nearly bled to death, twice, after phlebotomy, on one occasion being found unconscious with the bed soaked in blood. I. 1, had six daughters none of whom inherited the haemophilic tendency. Three of these married, viz. II. 2, Magdalena, II. 4, Ramona and II. 6, Angela, and each had a son affected with haemophilia. The other three, viz. II. 7, 8, 9, remained single. III. 1, Don Mariano Dominguez, manager of a printing office, a man of plethoric constitution. In 1857, being then 34 years of age, he was seized with a violent fever which was considered to indicate the necessity for a venesection. The proposal was, however, opposed by the patient and his friends as he was alleged to be a bleeder. At the age of 11 he had bled for ten days from a trivial cut on his finger. At 18 the haemorrhage after a tooth extraction was so great that the actual cautery had to be resorted to. At 24 the extraction of a molar tooth led to such haemorrhage that he almost died. In spite of the cautery the haemorrhage persisted for 18 days. II. 3, II. 5, stated to have been bleeders—no details. Our abstract of this case has been made from the original by Dr Huÿssen, of the London Hospital. (See Bibl. No. 245.)

Fig. 425. *J. Allan's Case.* Allan of Haslar Hospital has briefly described the history of three bleeders observed in 1820. I. 2, healthy, but Allan states that "some of her relations had been the subject of haemorrhage but cannot find out particulars." II. 1, A. R., no details. II. 3, aged 18, a bleeder. On many occasions he had nearly died of haemorrhage. He was a clerk and was under Allan's observation for two years. His left arm was black from pressure against his desk. He also suffered from epistaxis and haemorrhages from the gums. Later, his joints swelled and became discoloured like those met with in sea scurvy. He died shortly after but whether of haemorrhage it is not stated. III. 1, was attended by Dr Howie, who asserted that he was affected like III. 2, and bled to death. III. 2, a bleeder, seen by Howie and Allan in 1820, being then 5 years old. Black marks developed as the result of the most trivial blow or even from pressure. He received a slight wound from a piece of glass, and in spite of all remedies he bled to death on the eighth day. III. 3, other children unaffected, sex and number not stated. (See Bibl. No. 129.)

PLATE XXXVIII. Fig. 426. *Max Fischer's Case.* History of a large family in the Württemberg village of C. near Pforzheim. I. 2, Maria Dorothea B., died of inflammation of the lungs at the age of 20. She married firstly I. 1, Christian Friedrich K., who died of some unknown cause at the age of 25, and by

him had two children in the descendants of one of whom haemophilia occurred. She married secondly I. 3, and by him had a healthy family. II. 2, Christian Friedrich K., a bleeder. He was married three times, viz. first to II. 1, secondly to II. 3, and thirdly to II. 4. By II. 1, who died at the age of 36 of some pulmonary trouble probably contracted in the puerperium, he had five children, two sons, and three daughters. By his second wife, II. 3, he had six children, two sons and four daughters. The third marriage was childless. The evidence of haemophilia in II. 2 was derived from his surviving children. He had bled frequently and copiously from the nose, and haemorrhage from fresh wounds and old wounds received in battle was great. After a fall from a waggon he nearly bled to death. He had suffered much from toothache and a year before his death when 62 years of age he had a tooth extracted. From the socket he bled for three months and then died from "nervous fever." It was considered by his descendants that he was the source of the haemophilia which appeared in the family. With reference to this man it is difficult to reject him as a certain bleeder. At the same time it seems doubtful whether a true haemophilic could undergo military service and receive *wounds* in battle which did not prove fatal. II. 3, suffered greatly from toothache, pains in the joints and rheumatism. According to the death certificate she died in childbed. II. 5, married II. 6, but died childless. III. 1, female, died of "blauen Husten" at the age of 4 months. III. 2, son, died of inflammation of abdomen. III. 3, Frederike Jacobine, healthy, died aged 36 of pneumonia in her sixth confinement. III. 4, her husband, who married secondly his first wife's half sister, III. 15. III. 6, Christiane Dorothea, aged 67, healthy, married and had ten children. III. 7, boy, born dead. III. 8, Jakob Friedrich K., not a bleeder, died of cancer of the bowel at the age of 52. III. 10, Anna Maria, alive, aged 63. As a spinster she suffered much from epistaxis and when married lost a great quantity of blood in her confinements. She never bled much after injuries. In later life she ceased to suffer from epistaxis. She suffered from neuralgia and joint pains. In Fischer's pedigree she is marked as a bleeder, but we consider the evidence insufficient to justify this diagnosis. III. 11, Christoph Friedrich B., husband of III. 10, also suffered from epistaxis although the tendency to bleeding had gradually diminished. He is also said to have suffered from chest trouble with blood in his sputum. Purulent arthritis years before. Fischer figures him in his pedigree as a bleeder but, in our opinion, without justification. III. 12, Charlotte Franziska, died of wasting at the age of 19. III. 14, Christine Barbara, died of hectic fever at the age of 34. III. 15, Jakobine Dorothea, aged 58, the second wife of III. 4, often had copious epistaxis, but as age advanced this disappeared. Fischer figures her as a bleeder—on insufficient grounds in our opinion. III. 16, died of diarrhoea and vomiting when 5 weeks old. IV. 2, female, married, healthy. IV. 4, Franziska, aged 42, always healthy. IV. 5, stillborn male. IV. 6, Louisa, committed suicide at the age of 26. IV. 7, Christian Friedrich, a bleeder. As a boy at school he bled for 8—14 days from a contused wound on the head. Frequent haemorrhages from gums and nose. Bled to death at the age of 21 three days after having had a tooth extracted. IV. 8, Gottlieb, died of diarrhoea and vomiting, aged 3 months. IV. 9—IV. 18, family of III. 5 and III. 6, residing in America and so far as was known, healthy. Before they emigrated a boy, aged 2, IV. 11, bled to death from an ulcer in the throat. "He was apparently a bleeder" (Fischer). IV. 20, 21, 23, 24, 25, 26, not affected with haemophilia. IV. 27, Christian, not a bleeder, died of cancer aged 37. IV. 29, Franziska, died of diarrhoea and vomiting when 17 days old. IV. 30, Christoph Friedrich, alive, aged 38, a bleeder all his life. At 9 years of age was very ill with pneumonia. Three or four leeches were applied behind the ear. The haemorrhage was so great that he was described as practically swimming in blood. In 1867 he bled for seven weeks after the removal of a tooth and was nearly exsanguine. In 1872 began to serve as a soldier but was discharged as a recognised bleeder, enormous haemorrhage having followed the removal of a carious tooth. (This is the case described by Grossheim, Bibl. No. 328.) In 1873, while dancing, he got a feeling of congestion in his head with giddiness and an epistaxis so violent that the blood gushed out of his nose in an unbroken stream for two hours, and lasted off and on for 14 days. At a later period he got two grains of wheat in his eye at different times. The wound caused by the removal of one healed without trouble. In the case of the other an ulcer followed and in spite of treatment bled eight days. IV. 32, Gottlieb Friedrich, a marked bleeder. In childhood bled severely from the gums. Later copious haemorrhage from stomach and intestine. In one of these latter attacks lamb's blood (130—150 grm.) was transfused into him, as reported by Gissler and Wentzel (see Bibl. No. 343). The result was extremely good, the bleeding stopping very quickly. Nine months later however he had a similar attack and bled to death. IV. 33, Karl Friedrich, a bleeder; had haemorrhages from the gums in childhood. At 2 fell downstairs and bit his tongue; severe and prolonged haemorrhage followed. At 6 or 7 years he fell from a tree and bled profusely from a cut on his head. At 13 he sprang into a vat and, injuring himself, bled for a long time. In 1870 he bled for nine days from the socket of a tooth which had been extracted. Served as a soldier in 1873, but during some sports he fell and a large swelling formed on his thigh and knee and this having existed for a long time he was discharged from the army. In 1879 he married. In 1886 Fischer saw him in the hospital at Pforzheim, where he had been admitted on account of uncontrollable haemorrhage after tooth extraction. Everything was tried without avail, but ultimately spontaneous arrest occurred. In 1888 he had a similar severe haemorrhage from a minute cut on the gum. After most of his haemorrhages, recovery was extraordinarily rapid. IV. 35, Marie Friederike, always

healthy. IV. 37, Jakobina Franziska, formerly healthy, latterly suffered from haematemesis. IV. 38, Jakob Friedrich, aged 26, the worst bleeder in the family. Constantly suffered from bleeding from the gums like his brothers. The mother related with shudders how every morning these boys had their shirts soaked with blood. At 16 he cut his hand and bled till he was exhausted. He married his deceased brother's wife, IV. 28. IV. 39, Karoline, was never healthy and died of scarlet fever at the age of 5. IV. 40—IV. 44, healthy. IV. 46, Jakob Friedrich, "ein Spurius der Mutter" died at the age of 3 as a result of a fall downstairs. IV. 47, Jakobine, 29, married. As a spinster often had copious epistaxis "but otherwise had never bled violently." In spite of this statement however Fischer has entered her in his pedigree as a bleeder. IV. 49, Gottlieb, aged 27, did not bleed profusely from small wounds although he had frequent and profuse epistaxis. Considered by Fischer to be a bleeder. IV. 50, Karoline, aged 25, was a severe bleeder. Five or seven years before she almost bled to death after the extraction of a tooth. She suffered from headache and epistaxis and at her periods lost extraordinary quantities of blood, whereas during confinements the loss was not excessive. IV. 52, Ludwig Friedrich, aged 23, a severe bleeder. The slightest injuries caused great losses of blood, although epistaxis was not so severe. Spontaneous haematomata on arm. Exempted from military service because a wound on his finger bled for six months. On one occasion he fell on his gluteal region and got a large black blood tumour. IV. 53, Karl Friedrich, twin with IV. 52. At the age of 9 months while cutting one of his teeth a blood vesicle formed and from it he bled continuously to death. IV. 54, aged 21, not a bleeder, but had very defective teeth. V. 1—V. 3, not affected with haemophilia. V. 4, died young of convulsions. V. 5, Jacobine, 18 years old, had bad teeth, no tendency to bleeding. V. 6, Christian, aged 16, a bleeder. Had frequently had epistaxis, once trod on a piece of glass and bled for 10 weeks, losing colossal quantities of blood. V. 7, Luise, not a bleeder; defective teeth. V. 8, Wilhelm, died of convulsions at the age of 9 months. V. 9, Anna Marie, aged 12, bad teeth, not a bleeder. V. 10, Wilhelmine, not a bleeder. V. 11, Gottlob, aged 9, said to be a bleeder. At the age of 2 he fell "auf der Staffel" and vomited blood. For two nights, blood flowed from the wound. V. 12, Sophie, a weakling but not a bleeder. V. 13, Hermann, aged 3, healthy so far. V. 14—V. 27, not affected with haemophilia. V. 28—V. 36, nine children of first marriage of IV. 28. Eight of these children died of diarrhoea and vomiting during the first year of life, one of these eight dying of inflammation of the lungs. Only one was alive, a girl, aged 11, not a bleeder. V. 37, had epistaxis for a quarter of an hour. V. 38, healthy. V. 39, V. 40, both died from diarrhoea and vomiting. V. 41, suffered from bad teeth. V. 42, described by Fischer as a bleeder. As a boy he once bit his tongue and bled for eight days, on another occasion he bled from a wound of the hand for three or four days. V. 46, stillborn girl with spina bifida. V. 47—V. 51, not affected with haemophilia. V. 52—V. 57, six children, one died early. One girl, aged 10, suffered from hemicrania and vomiting. V. 58, healthy. In this family the total number of members was 114, among whom, according to Fischer, there were 13 male bleeders and four female bleeders. We have already pointed out that in our opinion the evidence that the two males, III. 11 and IV. 49, were bleeders is insufficient and the same remark applies to all of Fischer's four so-called female bleeders. For the most part the evidence of haemophilia in the females is epistaxis when young. In one case only, IV. 50, there is a history of severe bleeding after a tooth extraction and menorrhagia. It is however stated that this female did not suffer excessive losses of blood during confinements. There is in this family history in general a lack of precise information which renders the diagnosis of haemophilia very doubtful in several instances, a lack of information which is commented upon by Fischer himself (p. 23). The whole family seems to have been a very unhealthy one, apart altogether from haemophilia. (See Bibl. Nos. 328, 343, 541.)

Fig. 427. *Fildes' Case.* The history of a family, two members, IV. 32, IV. 34, of which have been patients in the London Hospital several times during the last ten years. The source of the information was obtained from the boys' mother, III. 13, but is unfortunately incomplete at present. I. 3, B., appears to have been a cousin of his wife, I. 4. I. 5, G. II. 3, T. B., healthy. II. 5, M. B., living in New Zealand, aged about 90. II. 1, R., came from Exmouth. He was the only child of his parents, I. 1 and I. 2. II. 6, wife of II. 1, alive but ailing. II. 7, W. B., dead, an alcoholic. II. 8, alive, aged 70. II. 9, J. B., died of dropsy. II. 10, G. C., a miller in Kent. II. 11, a vicar at Peckham. II. 13, H. A. G., well known as an accoucheur at Poplar. He was a student at the London Hospital, but so far as is known never obtained the necessary qualifications as a registered practitioner. III. 4, E. S. III. 5, W. R. III. 7, M. R. III. 9, J. R., died of scarlet fever in childhood. III. 10, B. R. III. 12, John R., a bleeder. Said to have died of consumption in June 1885 at the age of 23. This man was always covered with bruises and would bleed two or three days from a scratch. He had "rheumatism" of both knees but not so badly as IV. 34. He was very careful of himself and never had any severe haemorrhage. He is alleged to have been in Poplar Hospital and in the London Hospital at the age of 17 (1879). On this latter occasion he was bleeding from the pharynx as the result of an injury from a fish bone. No record of this man can, however, be found in the hospital notes. III. 13, A. R. This woman is the source of information. She was born at Torquay and married in 1886. She is a small spare woman and alleges trouble with her heart. She has had miscarriages but at no time any trouble with bleeding. She is in no way neurotic. III. 14, F. R. III. 16, A. R. III. 18, D. R., living with II. 5, in New Zealand.

III. 20, N. R. III. 22, C. C. III. 24, H. C. III. 32, twenty children—no exact details. III. 33, A. H. G., a musician. He has had a harelip operated upon. He suffers from "rheumatism" and "bruises easily," but has no tendency to bleed. III. 34, F. G. III. 36, E. G. III. 38, male, died of convulsions after a fall at the age of 3 years. III. 39, G. G., female, died at the age of 17 after falling downstairs, when she became paralysed and was unable to speak. III. 41, M. G. IV. 3, W. R., married about one year. IV. 5, C. R. IV. 6, M. R. IV. 7, A. R. IV. 13, male, died of a cold at the age of 1 month. IV. 14, A. IV. 15, probably a bleeder. He died in about twenty-four hours of umbilical haemorrhage, starting at birth. A doctor was present. IV. 16, K. IV. 17, J. IV. 18, healthy male, in New Zealand with his parents. He is now (1909) about 4 years old. IV. 19, at the age of $2\frac{1}{2}$ years she fell on an iron fender and, receiving an injury to the perineum, died of sepsis. IV. 20, aged 10 years. IV. 21, aged 7 years. IV. 22, aged 13 months. IV. 23, aged about 4 years. IV. 28, H. G., aged 22, healthy; a bandsman in the army. IV. 29, D. G., healthy female, aged 21. IV. 30, A. G., aged 19, a soldier. IV. 31, G. G., aged 17. IV. 32, Frank G., aged 17, a bleeder; dark hair and somewhat phlegmatic. At the age of six months, while cutting teeth, he bled considerably from the gums. At 3 he had attacks of epistaxis lasting a month. In the same year, while on his way to school, he fell and injured his forehead, producing a large haematoma. At the age of 4 or 5 he was in Poplar Hospital for some reason, connected with haemophilia, forgotten. At this time he was covered with bruises. In 1903, at the age of 11, he was in West Ham and East London Hospital with "haemophilia." At 13 he was in the London Hospital under Dr Hutchison ($\frac{503}{1906}$)[1] with slight haemophilic haematuria: this was his first attack. In this year (1909) he has again been in the hospital under Dr Hadley ($\frac{1633}{1909}$). He was admitted with a swollen left knee, the result of a slight shock from the skidding of a bicycle. He exhibited numerous bruises and a subungual haemorrhage. During his stay in the hospital the left knee became worse, and a similar condition arose in the right knee. In the course of his life he has never had purpura. Haematuria has been common as the result of "cold." Epistaxis has been frequent, and cuts and scratches behave in a similar way to those of his brother, IV. 34. IV. 33, stillborn. IV. 34, Cecil G., aged 16, a bleeder; light hair and of lively temperament. He takes no care of himself. At the age of 12 months he fell and ruptured the fraenum of the upper lip. A doctor was in attendance, but the haemorrhage lasted 14 days. When just starting to walk, at the age of 18 months, he fell while running after his father and injured his knee. He was twice in Poplar Hospital at the age of $2\frac{1}{2}$ years, owing to a condition of the knee diagnosed as tuberculosis. At 3, he bled for one month from the forehead after a fall. In January, 1899, at the age of 5, he was admitted into West Ham and East London Hospital with a cut finger, and remained six weeks. In the same year he was in the London Hospital under Mr Eve's charge ($\frac{2927}{1899}$). Six days before admission he had fallen and cut his right hand. The bleeding was continuous. A large blood-blister on the right hypothenar eminence was incised and cleared out. No bleeding occurred. While in hospital the patient cut his finger on a piece of glass. The wound bled profusely but was immediately controlled by adrenalin. In 1900 he was six months under Dr Percy Kidd ($\frac{1534}{1900}$) in the London Hospital. Two days before admission he tripped while running and hurt his right knee. It was painful the same night and swollen the next day. In hospital he was found to be extensively bruised. The knee was splinted and decreased in size, but swelled up again on removal of the splint. While in hospital a small knock on the chest produced an haematoma, and a large bruise appeared on the left scapula, as a result, it was said, of pressure of the box splint. The dorsum of the right hand and the right knee also became bruised, while an effusion occurred into the right ankle joint. In the following year ($\frac{643}{1901}$) he was again under observation, by Dr Kidd, suffering from the same knee. He was then said to have bled for three weeks after his finger had been pricked for a blood sample. At the age of 11 years he was under Dr Hadley ($\frac{2425}{1905}$). Three weeks before admission he was bit by a boy between the legs. A week after this accident he had an attack of haematuria, which returned again three days before admission. In the following year ($\frac{414}{1906}$) he was again admitted under Dr Hadley. He had been bleeding from the gums with intervals for one week. The haemorrhage had started from biting a crust. He is said on a previous occasion to have bled three days from the palate after sucking a sweet, and for two weeks from the gums from a scratch, the result of picking a bone. While in hospital and engaged in cutting bread he cut his finger slightly and "bled considerably." In 1908, at the age of 14, he slipped off a ladder and knocked his hip against a desk. On the following day there was slight pain. This increased and the boy became faint. He was admitted under Dr Head ($\frac{659}{1908}$) three days after the accident with haemarthrosis of the left hip joint, an haematoma on the right elbow, and a "black eye." While in hospital an effusion appeared in the left knee joint. This year he has been in the hospital under Mr Furnivall ($\frac{2566}{1909}$). Five weeks before admission he had an attack of pain in the small of the back and down the right leg. There had been no accident. He was constipated for five days. There was no haematuria and no bleeding. He was in bed four weeks. Shortly before admission, though told to keep in the house, he went out, and in trying to kick a ball fell down. He was found to have an haematoma in or around the calf muscles. The swelling progressively increased in size but eventually diminished. At the same time he was covered with bruises, and bruised on very slight pressure. In addition to the above, this boy has been a patient in this hospital on two

[1] These figures represent the year and the case number in the London Hospital Registrar's records.

other occasions with haemophilia. Cecil has never had purpura, but haematuria (from cold) and epistaxis have been frequent. From a scratch he usually bleeds three or four days. This boy and his brother frequently ask their mother for methylated spirits to apply to their joints, as they are then able to walk about. The history of these two boys may be regarded as typical of the disease so clearly described at the beginning of last century and named haemophilia. IV. 35, C. G., aged 10, unaffected. IV. 36, M. G., aged 8. IV. 37, I. G., aged 5. This child does not talk; she is "funny in her head" and used to take tabloids but has not done so for several months. IV. 38, died during breech delivery. IV. 48, D. IV. 49—55, all quite unaffected. (See Bibl. No. 884.)

Fig. 428. *E. Martin's Case.* Martin gives a very brief and very imperfect account of three haemophilic families, two of which were related. Of the two he says—and this is his entire description—"In this last family four boys bled to death, whereas four other sons and the daughters are not affected. The only married daughter had however severe flooding. The two mothers (sisters) had one brother, aged 19, who had a tumour of the thigh (Markschwamm) with repeated bleeding, and one sister who bled to death in her first confinement." We have not attempted to reproduce this graphically. The third family is described by Martin in the following terms. "Two small boys suffered from haemorrhage of the lips. Their mother also had haemorrhage from placenta praevia and died." A pedigree is given of "this extensive bleeder family from data obtained from the third daughter." From this it appears that I. 1, G. K., was not a bleeder. II. 1, and II. 2, died of haemorrhage. II. 3, not a bleeder. II. 4, not a bleeder. His descendants, III. 1, also free from haemorrhagic tendency. III. 2, died of phthisis. III. 3, married a "relation." III. 5, III. 6, died of phthisis. III. 11, suffered from "Flechten." IV. 3, died of haemorrhage, aged 4½ years. IV. 10, married his cousin. IV. 12—IV. 15 (sex not given), died of convulsions. IV. 17, died of placenta praevia with first child of her second marriage. IV. 22, suffered from "Flechten." V. 1, a bleeder, no details. V. 2—4, not bleeders. V. 6, died of haemorrhage, aged 1½ years. V. 7, died of convulsions, aged 11 weeks. V. 8, bleeder, 9 years old. V. 11, died of lockjaw. V. 12, a bleeder. The rest of Martin's paper is occupied with general remarks about haemophilia and its treatment. It seems to us that cases recorded in this manner should be rejected altogether as they are without any value. (See Bibl. No. 194.)

Fig. 429. *Poland's Case.* Poland has given the history of two brothers, apparently bleeders. The cases are however somewhat doubtful. They came from Baker's Rents, Rotherhithe Street, London, where they lived in very unhygienic and poor circumstances. Four other children who were born in a healthy neighbourhood had not suffered from haemophilia although all were dead. I. 1, aged 35. I. 2, Irishwoman, aged 40. II. 1, died from "inflammation" when 14 months old. II. 2, died from teething, aged 12. II. 3, died from croup at the age of 2. II. 4, died from some gastric complaint at the same age. II. 5, James R., aged 6½, admitted into Guy's Hospital bleeding from the tongue. He had been looking through a window and while drawing back his head bit his tongue. He bled all day in spite of the application of caustic and nitric acid. The actual cautery however was efficient. Next day he was covered with large purpura-like bruises. II. 6, John R., aged 19 months, admitted into Guy's Hospital in 1849 bleeding from the fraenum of the upper lip, the result of a blow. Lunar caustic arrested the bleeding for two days, but it restarted and continued till pure nitric acid was applied. Six months later he was again in hospital in a similar plight. (See Bibl. No. 196.)

Fig. 430. *Theinhardt's Case.* Theinhardt, of Wald (a village between Cologne and Elberfeld) communicated a case of haemophilia to Nasse. I. 1, N., of Wald, healthy. No family history of haemophilia. I. 2, no history of bleeding except one attack of menorrhagia. All her children appeared strong and healthy at birth. II. 1, a male, was suckled for 18 months. Any knock produced a large bruise. When nearly 3 he fell on the street and, injuring his R. thumb with a knife, bled to death. II. 2, died of consumption at the age of 2. According to the parents he was not a bleeder. II. 3, bled to death at the age of 2 following a bite of his tongue. II. 4, died at the age of 4 from scarlet fever. He was easily bruised, but had never bled. He was never injured. II. 5, a bleeder. At 3 he had epistaxis and bled to death at the age of 8 during his second dentition. III. 7, 8, 9, living. (See Bibl. No. 42.)

Fig. 431. *Wightman's Case.* Wightman, of Liverpool, has given a short description of a large family of male bleeders, the descendants of one Pieterson, I. 1, who is said to have been "subject to bleedings"; no other details. He married I. 2, his first cousin, of whom nothing else is stated. II. 2, Mrs G., not a bleeder. II. 3, bled to death at the age of 30 from a cut on the hand; not married. II. 5, female, died aged 60, not a bleeder. II. 6, Mary Ann P., not a bleeder. III. 1, P. C., bleeder. Swelling of elbows and knees; bled to death from the bowel, aged 35. III. 3, Jas. C., died of epistaxis, aged 29; not married. III. 4, female, not a bleeder. III. 6, male, bled to death; no details. IV. 1, Jane, aged 52, not a bleeder. IV. 4, Mrs I., married first IV. 3, David G., and secondly IV. 5, David I. She was not a bleeder, but by each husband had one alleged bleeder. IV. 6, Jessie, aged 48, healthy. IV. 8, Mary, died of haematemesis or haemoptysis *post partum*. IV. 10, William, not affected. IV. 12, Jemima, aged 48, unaffected. IV. 14, Wilhelmina, aged 40, not a bleeder. IV. 16, Robert S., a bleeder, had swellings of knee and arm and died at the age of 21 from gangrene, following an injury to his knee. V. 5, Peter G., suffered from epistaxis. V. 6, David I., aged 13, a bleeder, admitted into the Infirmary for Children, Liverpool, Nov. 28, 1893, with L. knee distended and discoloured. He bled from teeth and

from cuts. V. 11, died from epistaxis. V. 16, William, aged 14, suffered from epistaxis and swellings of the knee. V. 21, John G., aged 11, suffered from epistaxis, haematomata and swelling of the knee. It is unfortunate that the details in reference to this family are not given more minutely. (See Bibl. No. 617.)

PLATE XXXIX. Fig. 432. *af Schultén's Case.* af Schultén has published a short account of a doubtful case, III. 6, of haemophilia from Finland, and he also gives a pedigree of the family in which five of the members are described as bleeders. No details of their condition are given. Kolster (see Bibl. No. 630) has also published a pedigree of this family completed, as he says, by af Schultén. Kolster has added all the individuals in the fourth generation. He has however left out II. 17, II. 18, II. 19, II. 20, given in af Schultén's pedigree. No description of any of the cases is given by Kolster. I. 1, 2, 3, stated to be healthy. I. 2, "A. P.," dead, married I. 1, M. W., now deceased, and then I. 3, Sofia A. II. 1—II. 6, healthy. II. 7, not a bleeder. II. 9, II. 16, and II. 20, marked in af Schultén's pedigree as bleeders—no data given except that two got well while young; one still suffered. III. 6, Ragnar H., licentiate, seen by af Schultén for persistent bleeding from a tonsil. His previous history showed that he had had severe haemorrhage after a tooth extraction. The onset of the present illness was angina followed by ulceration of tonsil, the ulcer bleeding for about five weeks, when it ceased spontaneously. No other data. IV. 3, marked a doubtful case in Kolster's paper. We consider this case altogether a very doubtful one of haemophilia, no data being presented which could justify this diagnosis. (See Bibl. Nos. 465 and 630.)

Figs. 433, 434, 435, 436 and 437. *Dunn's Cases.* Dunn republished, in 1883, the history of a series of families which had already been made public towards the beginning of the 19th century, adding numerous details, which he had himself collected. *Case I.* Fig. 433. The fourth family described by Dunn was resident in Philadelphia. Some of the members had been previously described by W. M. Holton (see Bibl. No. 345). In this family the daughters of I. 1, a member of a bleeder family, propagated the disease through their daughters, III. 2, and III. 5 (*owing to a slip, the rule from II. 3, and II. 4, which goes to III. 4 in our pedigree, should be to the* female *III. 5*), to a large number of their male grandchildren. The males did not transmit the disease, whether affected or not. It is stated of II. 4, that the history of her parents' family, I. 1 and 2, could not be accurately determined, but that it was known that several were bleeders and some had died therefrom. I. 2, his name was F. II. 2, was not affected. II. 3, one B., came of a family affected by tuberculosis. Of their children, it is stated that several, III. 3, died in infancy of causes unknown, and that III. 5 was the only survivor (*owing to a slip, the rule from II. 3, and II. 4, which goes to III. 4 in our pedigree, should be to the* female *III. 5*). III. 4, was named C. (*owing to a slip, the rule from II. 3, and II. 4, which goes to III. 4 in our pedigree, should be to the* female *III. 5*). III. 6—9, were living, not affected; but III. 10 was dead of a cause not stated. IV. 1, and 2, it is stated that the males were bleeders and that the females were exempt. The order of the 13 children, IV. 3—6, 8—16, is open to doubt, but that of IV. 3—6, 8—10 is probably as shown in the pedigree. IV. 3, in infancy this boy sustained an effusion of blood under the scalp. This was punctured and required suturing to stop the haemorrhage. At the age of 5 haemorrhage started as the result of a slight injury to the toe. The bleeding was checked, but the toe swelled up and, on separation of the slough, a frightful haemorrhage started and induced death in two days. IV. 4, died of umbilical haemorrhage lasting 15 days from birth. In the case of IV. 5 the tendency was manifest from birth. Every cut or abrasion produced profuse and long standing bleeding. On one occasion he injured the fraenum of his upper lip and the blood flowed till all its colour vanished. A deep sleep ensued and he slowly recovered, but was left with a partial paralysis of the left side. At the age of 22 he slipped and ruptured a blood vessel in an attempt to save himself. The bleeding was internal and death ensued. In the course of his life epistaxis had been frequent and he had been much troubled with swollen joints. The haemorrhages of IV. 6 had continued from birth, but were not severe. He died of haemoptysis at the age of 21. IV. 8, showed the symptoms early. Frequent epistaxis and ecchymoses. He once bled badly from his gums, which had been injured by a tooth-pick, and again from an injury to the fraenum linguae. After a tenotomy of the tendo Achillis he bled severely for six days. He died of haematuria at the age of 29. IV. 9, manifested chiefly rheumatism of the knee and elbow joints. Sometimes his entire leg swelled up and became purple. These attacks were very painful but of short duration. IV. 10, is stated by Dunn to have been affected early, but he went West and was lost sight of. Holton states that Harry C. was seen by him in 1870 at the age of 14. He had scratched his hand slightly with a saw and there was continual and uncontrollable oozing of blood. The wound five days after the accident was the size of a "silver dime" and "disposed to slough." The whole hand was swollen and the bleeding defeated the efforts of other doctors. As the sloughing extended, it was thought that the blood was derived from the *arteria superficialis volae.* The actual cautery was applied but the blood was not arrested till the 24th day after the accident. The rest of the family was not affected. (See Bibl. Nos. 345, 459.)

Case II. Fig. 434. This is also one of Dunn's own cases, occurring in the hospital of the University of Pennsylvania. E. K., II. 2, a labourer, was aged 26. The haemorrhagic tendency started when he was a small boy, and on two occasions he bled profusely for hours from slight wounds. Several times he nearly died of epistaxis. Trifling blows would give rise to enormous swellings, which rapidly became black. Rheumatism with swelling of the right knee and shoulder is also recorded. He came to hospital with a lacerated finger which required amputation. At the operation the bleeding was profuse, and next day

the haemorrhage was so alarming that many ligatures were applied. A few days later, suppuration having occurred, an incision was made into the palm, and the resulting haemorrhage was great. This was also the case on a second occasion. The family history of this man is not of much value. I. 1, died of haemorrhage into the lungs. I. 2, was alive but had been a bleeder all her life, both spontaneously and as the result of injury. II. 1, died of croup at an age not stated. (See Bibl. No. 459.)

Case III. Fig. 435. The third family described by Dunn is that originally published by William Pepper, in 1881. (See Bibl. No. 443.) A tendency to haemorrhage, usually manifested as epistaxis, occurs in five generations in males and females. In those cases which came under the observation of Dunn a local lesion was demonstrated in the nose. The family, resident in Lancaster County, U.S.A., starts with M. S., I. 1. There is no information about his relatives. Epistaxis and haemorrhage after slight injury are said to have occurred from infancy. He died of haemorrhage (site not stated) at the age of 50. His wife, I. 2, was subject to epistaxis. Their elder daughter went West and was lost to sight, while the younger, M. S., II. 2, bled from childhood chiefly from the nose. II. 3, her husband, L. B. III. 1, Benjamin, started to bleed in infancy from small ulcers on the margin of the tongue and in the nose. The ulcers disappeared at puberty and with them the diathesis vanished. III. 3, Anna, early bled from nose and gums. Her husband was Mr S. III. 5, Catharine, suffered from attacks of epistaxis from birth, so that at times her life was despaired of. She died at the age of 32 of a cause unknown to Dunn, but stated by Pepper to have been "spinal disease." Her husband, Mr H., was healthy. His son, IV. 15, to be described shortly, is given by Dunn the initials C. S. B. Of III. 7—11, there is no information, and of IV. 1—8, none except that they were unaffected. IV. 9, is stated to be a bleeder. IV. 13, Mary, who married IV. 14, W., was not affected. IV. 15, C. S. B. mentioned above, is the chief case in the family. He was a farmer aged 43, and had suffered from epistaxis almost daily from birth. Tooth extraction had also occasioned severe haemorrhage. He had had rheumatism of the left knee. On examination ecchymoses were found on his face and inside the mouth. Two small round ulcers were present on the middle turbinate bones, which bled profusely on removal of the scab. Dunn says that his liver was enlarged, but Pepper recorded the opposite, stating that he was sober and moderate. The latter observer further added that he once bled without cause from the lip. With regard to IV. 17, it is stated that one of the sisters of IV. 15 died of a cause unknown and had frequently suffered from epistaxis. V. 3, aged 7, had suffered from dangerous epistaxis from birth. She also was found to have ulcers on the turbinates. V. 4, Harvey, aged 11, had bled from birth. His brother Jefferson, V. 5, aged 9, was not so affected, while his sister, Anna, manifested epistaxis. It is stated that there was no case of gout, scrofula or tuberculosis in the family. Although some readers may consider some of these cases to be haemophilia, we are not disposed to admit it. We prefer to direct comparison with B. G. Babington's family (see Bibl. No. 277) and with the condition referred to in Osler's "Principles and Practice of Medicine," London and New York, Ed. 6, 1905, p. 749. (See Bibl. Nos. 443, 459.)

Case IV. Fig. 436. Dunn's last family resided in Chester and Delaware Counties. Gideon Humphrey, one of the doctors who saw the cases, had previously published a few admittedly immature observations (see Bibl. No. 39), while Reynell Coates had given a valuable account (see Bibl. No. 51). In a later publication, Dunn returned to an account of the descendants of IV. 12 (see Bibl. No. 599). In this family I. 2, a woman supposed to be of a bleeder stock, propagated the disease to four grandsons through her unaffected daughter and to her son, II. 10. This son appears to have transmitted it through his daughters to several grandsons, and through one of these grandsons, IV. 12, through a great grandson, V. 2, to two great great grandsons, VI. 4, and 6. The tendency to bleeding was thought to have originated in the family of I. 2. Her husband, I. 1, was named W. II. 1, was named G. II. 10, the man who appears to have transmitted the disease, was R. W., "a member of the medical class," of Delaware County, Penn. No information is given before the age of 11, when he cut his thumb with a penknife. The bleeding was stopped after several days by the actual cautery. This method was adopted to arrest the continual oozing from the gum after tooth extraction later in the same year. At the age of 24, after removal of another tooth, the bleeding lasted 10 days, in spite of the efforts, fully described, of several doctors. The blood could not have been less than three gallons. While engaged on his father's farm R. W. had been several times wounded with a sickle: once badly in the hand, but the haemorrhage in this case was not remarkable, though other less severe wounds required the application of the cautery. Shaving cuts did not bother him. He died at the age of 42 of "rheumatism of the heart." III. 1, John; at the age of 15 months he fell and ruptured the fraenum of the upper lip, which occasioned severe haemorrhage. When 10 or 13 years old he was struck by a stone on the forehead. The surgeon was unable to stop the bleeding, so he ligated the temporal artery and the child died. His brother Ober, III. 2, is variously described. Dunn says he was "all out of shape and troubled with rheumatism," and before his death at $2\frac{1}{2}$ years from a bitten tongue had shown a tendency to bleeding. Coates says he died at 18 months from a ruptured fraenum of the upper lip. III. 3, Milton, was a bleeder from infancy and died at the age of 28 of cerebral haemorrhage, the result of a fall. With regard to III. 3, or III. 4, it is related that a slight contusion of the scrotum produced a great extravasation, and that after a wound of the thumb the blood flowed for several days, the haemorrhage being suddenly arrested by a shower bath. It does not appear which of these two boys sustained these injuries. The boy III. 4, Worral, had bled from birth spon-

taneously from the nose, kidney and bowel. He had rheumatism of the right knee. This joint, after exposure to cold, would swell, become painful and undergo discolouration. The tendency to haemorrhage disappeared at 30 and he died at 63 of other causes. IV. 1, represents a number of children all dead from children's complaints. IV. 2—5, were unaffected. IV. 6, bled from the gums and suffered from "joint trouble." He died at the age of 4 of haemorrhage from a bitten tongue. IV. 9, Mordecai, exhibited the haemorrhagic tendency and died at the age of 40 of haematemesis, as also did his brother, George, IV. 10, at the age of 20. IV. 11, Davis, exhibited similar tendencies and died after venesection at 12. IV. 12, not affected. V. 2, not affected but suffered from rheumatism of the joints. VI. 1, 2, 3, 5, 7, all unaffected but rheumatic. VI. 4, older than VI. 6, aged 15, suffered from epistaxis and severe haemorrhage after trivial wounds. VI. 6, Robert L., aged $8\frac{1}{2}$, was described and figured in Dunn's last publication. He was rheumatic also and suffered from epistaxis and severe traumatic haemorrhages. At the age of $3\frac{1}{2}$ an abscess bled severely after being opened, and tooth extraction had occasioned bad bleeding. When examined his lungs were congested. He had pains in the knees and ankles. His right ear was discoloured and he was covered with purpuric spots on skin and mucous membranes. The discolouration of the ear passed into a remarkable tumefaction of the whole face. T. 102·4 (Photo.). (See Bibl. Nos. 39, 51, 459, 599.)

Case V. Fig. 437. Dunn's first family, residing in New Jersey, was hitherto not published. Three females and one male are alleged to be bleeders and children of a bleeder mother. I. 1, and 2, were both healthy, as also were all the persons in generation II. II. 1, and 4, are described as Mr W., and Miss J. who were sprung from healthy parents. III. 1, the mother of the children, was named Anna, and was 35. Her haemophilic tendency was lost at the age of 12, and was first noticed when her gums were lanced. Epistaxis was frequent and sometimes alarming, while cuts and scratches were followed by profuse haemorrhage. Her husband's initials were J. H. A sister, III. 3, aunt of the bleeder children, was not affected. Martha, IV. 1, started to bleed at 2 months: she died of acute enteritis following an attack of epistaxis in her 11th month. Lizzie, her sister, IV. 2, had repeated attacks of epistaxis and died therefrom at 2 years, while another sister, a delicate child, died of it at 3. The only brother was IV. 4; he had one haemorrhage from the gums "last March." He was 4 years old. We cannot accept epistaxis alone and without local examination, as a sign of haemophilia, and in the case of the mother the references to profuse haemorrhage are purely relative. We, therefore, consider that this family does not present sufficient evidence of haemophilia. (See Bibl. No. 459.)

Fig. 438. *Vanderveer's Case.* I. 1, and 2, stated to be healthy. II. 3, alive, a carriage trimmer by occupation. Has always suffered more or less from epistaxis. Otherwise well. II. 4, J. D., a painter by occupation. Began to suffer from haematuria at the age of 30 and it lasted for years. Came under Vanderveer at the age of 45 still suffering from haematuria. No evidence of stone; ultimate recovery; death at the age of 70. P. M., no marked changes, kidneys looked perfectly normal. II. 5, normal. III. 1, Mr W., married to III. 2, she being then 24. No excessive bleeding at confinements. III. 3, Mr J. W., married III. 4, she being then 20. III. 5, R. D., alive, aged 40, had epistaxis. III. 6, his wife. IV. 1, not a bleeder. During teething got convulsions and hemiplegia, and finally became an epileptic. IV. 2, not a bleeder. IV. 3, a bleeder. When nearly 3 years old fell on a "tin bean blower" and injured his tongue. Uncontrollable oozing took place and he died on the 14th day. IV. 4, at the age of 20 months while teething a physician lanced his gum. Constant bleeding followed and he died on the 8th day. IV. 5, strong and healthy. IV. 6, when one year old had severe haemorrhage from a slight cut on the finger. Died of spinal meningitis aged 14 months. IV. 7, aged 9 years. Had had three or more attacks of swelling of both knees which the mother believed was rheumatic. Vanderveer however thought it was effusion of serum resulting from the haemorrhagic diathesis. IV. 8, sex not stated; died in infancy but not of haemorrhage. IV. 9, at the age of 5 received a wound on the left frontal region; haemorrhage at first was free but was controlled. Secondary haemorrhage then set in and could not be stopped; death about the 14th day. IV. 10, at $2\frac{1}{2}$ years bit his tongue; gradual haemorrhage; death on the 8th day. IV. 11, sex not stated; died of haemorrhage. Vanderveer was unable to learn particulars as mother had removed to an adjacent State. (See Bibl. No. 578.)

Fig. 439. *Brigstocke's Case.* I. 1, died early of consumption. I. 2, died at the age of 76. I. 3, and 4, died of dropsy. II. 1—4, healthy. II. 5, aged 41. Weakly, occasional epistaxis. Nearly died of haemorrhage from the rupture of a varicose vein. II. 6, aged 39, healthy. II. 7—10, died of dropsy. II. 11, and 12, weakly. II. 13, healthy. III. 1, H. C., aged 12. Suffered in a manner similar to III. 3, with haematemesis and haematuria. Died from internal haemorrhage distending the abdomen 10 years before. III. 2, A. C., aged $3\frac{1}{2}$, was constantly afflicted with epistaxis. He died 12 years before, apparently from haemorrhage from the gums. III. 3, H. C., aged 13. Under the care of Brigstocke. At birth large extravasations of blood were found over each shoulder. Shortly afterwards an effusion occurred into the ankle joint. At 8 months profuse epistaxis. Ever since, at intervals, effusions into joints, ankles, knees, elbows, wrists and small joints of fingers and toes. Extravasations of blood all over his body, long walks producing discolouration of the calves. No haematuria and no haemoptysis or haematemesis. Profuse haemorrhage occurred after tooth extraction. He had been twice vaccinated without ill results. III. 4—9, healthy. (See Bibl. No. 323.)

Fig. 440. *Krimer's Case.* Krimer communicated to Nasse the history of a bleeder family living in

Saxony. I. 1, and I. 2, no information. II. 1, 2, 3, suffered from pains in the joints and haemorrhoids, but were not bleeders. II. 4, died of consumption at the age of 24, frequently suffered from epistaxis after the slightest shaking such as during running. Bleeding lasted as long as seven days. At 22, he had painful joints and started piles. II. 5, died of apoplexy aged 59; of hasty temperament; an officer. II. 6, female, lived to a fair old age; was not a bleeder. III. 1, Herr von K. III. 2, aged 60, living, informant. III. 3, and III. 4, healthy. III. 5, healthy. III. 6, died of "nervous fever" at the age of 20. III. 7, a bleeder, cut himself between the thumb and index finger and bled to death in a week. III. 8, a bleeder, fell on his nose at the age of 7 and bled to death in a week. III. 9, died after a small superficial cut on the left hand. III. 10, bled to death from the nose at the age of 9. III. 11, bled to death after the removal of a tooth at the age of 7. III. 7—III. 11, were all well made. IV. 3, died of convulsions, aged 3 days. IV. 4, bled to death at the age of 7 after falling on his face and knocking out a tooth. He suffered from epistaxis and pains in the joints. After a slight blow he was observed to develope a painful blue swelling. IV. 5, affected in a manner similar to IV. 4, bled to death from a deep cut on his L. index finger. IV. 6, aged 36, a mechanic, living. As far as he could remember he began to bleed about the age of 5. Between the ages of 7 and 9 he was severely affected, and later at the age of 13. He would bleed from cuts for 8—12 days until he was in an extremely anaemic condition. From 7 onwards, had pains in the joints, especially in hot weather. At 18 had pain in the kidney region associated with haematuria for eight days. This recurred every month, but not so badly. About 22 tooth-ache drove him to have a tooth extracted. Although the operation was carried out skilfully great haemorrhage ensued for nine days, until he was given up for dead by many consultants who had been called to see him. The relatives however called in a man who practised "a sort of magnetism." He made passes over the dying man's face and then called on God, whereupon the blood was instantly stilled. After chewing something hard however haemorrhage began again, but it was immediately arrested by the magician for a second time. Two years later he married, and shortly after had another attack of pains in his joints and haematuria. He no longer however bled so much from wounds, but still bruised readily. At 28 he began to suffer from piles, and noted that when they were not bleeding his joints began to be painful. Severe cuts no longer caused bleeding as formerly. IV. 8, started haematuria at 2 years and died at 5. IV. 9, had severe signs of "gout." At the age of 13 he fell on his face and bled to death from the nose after 7—9 days. IV. 10, living, aged 18; a thin and pale nervous subject. At 7 had pains in his joints. He was easily bruised and suffered from epistaxis. IV. 11, died of convulsions a few days after birth. IV. 13, and IV. 14, healthy, 28 and 20 years old respectively. V. 1, V. 2, V. 3, aged 5, 4, and 2 years, healthy. V. 4, V. 5, V. 6, no information. (See Bibl. No. 33.)

Fig. 441. *Schrey's Case.* Case in the author's own family living probably at Mühlfurt (Rhein Preussen). I. 1, and I. 2, no information. II. 1, P. Kamphausen, suffered from grave and almost fatal epistaxis. II. 2, was wont to bleed for several days from small wounds, but lived to old age. II. 4, no information. III. 2, died at the age of 84, having suffered from menorrhagia at the climacteric. III. 4, M. K., suffered from very severe bleeding from the gums, which the actual cautery could not quell. III. 5, suffered from epistaxis at the climacteric. She was once venesected for epistaxis and blood came from a broken-down scar. Died of phthisis at the age of 66. IV. 4, Henrica Dürselen, no data. IV. 5, S. (Wilhelm Schrey). IV. 6, girls, free, number not stated. IV. 7, A. S., slight injuries produced prolonged haemorrhage. Light pressure induced ecchymoses, and after tooth extraction bleeding lasted for weeks. IV. 8, male children suffering from various haemorrhages, number not stated. V. 1, no symptoms of haemophilia except occasional profuse epistaxis. V. 2, normal. V. 3, the author, J. A. Schrey, born 1833. At 5, shortly after scarlet fever, he developed an inflammatory swelling of the R. hip. An incision was made over the great trochanter without any great bleeding. He used to suffer from epistaxis, and frequently showed ecchymoses and bruises, especially on the extremities. At 12 he fell and sustained a large haematoma on the forehead. At 14 he injured his occiput. It bled and swelled, and after ten days began to pour blood. The bleeding was temporarily arrested, but recurred and continued for four weeks. After a tooth extraction at the age of 21 there was much and prolonged bleeding. Near the site of the wound the gum became spongy and a bleeding granulation tumour formed. A ligature was applied to the mass but without causing the blood to stop. It ultimately ceased spontaneously. From that time he was not troubled with severe haemorrhage. Occasionally, however, after severe coughing or after exaltation with spirituous liquors, a little blood would come from his mouth. As he grew older small wounds no longer troubled him. He had never had affection of the joints except the attack above-mentioned in his hip. V. 4, was also a bleeder. No one in this family ever died of haemorrhage, and it is to be regretted that more information of the other alleged bleeders is not forthcoming in view of the somewhat ill-marked features of Schrey's own case. (See Bibl. No. 237.)

PLATE XL. Fig. 442. *Weil and Boyé's Case.* Slight haemophilia existing in three generations. In most of the cases the evidence of haemophilia is very slight indeed, scarcely meriting the name. I. 1, M. G., bled from slight causes; had epistaxis and haematuria, and grave intestinal haemorrhage; died suddenly at 60. II. 1, II. 2, II. 3, died young. Cause unknown. II. 4, M. G. Joseph, died at 32, leaving a healthy daughter. II. 6, M. G. Simon, 54, not a bleeder, had six children, five of whom (3 ♂ 2 ♀) were dead; the

other, III. 7, was a bleeder. II. 9, Mme M., 56, not a bleeder, but had two sons bleeders. II. 11, Mme V., 46, not a bleeder. II. 13, Mme G., 44, bleeder. Suffered from metrorrhagia without sufficient cause genitally. Menstruated at 14 with periods lasting at first 8 days, latterly 15 days. She lost much blood at her confinements, and on one occasion this was so serious as to require *tamponnement* in the hôpital Necker. She frequently had epistaxis and bruised easily. II. 15, Mme B., bleeder, "elle avait des pertes abondantes à chacune de ses couches." II. 17, Mme B., aet. 30, not a bleeder. II. 16, her husband, died of phthisis at 30. III. 7, lost considerable quantities of blood constantly and without sufficient cause; a bleeder according to Weil. III. 8, a bleeder, died at 6 years; no details given. III. 9, aged 33, suffered from epistaxis and ecchymoses; regarded as a bleeder by Weil. III. 11, a bleeder, died at age of 10 from haemorrhage of bowel in enteric fever; no details of haemophilia given. III. 12, alive healthy. III. 13, died of variola aged 4½ months. III. 14, often had epistaxis when she was young. Latterly she bleeds much "quand elle est contrariée"; often had ecchymoses. Her periods lasted 15 days, although there was no local cause for this prolongation. III. 16, a bleeder, "epistaxis, hemorragies diverses"; died of nephritis at 4½. III. 17, III. 18, died at 4 and 6 months respectively. III. 19, alive and well, aged 14. Two daughters, III. 20, III. 21, were dead; no details. III. 22, 23, 24, dead. III. 25, 26, 27, alive, aged 16, 14, 12 respectively. III. 27, a bleeder; no details. III. 28, Weil's patient, Br... Réné, 4½ years, admitted into hôp. Trousseau with haematuria which had lasted 15 days. Was born at term and was breast-fed. Epistaxis, ecchymoses easily produced and lasted long. On admission, he had a temporo-parietal haematoma and bruises on his legs and thighs. Red blood corpuscles, 3,480,000, leucocytes 4500. III. 29, died of meningitis. III. 30, a miscarriage. IV. 1, recently had haemorrhages, which were difficult to arrest; regarded by Weil as a bleeder. (See Bibl. No. 871.)

Fig. 443. *Weil's Case I.* I. 1, 2, 3, 4, no information. II. 2, II. 3, alive and well. II. 4, aged 65, had suffered all his life from recurring arthropathies and ecchymosis. III. 1, III. 3, alive and well. III. 2, "Ar." A Turk. At the age of 4 fell and cut his lip; bled 25 days. At 8 years joint affection developed in R. knee, with recurrences. Epistaxis frequent up to the age of 12. At 18 and 20 respectively had haematuria lasting 2—4 days. The extraction of a tooth caused haemorrhage for a week. In 1895, after abdominal massage, symptoms of peritonitis ensued, followed by intense melaena. Recovery in eight days. Blood examined by Weil. (See Bibl. No. 826.)

Fig. 444. *Weil's Case II.* I. 1, I. 2, no information. They had 11 or 12 children. I. 3, I. 4, not bleeders. II. 1, not a bleeder. II. 2, a bleeder; had ecchymosis, haemarthroses, epistaxis; died of haemorrhage at the age of 36 as a result of a fall downstairs. II. 4, a bleeder, died in childhood of haemorrhage from wound of tongue. II. 6, not a bleeder, died aged 46 after operation on breast, probably cancer. II. 7—II. 11, no information. III. 1—17, family of II. 6, and II. 12, viz. eight girls and eight boys and one miscarriage. Of this family ten were alive, five being girls not affected with haemophilia. Three girls died young but were not bleeders. Five boys living. Two male bleeders dead, and another son also dead. He was an idiot, and had ecchymoses, buccal haemorrhages and haemorrhages from the ear. Among the living boys one, aged 16, had joint affections and suffered from ecchymosis and epistaxis. These are probably III. 9, III. 12, III. 16, mentioned on Weil's pedigree. III. 14, "Bern"... a bleeder, aet. 26. At 2 years bled for eight days from a bite on the tongue. At 3 had haemarthrosis of left elbow and ankle and both knees. At 4 had epistaxis for one week. Joints attacked every two or three weeks through the whole of infancy. At 13 had haematuria for eight days. At 14 great haemarthrosis of left knee, with consequent ankylosis and atrophy of the muscles. At 17 painful haematuria lasting a month, with frequent recurrences. At 20 had melaena for three days. This family was of German origin. (See Bibl. Nos. 826 and 871.)

Fig. 445. *Weil's Case III.* I. 1, I. 2, no information. II. 2, II. 3, II. 5, stated to have been bleeders; no details. III. 1—4, males, nothing known about them. III. 5, and III. 7, not known to be bleeders. IV. 2, healthy. IV. 3, five or six children dead in infancy; cause of death not stated. IV. 4, healthy, but had cutaneous ecchymoses. IV. 7, healthy. IV. 8, a bleeder, died at 22 years from haemorrhage following tooth extraction. IV. 9, healthy. IV. 10, died of internal haemorrhage at the age of 23. IV. 11, not a bleeder. IV. 12, aged 45, had haemarthroses and haematuria. IV. 14, not a bleeder. IV. 16, a bleeder, died young; no details. V. 1, V. 3, healthy. V. 2, a bleeder, died from cerebral haemorrhage, the result of a fall, age not stated. V. 4. healthy. V. 5, "Marc. Flam.," a bleeder. At 18 months had an enormous haematoma on the head, the result of a blow. It was necessary to incise it, and grave and prolonged bleeding ensued. As a result of a bite on the tongue copious haemorrhage for eight days. He bled from the teeth as they came through the gums. At 7, developed haemarthrosis. At 8, had haematoma on the shoulder. At 9, bled for eight days from a cut. Had frequent cutaneous ecchymoses. V. 6, V. 7, male bleeders; no details. V. 8, not a bleeder. V. 9, healthy, sex not stated. V. 10, not a bleeder, male. (See Bibl. No. 826.)

Fig. 446. *Weil's Case IV.* I. 1, 2, 3, 4, died about 60. I. 5, 6, 7, 8, no information. II. 1, and 2, no information. II. 3, and 4, died very old. III. 1, and III. 2, alive and well. III. 3, died of paralysis at 71. III. 4, alive, aged 81. III. 5, alive, aged 75. III. 6, died of an accident at 35. IV. 1, alive, not a bleeder. IV. 2, Dr B., at 7 developed pupuric scarlatina; at 12 great epistaxis; at 18 bled for eight

days after extraction of tooth. Had ecchymoses. Great haemorrhage followed trivial cuts. Gums bled easily. In August severe renal pain and haematuria. Perirenal abscess. Prior to operation was injected with serum intravenously. At operation, no great bleeding ensued. (See Bibl. No. 826.)

Fig. 447. *Weil's Case V.* I. 1, 2, 3, 4, presumably healthy. II. 2, died from the effects of an accident. II. 3, alive and well, not a bleeder. II. 2, and II. 3, had four children. III. 1, sex not stated, died from an accident during delivery. III. 2, living, aged 14 (16 in Weil's pedigree), not a bleeder. III. 3, had haemorrhage from teeth during first dentition. Bled to death at the age of 7 from a cut in the upper lip, the result of a fall. III. 4, Maurice P., had ecchymoses from birth onwards. Bled from gums at 14 months and 2 years respectively. Five months ago had great frontal haematoma as a result of a fall. It suppurated, and he was three months in the hôpital Trousseau. His blood was studied by Weil. Now aged three. (See Bibl. No. 826.)

Fig. 448. *MacCormac's Case I.* MacCormac has recorded the case of a boy, II. 1, aged 15, who had been an inmate of Saint Thomas's and other hospitals under Simon and Jones. He was admitted for separation of the epiphysis of the femur. It looked as if the skin would burst. Aspiration was performed and serous fluid evacuated. Black bruises appeared on his arms from merely leaning on an iron bed. At the age of 2 it was recorded that his whole face became black as the result of a fall. Later he had swellings in most of his joints. A fall from a chair resulted in a haematoma extending from the thigh to the knee. His father died before he was born, but the mother married again and had three or four healthy children. (See Bibl. No. 355.)

Fig. 450. *MacCormac's Case II.* I. 1, and I. 2, no data. I. 3, Robert Chubb, aged 70. I. 4, Eliza Chubb, aged 70. Both healthy. No history of haemophilia. II. 1—II. 11, not affected with haemophilia. II. 12, James Burril, aged 41, healthy, not a bleeder. II. 13, Robert, aged 45, healthy. II. 14, Fanny, aged 39, healthy. II. 15, Frederick, aged 38, healthy. II. 16, Elizabeth, aged 34, healthy. II. 17, Joseph, a bleeder, continually bled from nose and gums, and was very easily bruised. At the age of 4 he stepped off a chair and sustained a small cut on his head. From this he bled to death in 24 hours. II, 18, Tom, a bleeder. He nearly died on several occasions from small cuts. He also had swollen knees. At the age of 17 he had a tooth extracted and bled to death on the 11th day. II. 19, Eliza, aged 30, and II. 20, Sarah, aged 20. Both healthy. III. 1, Joseph, aged 19, not a bleeder. III. 2, Eliza, died of bronchitis at the age of 1 year. III. 3, Walter, a bleeder. Epistaxis, from which he nearly died. Easily bruised. Bled profusely from the gums. Aged 15. III. 4, Alfred, aged 13, a bleeder, was the patient under MacCormac. Bled from the nose and teeth. Nearly bled to death at the age of 7. Bruised easily. Haemarthroses, etc. III. 5, Emily, healthy. III. 6, William, a bleeder. Bit his tongue and bled three weeks. Bled to death at the age of 3 from cut with scissors. III. 7, Fanny, aged 4, healthy. III. 8, Annie, aged 2, healthy. III. 9, Ada, aged 14 days, healthy. (See Bibl. No. 355.)

Fig. 451. *MacCormac's Case III.* I. 1, and I. 2, no data. I. 3, Charles Eaton, aged 60, a carpenter. Bled for more than a fortnight after a tooth extraction. Often cut himself at his work and always bled freely. II. 1, Charles, aged 35, used to suffer from epistaxis when young. II. 2, Edwin, aged 32, carpenter, frequently cut his fingers and bled easily. From a superficial chisel wound he would lose a teaspoonful of blood or more. Epistaxis frequent. II. 3, Emma, aged 30. II. 4, Eliza, aged 25, bled for a week when she had a tooth out. Had menorrhagia and haemoptysis. Once nearly bled to death from a tooth. II. 5, Louisa, aged 20. A year ago she vomited a large quantity of blood at the menstrual period. II. 6, not a bleeder, wife of II. 2. II. 7, healthy. II. 8, died of consumption. II. 9, and II. 10, suffered from profuse epistaxis once a month. II. 11, and II. 12, well. II. 13, several members of the family who died in infancy. III. 1, Edwin, aged 6, the case seen by MacCormac. During the past two years had epistaxis three or four times. Bruised very easily. When cut or scratched bled easily. From the scratch of a pin several drops of blood were lost. This boy was brought to the hospital with "a tense, hard, freely moveable tumour...over the seventh rib on the L. side and just beneath the nipple...no discoloration of the skin, no pain or tenderness. The tumour was sharply defined and the bulk of half a walnut. Only on enquiry into the history was it possible to say what the swelling consisted of." The cause of the swelling was a blow from a pencil and must have been slight, as nothing was felt at the time. III. 2, Harry, aged 4, had bled freely from the nose two or three times. Was easily bruised and if cut bled a good deal. III. 3, Eliza, aged 1. III. 4, an infant, nearly died during its birth. The "diathesis" in this family appears to us so slight as not to justify the diagnosis of haemophilia. The account of the swelling on the side of III. 1 is unlike that of a haematoma and its subsequent behaviour is not stated. (See Bibl. No. 355.)

Fig. 449. *J. A. Milne's Case.* Dr Milne has communicated to us an unpublished record of a bleeder, III. 10, who was under his care in H. M. Hospital, Stepney Causeway, London. Further data were supplied by II. 5, and III. 5, living in Cardiff, but it was impossible to trace the disease in the ascendants. All the members in the first two generations were stated to be healthy. III. 8, cut his gums slightly and bled to death at the age of 2½ years. III. 9, cut his lip and bled to death at about the same age. III. 10, a bleeder. His uncle, II. 5, informs us that III. 10 was afflicted with the disease from childhood. He would go to bed at night in perfect health but next morning would awake covered with bruises for which

no cause could be assigned. Haemorrhages from mouth and nose were constant and lasted for days. Later, the intervals between the haemorrhages became longer. He grew very quickly but at frequent intervals was unable to walk (haemarthrosis?). The haemorrhages and stiffness of legs did not occur simultaneously. He attended the Cardiff Infirmary from the ages of 8—12. When 15 years old, in 1904, he came under Dr Milne's care in Dr Barnardo's Homes, in which he remained until his death in 1909. During this time he was repeatedly under treatment for haematomata, bleeding from the gums and after tooth extraction. On one occasion he had blood in the stools. In four years he had to be admitted into the hospital eight times. In Feb. 1905 he developed a tuberculous focus in the head of the tibia. An operation was performed and great haemorrhage resulted. In May, 1907, another tuberculous focus developed in the lower end of the femur, and again great haemorrhage resulted, the carious cavity being plugged with wax. The dressings were constantly soaked in blood and he nearly lost his life. Calcium chloride and adrenalin were used with success. Nearly all his joints became involved at one time or another, the clinical picture being one of acute rheumatism. Ultimately stiffness set in and he was crippled. Finally, in 1909, he was admitted with vague symptoms of abdominal trouble associated with pain and tenderness. The day before he had been perfectly well. Suddenly, however, he became collapsed with very small rapid pulse, intense anaemia and thirst, and he died next day. The autopsy revealed an enormous retroperitoneal haematoma enveloping the left kidney and extending across the vertebrae to the right kidney it reached down to and half filled the pelvis. The origin of the bleeding could not be detected and subsequent enquiry failed to elicit any history of trauma. With the exception of a slight cirrhosis of the kidneys his organs and tissues appeared to be perfectly normal. (See Bibl. No. 897.)

Fig. 452. *von Limbeck's Case.* I. 2, female, died of marasmus, aged 75, said to have been a bleeder and to have first shown the symptoms at the age of 60. In that and in subsequent years she had epistaxis till she died. I. 1, died of marasmus after pneumonia. II. 1, and II. 2, not affected. II. 5, had epistaxis often lasting for months, married at the age of 16 to II. 6, and by him had 14 children. III. 1—4, not affected. III. 5, ♂, not a bleeder. III. 7, ♀, aged 47. Subject to bleeding. III. 9, mild attacks of bleeding till he was 16 when he was no longer affected. III. 11, ♀, a severe bleeder since 12 years of age, no data. III. 13, a very severe bleeder, violent attacks of epistaxis since she was 7. III. 15, ♂, frequent attacks of epistaxis. III. 17, not a bleeder. III. 19, a bleeder, died aged 16 of cerebral haemorrhage. III. 20, aged 24, mild bleeder, no longer affected. III. 21, now 16, from 6—14 years severe attacks of haemorrhage. IV. 1—3, not affected. IV. 4, aged 33. Suffered from epistaxis. IV. 6, IV. 7, mild bleeders. IV. 8, a severe bleeder. This case, which has frequently been referred to, does not in our opinion bear the stamp of genuine haemophilia. (See Bibl. No. 572.)

Fig. 453. *Broca's Case.* In the course of a paper on the arrest of haemorrhage in haemophilia Broca refers to a family in which three boys were afflicted. I. 1, and I. 2, no details, presumably normal. II. 1, male, was under Broca's care for "chronic arthritis of haemophilic character," and while under observation developed haematuria and melaena. A history of previous bleeding was also obtained. II. 2, male, aged 14, sustained great haemorrhages after injuries and also bled from the gums after tooth extraction. He was under Broca for great haemarthrosis of R. elbow. II. 3, a bleeder, no details. Broca also describes the case of a male bleeder aged 8, but does not refer to the family. When two years old he was observed to have spots and he also had repeated haemarthroses in his joints. Great and prolonged bleeding occurred from slight wounds. He was treated by serum with beneficial results. (See Bibl. No. 829.)

Fig. 454. *Stoehr's Case.* I. 1, 2, 3, 4, healthy, no tendency to haemorrhages. II. 1, 2, healthy. II. 3—7, five brothers of II. 8, healthy, not bleeders. II. 8, J. Sch., a forester of Gossweinstein, always healthy, no disposition to haemorrhage. Married to II. 9 in his 50th year, died of senile marasmus at the age of 76. II. 9, Barbara N., strong, healthy woman, married at the age of 20 to II. 8, died of pulmonary phthisis, aged 56. Menstruation regular. II. 10—14, two brothers and three sisters of II. 9, of whom it is specifically stated that there was no disposition to haemorrhage. III. 2, 4, 6, 9, 11, all healthy. Menstruation appeared at the usual time and was regular. All married young and, with the exception of III. 9, all the families were healthy. III. 9, Margaretha, born when her father was 59 and her mother 29. She was of medium size, slender and gracile, with brown hair and a fine transparent white skin. She began to menstruate at 15 but was at first chlorotic. She married III. 8, B., at the age of 20, he being then 25, and by him had six children. In the first five pregnancies she lost no excessive amount of blood, but in her sixth confinement she had placenta praevia perfecta and died of rupture of the womb, aged 33. III. 8, her husband, big, well nourished man, who always enjoyed good health till six years before, when he began to be troubled with gout. III. 7, his three brothers and sisters, healthy. III. 12, 13, 14, bleeders. From their earliest years showed bruises, ecchymoses and suggillations, and frequently copious and even dangerous haemorrhages. III. 12, bled to death from socket of tooth at the age of 9. III. 13, received a fall with consequent trivial wound of the gum and bled continuously to death, which occurred on the tenth day. He was eight years old. III. 14, slightly cut his lip and bled to death, aged 5. III. 15, families of II. 10—14, number not mentioned, but stated to be healthy. IV. 1, 2, 3, healthy families of III. 2, 4, 6. IV. 4, born healthy and was breast fed. Soon after birth livid spots of changing colour were observed upon him. At the age of 18 months fell from a table and received a slight cut on the gum. Bled very

profusely for five days. The actual cautery caused a temporary arrest but haemorrhage recurred and he bled to death—no autopsy permitted. IV. 5, showed bruises and suggillations like IV. 4, then an enlarged mesenteric gland appeared with diarrhoea mixed with blood, finally he evacuated pure blood and died aged 3 months. IV. 6, alive, aged 16, not a bleeder, had a tooth out in 16th year and no unusual haemorrhage followed. IV. 7, born in the seventh month, died of atrophy aged 6 weeks. IV. 8, died of convulsions shortly after birth. IV. 9, sex not stated. With this child the mother died. IV. 10, healthy family of III. 10, and III. 11. (See Bibl. No. 188.)

Søren Hansen's Cases. Søren Hansen gives a short account with pedigrees of four haemophilic families in Denmark, two living in Zealand, the third in Laaland and the fourth in Funen and Jutland. Hansen does not appear to have seen the haemophilic individuals himself.

PLATE XLI. Fig. 455. *Case I.* Besides the pedigree given by Hansen it is stated that II. 3, a male bleeder, died at the age of 12 from a perfectly trivial wound of the lip. III. 7, suffered from copious and frequent epistaxis and bled to death at the age of 6, from the leg, during desquamation following scarlatina. IV. 8, had shown unequivocal signs of haemophilia—no details, the same statement being made of IV. 15, V. 4, and V. 7. It is stated that none of the females suffered from haemophilia, although III. 13 died of post partum haemorrhage. III. 16, a female, died of apoplexy. III. 8, suffered from rheumatism.

Fig. 456. *Case II.* A family with three male bleeders in two generations. Females all healthy. III. 8, fatal epistaxis at the age of 6. III. 10, aged 37 years, stated to have been a definite bleeder. IV. 2, a bleeder; no details. III. 6, strong and healthy, but all her children, IV. 1—5, small and weakly.

Fig. 457. *Case III.* Described by Søren Hansen as a case of multiple congenital haemophilia. I. 1, and I. 2, no information. I. 3, and I. 4, healthy. II. 2, was scrofulous as a child and began to menstruate early but without abnormality. She married a healthy man, II. 4, of healthy race, and by him bore eight living children, five boys, three girls, two miscarriages, III. 9, and one stillborn boy, III. 10, who was premature (placenta praevia totalis). All the boys were bleeders in excessive degree, three dying within the first year. Two were still alive aged 11 and 10 years respectively. One daughter died a few days after birth, the other two were alive and aged 16 and 8 years respectively. They were rather weak but had never shown any signs of haemophilia.

Fig. 458. *Case IV.* The origin of the disease in this family was uncertain. I. 1, suffered from habitual epistaxis from which he is said to have died at the age of 63. I. 2, died from pulmonary disease (no haemoptysis) at the age of 51. It was impossible to trace any haemophilia in the families of I. 1, and I. 2. Of their four children one daughter, II. 3, suffered from bleeding from the bowel, and one son, II. 7, when young had epistaxis, but did not show any definite sign of haemophilia. III. 5, died from epistaxis at the age of 5. III. 6, bled to death from a wound on the hand, aged 2 years. III. 7, regarded by Hansen as a bleeder on the ground that he died of phthisis with haemoptysis at the age of 18. III. 16, nine years old, had been a bleeder since childhood, no data. III. 17, bled to death from the bowel when 9 months old. In the third generation in this family it is stated that six of the sons died in the first year of life, although showing no signs of haemorrhagic diathesis. All the daughters were healthy. We consider these cases to be of little value. (See Bibl. No. 503.)

Fig. 459. *Ledoux's Case.* Detailed history of a male bleeder, IV. 2, with incomplete account of haemophilia in two other males on maternal side. II. 3, bled to death from a hatchet wound of calf, no details. III. 3, bled to death at the age of 4 from a slight wound of the head, the result of a fall. IV. 1, female, aged 11, presented no tendency to excessive bleeding. IV. 2, a typical bleeder, aged 9. From early infancy he was a bruiser, a slight injury causing the appearance of hard ecchymotic tumours. When about the age of 3 he slightly injured the mucous membrane of the nose and bled 11 days. He had bled from the gums for 10—12 days and had sustained serious haemorrhage from a bite on the tongue and as the result of the fall of one of his milk teeth. Epistaxis was also frequent and severe. At about 6 years he began to suffer pains and swellings of the joints, especially the ankles, shoulders and knees. Some months before he received a bruise on the malar prominence. Great periorbital swelling and exophthalmos occurred and severe haemorrhage followed the bite of one leech which had been applied to reduce the swelling. Retrobulbar abscess ensued with lesion of the bulbus itself and perforation of the cornea. A month later he was seized with violent girdle pains and extensive paraplegia suddenly set in involving the lower limbs, trunk and partly the upper limbs. Ledoux regarded this as the result of haemorrhage into spinal cord. The boy was alive at the time of Ledoux's publication. We are indebted to Professor van Gehuchten of Louvain for our account of this inaccessible case. (See Bibl. No. 364.)

Fig. 460. *Thompson's Case.* Enquiries into the history of this family were first made in connection with V. 1, Jack Duckworth, a typical bleeder, under the care of Dr Theodore Thompson in the Hospital for Sick Children, Great Ormond St., London. His mother, IV. 16, Mrs Duckworth, living at Northumberland Heath, Belvidere, Kent, was visited and she was able to supply a large part of the family history. In spite of some considerable enquiry we have been unable to obtain evidence of the disease except in the fifth generation. V. 1, Jack Duckworth, aged 11, a severe bleeder. First showed definite evidence of the disease at the age of 18 months, when he injured the fraenum of the upper lip and bled for six days. He had also bled on other occasions, especially from the mouth, but his chief trouble was painful swellings

of his joints. When seen he was covered with bruises and had a large blood lump on his forehead. V. 2, Frank, was a typical bleeder and at the age of 18 months fell and bit his tongue and bled to death. V. 3, Alexander, a typical bleeder, with frequent attacks of haemarthrosis and bruises. V. 1, and V. 2, were very delicate looking boys with unusually fine white transparent skins. IV. 16, affirmed that the haemorrhages had a very marked periodicity in both V. 1, and V. 2. V. 4, dead, not a bleeder. V. 6, and V. 7, not affected. V. 8, and V. 9, according to IV. 16, were typical bleeders. V. 10, was a seven month child and died at birth. (See Bibl. No. 903.)

Fig. 461. *Murray's Case.* Five boys, four being brothers, while the fifth (marked III. 4 in pedigree) was "distantly related" to the mother of the four bleeders. These cases occurred in the parish of Keig, Aberdeenshire, and were described by Murray in 1826. We have instituted enquiries relative to the descendants and collaterals and with the help of Mr E. Alexander, present schoolmaster of Keig, we have been able to trace a considerable portion of this family down to the present time, the additional information being obtained from IV. 20, and her daughter, V. 26, and from a niece, V. 2, of the bleeders, an old woman aged 75, who was interviewed by one of us (W. B.) in Aberdeen (Aug. 1909). The tendency to haemophilia was known to all of these, as the deaths of IV. 1, 2, and 3, had created a great impression. It is unfortunate that a possible female conductor of the disease, IV. 11, went to America long ago and was lost sight of, nothing being known with regard to her children. Concerning generation I. nothing is known. It was elicited from V. 2 that II. 1, and II. 3, were brother and sister. III. 1, Coutts of Cobbleseat (Keig), married to III. 2, Cobban by name. III. 5, male, killed at Waterloo. III. 6, 7, 8, 9, all lived long. IV. 1, a bleeder. At the age of 20 weeks he had epistaxis lasting twenty-four hours and recurring every month till he was 3, when he died of it. Before the onset of bleeding he showed large and small blue spots and bled occasionally from the bowel. He was known to have bled severely from a pin scratch. IV. 2, a bleeder, had livid spots and haemorrhage from his bowels like IV. 1, and died, in 1802, when 15 months old, from haemorrhage from the mouth, the result of a fall. IV. 3, a bleeder. From 1 year onwards he bled occasionally from the mouth and nose, eyes or eyelids, and died after bleeding from the mouth and nose for a month. IV. 4, William Coutts, aged 13, attended by Murray in 1824. He bled from the gums when 1 year old and subsequently from the nose. Livid spots and copious haemorrhage resulted from injuries. He had bled spontaneously from the crown of his head, and when seen by Murray had a black eye, which was brought about by a trifling injury. Later on, his tendency to bleeding was diminished and no dangerous haemorrhage resulted from venesection. V. 2, informed us that he got well and lived to old age. IV. 5, Peter Coutts, not a bleeder, died aged 73. IV. 7, no details. IV. 8, 9, 10, unknown to V. 2, and probably died early. IV. 11, went to America and married, but her descendants have been lost sight of. IV. 13, lived to old age. IV. 15, Isabella Cobban, married and had a family, all of whom were healthy. IV. 19, was "dull." IV. 20, Rachel Cobban, alive, very old and eccentric. V. 1, died at the age of 2, but was not a bleeder. V. 2, Mrs Gerrie, aged 75, healthy, seen by one of us in Aberdeen in Aug. 1909. V. 4, William Coutts, dead; lived to old age. V. 5, Alexander, alive, old man. V. 6, Robert, an old man, living in America. V. 7, James, died of croup at the age of 8. V. 8, Jane, alive, aged 60, unmarried. V. 9, George, alive, living in Newcastle. V. 11, James, alive. V. 12, Margaret, alive, unmarried. V. 13, stillborn. V. 14, female, gored to death by a bull when 15 years old. V. 16—26, not affected with a tendency to bleed. Generations VI., and VII., were unaffected. Murray saw another member of this family, a "distant relative" of III. 2. He is marked III. 4, and was $6\frac{1}{2}$ years old. He was liable to bruises from slight injuries and had bled for two days from a small cut. In 1824 Murray was called to see him as he had been bleeding from the fraenum of the upper lip for a week. It was impossible to stop it and he died on the second day. (See Bibl. No. 47.)

Fig. 462. *Kingsford's Case.* I. 1, and I. 2, not mentioned. I. 3, I. 4, no information. II. 3, healthy. II. 4, and II. 5, died of haemorrhage during confinement. II. 6, II. 7, died of haemorrhage. III. 2, stated to have been a bleeder during the first thirty years of his life. IV. 1, 2, 4, 6, not affected with haemophilia. IV. 8, six children, no information. V. 1, menstruated freely. V. 2, healthy. V. 3, a marked haemophilic, no details. V. 4, aged 10, a bleeder. He was subject to haemorrhagic joints and had bled for days after tooth extraction. He nearly died after circumcision. On Nov. 24th, 1905, he became constipated and had abdominal pain and vomiting. Copious chocolate coloured evacuation of bowels ensued on the 25th, and on Nov. 27 he suddenly collapsed and died. Autopsy showed a large irreducible intussusception involving more than a foot of the ileum, the distal extremity being about three inches from the caecum. Protruding from the intussusception was the blind end of a long Meckel's diverticulum. Kingsford emphasises the fact that three female members of the family died of haemorrhage, although it cannot be asserted that they were bleeders. There are no details to show that III. 2, and V. 3, were bleeders, although they may have been. (See Bibl. No. 858.)

Fig. 463. *Wilson-Lane Case.* Wilson has described the history of a family in which, although the parents were healthy, all the male children were bleeders. None of the females were similarly affected. Wilson describes in particular a boy, II. 1. Lane, in an article on the haemorrhagic diathesis, reported the facts with reference to II. 2, II. 3, II. 4, and II. 5, as they were verbally communicated to him by

Wilson. I. 1, robust and large. I. 2, enjoyed very good health. II. 1, a male bleeder. It was observed that the nurse, although very careful, left marks upon him when she dressed him. Wilson saw him frequently in a bruised condition. Great difficulty was experienced in arresting the haemorrhage from a pin scratch, while a leech bite nearly killed him. Between the ages of 3 and 4 he accidentally bit his tongue. Great haemorrhage ensued, which Wilson was unable to control, and he bled to death five days later. The autopsy revealed a great thinness of the aorta and its branches, which were more like veins than arteries. II. 2, bled to death after tooth extraction. II. 3, and II. 4, both died from intracranial haemorrhage as the result of falls, which would have produced no serious result in healthy children. II. 5, alive, but a severe bleeder. He nearly died from haemorrhage after tooth extraction and on another occasion from haemorrhage from the bowel. (See Bibl. Nos. 32 and 118.)

PLATE XLII. *Wickham Legg's Cases.* Fig. 464. *Case I.* I. 1, and I. 2, healthy. I. 3, unaffected. I. 4, the source of the information, was aged 55, and suffered long from "gravel in the kidneys." None of her relatives were known to be affected with haemophilia. II. 2, aged 35, a paperhanger, born in Devon. He was not related to his wife. Neither he, nor his brothers or other relatives, had any tendency to bleed. II. 3, died at the age of 29. One month after the delivery of her last child, she was sitting in a chair, when she suddenly cried out, "Mother, it is dark!" She fell back, and died in a few minutes. She was an only child and always weak in health. III. 1, Richard Bickell, born 1859, was seen at St Bartholomew's Hospital, London, in 1871, *i.e.* at the age of 12. He had been suckled for 12 months. At 3 he had measles, and at 5 whooping cough. The first dentition decayed early, and at the age of 4 or 5 one of the back teeth was removed. The bleeding was uncontrollable and lasted three weeks. This was the first haemorrhage of any kind noticed. At the age of 6, his left knee and subsequently other joints became swollen and painful. Three months before observation a tooth fell out, and the socket bled for one week. There was, then, a short pause, followed by a further haemorrhage for one week. No epistaxis or other spontaneous haemorrhage. Blisters reacted normally. He remained in hospital while cutting his teeth, during which time a slight oozing took place. His left knee and right ankle were swollen and full of fluid, though painless. The heart presented no abnormalities. In a personal communication Dr Wickham Legg informs us that this man was seen again in August, 1885, at which time he was aged 26 and a commercial traveller. At 21, after rowing on the river, he vomited blood for three days. At 25 he was again laid up with the affection of the knee. III. 2, Annie, aged 10, healthy. III. 3, Nellie, aged 8, healthy, though she had an attack of epistaxis. III. 4, Harry, aged 6, was frequently subject to epistaxis, lasting three or four days. Large "black and blue" patches appeared on his skin when he was bruised; these did not appear spontaneously. (See Bibl. Nos. 321 and 331.)

Fig. 465. *Case II.* I. 3, a bleeder, the first known in the family; he was no relation to I. 4. II. 2, was dead, and had suffered from gout. II. 3, aged 68, was healthy: there was no history of bleeding in his family. II. 4, aged 64, source of the information. She enjoyed good health, but died one year after publication of the account (1871). II. 6, died of cancer of the tongue at the age of 64; he was unaffected. No children. This man and his sister were the only members of the family to reach maturity. II. 7, numerous boys and girls, who died early of unknown causes. III. 1, and 2, idiots. They both died in an asylum. III. 3, over 35 years of age, unaffected. III. 4, died of whooping cough at 3. III. 5, aged 32. III. 6, died of "typhus" at the age of 6. III. 7, a bleeder. The tendency was first noticed at the age of 18 months, when he fell down and bit his tongue. When a tooth was loose or he cut himself he bled "awfully." III. 8, died at the age of 25. He had heart disease following rheumatic fever. He is described as a bleeder. He had "chalk stones up the sides of his feet." III. 9, a bleeder, died of scarlet fever at the age of 3 or 4. III. 10, a six months miscarriage. III. 11, James Cameron, aged 24, a sailor, on Australian ships. He was born in Clerkenwell, London, and was below the average intelligence. He was first noticed to be a bleeder at the age of 6 when he was shedding his milk teeth. He said that all his life he had often bled from slight accidents, and had been in hospital for them. He never had swelling of joints. He was seen in 1868 when he had erysipelas of the head and neck. There was an abscess below the parotid gland, which was incised. The skin sloughed, and some bleeding occurred from the granulating sinus. He was alive and well in 1871. III. 12, died of "typhus" at the age of 4. III. 13, poisoned himself at the age of 40[1]. III. 14, died of smallpox at 5. IV. 1, unaffected; whose child not stated. (See Bibl. Nos. 321 and 331.)

Fig. 466. *Case III.* II. 1, an armoury sergeant, subject to excessive bleeding from slight causes. He was killed by an explosion at an unknown age. II. 3, aged 40. When he had a tooth out or wounded himself, the bleeding was difficult to stop. II. 4, died of consumption at the age of 30. II. 6, aged 30, unaffected, born in Whitechapel, London. II. 7, *née* Sawyer, source of this information, unaffected. No miscarriages. II. 8, and 9, both unaffected, as were their ascendants. III. 1—9, unaffected. III. 10, James Day, aged 8, born in Bethnal Green, London; suckled for 15 months. He was not subject to purpura, bruises, or swelling of the joints. At 5 he had an attack of epistaxis, which lasted 15 minutes or more. Two years before he had had measles, and had never fully recovered his health. Latterly, epistaxis had given way to bleeding from the mouth. No unusual haemorrhage occurred after cuts or

[1] *Owing to a slip of the engraver the mark of died young should be attached to III.* 12 *and not to III.* 13 *in Fig.* 465.

tooth extraction. He was thin, but of good appetite. The mouth was normal. He recovered his general health in six months after taking cod liver oil and steel wine. (See Bibl. Nos. 321 and 331.)

Fig. 467. *Case IV.* II. 1, aged 45, healthy. He frequently had attacks of epistaxis, and as a young man used to bleed "just as I do" (*vide* III. 1). He was not related to his wife. II. 2, aged 47, healthy. II. 3, "mother's brothers," unaffected. III. 1, James Hicks, born in 1851, at Bermondsey, London. He was a Thames lighterman, and supplied the information. In the first account (1871) it is stated that his evidence was open to doubt. He was subject to epistaxis from birth. When wounded the blood was difficult to stop, but tobacco succeeded quickly. A cut on the finger five days old was found to be quite healed. Occasionally a little blood was seen in his sputum. He had pain, in the right knee, which increased at night. The epistaxis having become worse he went to St Bartholomew's hospital. III. 2, aged 18, suffered from epistaxis and a difficulty in arresting haemorrhage. III. 3. died of measles at 18 months. III. 4, aged 14, affected similarly to III. 1, and III. 2, as regards wounds. III. 5, aged 12. III. 6, aged 8, his mind had been unhinged from fright. III. 7, died of "fever" at 12 months. (See Bibl. Nos. 321 and 331.)

Fig. 469. *Case V.* II. 1, Ferdinand Uhle, born 1861, in Hanover, seen at St Bartholomew's hospital in 1872. At 17 months, spontaneous ecchymoses were seen. At 2, he started frequent attacks of epistaxis. He also at that time fell and cut his forehead; though treated by a doctor, in Hanover, he nearly died. At 7, he had haematuria. Latterly he bled occasionally from the gums, especially when he was changing teeth. At 2, he first had joint lesions; knees, elbows and ankles swelled, as also did the small joints of the fingers. Leeches were applied to one of the knees in a London hospital, and the bleeding lasted a day and a night. A brother, II. 2, also is affected. (See Bibl. No. 337.)

Fig. 470. *Case VI.* II. 1, aged 47, came of a gouty family. He had "rheumatic gout" in all his joints for 20 years, and chalk stones in his hands: unaffected with haemophilia. No relation to his wife. II. 2, aged 40, unaffected. II. 3, died at the age of 20. He had been subject to bleeding from slight injuries from birth. He died of haemorrhage, following amputation of the leg for fracture. III. 1, William Robins, aged 23, a painter, attended at St Bartholomew's Hospital in 1871 for swelling of the knee. There was a slight "blue line" on his gums. Unmarried. The tendency to haemophilia was first noticed at the age of 2, when he frequently had epistaxis. As a child he bled abundantly when pricked or scratched, and bruised readily. At 20 he was treated for three weeks at St Bartholomew's Hospital for haemorrhage from a cut in the palm. A swelling of the right knee was first noticed at 4; it recurred till the age of 13, from which time he had been crippled by it. He had also had swelling of the ankle. His gums sometimes bled, and haematuria had occurred. Recently, epistaxis had been most troublesome. III. 2, healthy, aged 21. III. 3, died at the age of 7. He was subject to bleeding. III. 4, died in infancy. III. 5, died at 4 of a cause unknown. III. 6, healthy, aged 5. (See Bibl. No. 331.)

Fig. 471. *Case VII.* I. 1—4, no information. II. 2, and 3, healthy, came from Ireland. No history of haemophilia in their families. III. 2, died at the age of 4 in St Bartholomew's Hospital after a plank had fallen on his back. He was subject to epistaxis, on one occasion for three weeks, when he was in King's College Hospital, London. III. 3, C. S(utton), was subject to epistaxis and great haemorrhage from trifling injuries. When 21 months old he was in Charing Cross Hospital, London, for five months with a swollen knee. At 3, he was in King's College Hospital, London, on account of bleeding from the lip. At the age of $3\frac{1}{2}$, he was again in hospital for 14 days, after a cut on the lip. At 6, he was admitted to King's College Hospital with a swollen ankle. An incision was made, and the haemorrhage lasted for one week. At 7, he was in St Bartholomew's Hospital, and again, at $7\frac{1}{2}$ years, for an injury to his ankle. At $8\frac{1}{2}$, he nearly cut off the top of his thumb; the haemorrhage lasted 14 days. At 9 years he was again admitted to St Bartholomew's Hospital for a bite of the lower lip. After 14 days' bleeding he died. At the autopsy large ecchymoses were noticed on the thighs and legs. There was an extravasation of blood in the right popliteal space. The knee joint contained adhesions. No microscopic changes were found. III. 4, unaffected. III. 5, living, no information. III. 6, a miscarriage. III. 7, 8, 9, dead. (See Bibl. No. 450.)

Fig. 472. *Case VIII.* II. 1, Charles B. At the age of 3 he bled for a long time from a small wound of the tongue. At 6, for eight weeks after tooth extraction. He bruised easily, and was frequently attacked by painful swelling of the joints. At the age of 13 he was admitted to St Bartholomew's Hospital, for epistaxis, of which he died. A few drops of blood were also noticed coming from the inner canthus of the right eye. The left ankle joint was swollen, and a large ecchymosis was found in the left groin. At the autopsy "atheromatous" changes were noted in the heart valves. The thymus was persistent, and the spleen large. A few patches of pneumonia in the lungs. Old and recent blood in joints. II. 2, died of epistaxis at the age of $3\frac{1}{2}$ years. These two boys were the only instances of excessive bleeding in the family. (See Bibl. No. 489.)

Fig. 468. *Case IX.* Dr Wickham Legg has published, with the aid of materials supplied by Mr W. B. Rigby, the history of the Clitherow family of bleeders from Prescot, Lancashire. The 23 bleeders, all of whom were males, were distributed as follows: three in the second generation, one in the fourth, three in the fifth, twelve in the sixth, and four in the seventh generation. The original ancestor, I. 1, so far as is known, was not a bleeder, but he had three haemophilic sons, viz. II. 1, II. 2,

II. 3, the first two of whom died from bleeding. The third, II. 3, died of haemorrhage of the bowel in 1770. He married a widow, II. 4, of unknown descent, and by her had two sons and two daughters, all free from the disease[1]. His grandson, IV. 1, was however a bleeder, and died, aged 3, of bleeding from a wound of the head. Of his two granddaughters, IV. 2 married, and had four boys unaffected; the other, IV. 4, had three sons affected, viz. V. 14, V. 16, V. 17. V. 14, died of bleeding from a graze of the thumb. V. 16, aged 3, died of bleeding from a bite of the tongue, and V. 17, died at the age of 4 of haemorrhage from a wound of the finger. V. 10, herself healthy, had by one Burrows, V. 11, three haemophilic sons, VI. 1, VI. 2, VI. 3, and two normal daughters, one of whom had four bleeder boys out of eight. VI. 1, VI. 2, VI. 3, all died of haemorrhage when young. VI. 9, died of epistaxis at age of 4. VI. 11, alive, aged 25, a bleeder. VI. 12, died of haemorrhage of bowels, aged 7. VI. 13, aged 10, a slight bleeder. VI. 16, died, aged 3, of haemorrhage from the mouth. VI. 18, died, aged 2, bleeding from mouth. VI. 20, died at the age of 2 days, a blood tumour on the scalp having been opened. VI. 21, died, aged 7, from bleeding from mouth. VI. 22, died, aged 7, from bleeding from mouth. VI. 23—VI. 28, not affected. VII. 1, died, aged 7, from bleeding from wound of head. VII. 2, died of haemorrhage from mouth. VII. 4, a bleeder, alive. VII. 5, a bleeder, alive. VII. 7, VII. 8, twins, unaffected. VII. 9—VII. 14, all healthy. (See Bibl. No. 437.)

Parks M. King's Cases. King, of the Presbyterian and St Peter's Hospital, Charlotte, N.C., has described two cases, one of which, Case II, belongs to a large family of bleeders.

Fig. 473. *Case I.* Short account of a bleeder, III. 3, with a history of haemophilia in three maternal uncles. I. 1—4, no data. II. 3, was a Miss Flora Giles, of Rockingham, N.C. II. 4, 5, 6, three males said to be bleeders, no data. III. 1, died of pneumonia, aged 2. III. 3, William Wallace, aged 5, began to bleed when he was 3, and had four attacks of haemorrhage since then, the bleeding lasting ten days to two weeks. In Sept. 1908 he fell and cut his tongue slightly, and bled so badly that he had to be admitted into the hospital, where he was treated with thyroid extract and calcium lactate. After three or four days of this treatment the bleeding ceased.

Fig. 475. *Case II.* King's patient was Sam Millwee, V. 3, aged 25, and he gave the history which was "verified to a large extent by Drs I. M. Heron and Jno. Knox, of Mecklenburg County, who have practised among the families for a number of years." I. 2, Neeley, healthy. He married secondly a Miss Black, I. 3, of Fishing Creek, S.C., and had by her one boy, Thomas, II. 2, not a bleeder, and two girls, viz. II. 3, Hannah, and II. 7, Jane, who became the second wife of one Sam Knox, II. 6, who had four normal children by his first wife, II. 5. The marriage of II. 6, and II. 7, resulted in twelve children, six boys and six girls. Four of the boys bled to death as follows. III. 3, Leroy, at the age of 12, was kicked on the leg by a horse, and died from haemorrhage from a small lacerated wound. Henry, III. 4, at the age of 2, bit his tongue and bled to death. III. 5, Albert, at the age of 3, stuck a cane in his mouth and bled to death. III. 6, Joseph, "who was also a bleeder, is supposed to have bled to death in the civil war." III. 7, Thomas, was alive, aged 73, and had never bled. He was the father of two boys, IV. 1, and 2, who were not bleeders; both however died in childhood. III. 9, the sixth son, no information. III. 10, Nancy Knox, married Carruthers, III. 11, and had one bleeder son. III. 12, Jane, married III. 13, Wyatt, and had 11 children, of whom four were bleeders. III. 14, another sister, married; no data. All the daughters of II. 6, and II. 7, were healthy, and "none of them ever bled." IV. 1, and 2, were healthy. IV. 3, bleeder; bled to death from a cut on his cheek at the age of 2. IV. 4, female, "was born blind, and is alive at the age of 50." IV. 5, Walter, was a bleeder, and died of diphtheria at the age of 4. IV. 6, William, died at the age of 20 from a cut on his finger. IV. 7, John, died at the age of 28. "He had been a great sufferer from rheumatism. His knees became stiff and flexed so that he had to use crutches. During one of his attacks of pain his knee became so swollen that the skin ruptured and he bled to death." IV. 8, Sam, "a chronic sufferer from arthritis, died at the age of 20 from haemorrhage with typhoid fever." The five daughters of III. 12, and 13, were healthy. One, Carrie, IV. 11, had recently married and had no children. Margaret, IV. 14, was the mother of King's patient. Ellie, IV. 17, the third married daughter, had one son, who so far had not shown evidence of the disease. V. 1, male, "died at the age of 4 days from haemorrhage from the cord." We have marked him a bleeder, although this perhaps is doubtful. V. 2, fell from his cradle, cut his upper lip, and bled to death at the age of 14 months. V. 3, Sam Millwee, King's patient, aged 25. He began to bleed at the nose at the age of 2, and had a number of attacks of epistaxis. About the same time he began to suffer with pains in his joints, especially his knees, which became swollen and black. His left knee was partially ankylosed. At 8 he bled for two weeks from a small punctured wound of the knee. Four years before the calf of his R. leg became black and blue, and the muscles subsequently atrophied. When 21 he cut his left foot and nearly bled to death. His left ankle had been ankylosed in an equine position since that time. Admitted into hospital suffering from haemorrhage from the bowel which, in spite of treatment, almost proved fatal. V. 4, 6, 7, and 9, unaffected. VI. 1, died of atelectasis in the first day of life. VI. 2, was a bleeder, died of enteritis. VI. 3, John, cut his finger when 3 years old and bled to death. VI. 4, Samuel, aged 15, VI. 5, Charles, aged 13, VI. 8, Albert, aged 4, "are bleeders having epistaxis and pains in the joints." A plate, showing VI. 4, 5, 6, 8 with bared legs, accompanies

[1] *Owing to a slip of the engraver in generation II. the words* 2nd Husband *and* 1st Husband *are interchanged in Fig.* 468.

Dr King's paper. The knee joints of the bleeders VI. 4, VI. 5, and VI. 8, are seen to be enlarged. VI. 6, Knox, aged 9, not affected. VI. 7, female, died of gastro-enteritis. VI. 9—14, healthy females. VI. 15, born prematurely, and died at birth. VI. 16, John, aged 14, not affected. VI. 17, William, aged 4, was a bruiser, but so far had not bled. (See Bibl. No. 889.)

Fig. 474. *Delmas' Case.* (See remarks attached to Simon's case, Bibl. No. 350, Fig. 512.) This is the history of a male bleeder, V. 6, whose parents came to France from America (Louisiana). I. 1, and 1. 2, presumably normal. II. 2, not affected. II. 3, is said to have been a very severe bleeder, but owing to extreme precautions he lived to old age, passing the greater part of his life in an armchair, so to speak. At the age of 75 he had an epileptiform seizure which was followed by dementia and death. III. 1, and III. 2, two normal males. III. 3, suffered greatly from rheumatism. III. 4, a bleeder, and bled to death at the age of 14. III. 5, maternal grandfather of Delmas's patient, was a clerk and had suffered from haemophilia all his life. As a child he was frequently covered with ecchymoses as a result of movements such as throwing a ball. If he remained seated for any length of time his thighs and buttocks would become covered with bruises. At the age of 47 he had epileptiform attacks which were succeeded by general paralysis, from which he died. III. 7, 8, 10, 11, four sisters of the bleeder III. 5, were healthy. III. 5, married III. 6, and by her had four daughters and four sons, IV. 1—4, 6—9, none of whom manifested any symptoms of haemophilia. One of the daughters was however the mother of the bleeders V. 3, 4, 6, 8. A normal sister of III. 5, married and had three boys, IV. 10, IV. 11, IV. 12, all of whom bled to death from the gums at 3, 2, and $1\frac{1}{2}$ years respectively. IV. 4, had 14 children in 11 pregnancies. Once she had triplets, V. 9, 10, 11, who were born prematurely in the seventh month. They were all males, and succumbed a few hours after birth. On another occasion, also at seven months, she had twins, boy and girl, both dying shortly afterwards. Of the eight children who survived seven were male, and of these four were bleeders, viz. V. 3, 4, 6, 8. V. 1, a girl, normal, aged 17. V. 2, boy, aged 14, not affected. V. 3, a boy, aged 10, a bleeder. He suffered such excruciating pains in his joints that at one time he was in danger of becoming a morphinomaniac. When 5 years old an attack of epistaxis lasted continuously for a month. As a last resource inhalations of chloroform were tried, and after two administrations the bleeding stopped. V. 4, a bleeder, died at the age of 13 months from an intestinal haemorrhage during whooping cough. V. 5, male, aged 6, normal. V. 6, M. J., aged 5 years, the bleeder, seen by M. Delmas. At the age of one year he was observed to show ecchymoses. At 2 a furuncle on the buttock gave rise to profuse haemorrhage which lasted a fortnight, and rendered him almost exsanguine. When 3 he had a post-traumatic haemorrhage from the orbit. He also was the subject of haemophilic arthropathies. V. 7, male, aged $3\frac{1}{2}$, normal. V. 8, male, aged $2\frac{1}{2}$, a bleeder. Delmas has published another case regarded by him as a haemophilic female, by name Pétronille Béron, aged 32. Her mother had died of haemorrhage at the age of 45. A sister, who was considered to be tuberculous at the age of 15, began to menstruate two years later. She had violent haemoptysis, from which she succumbed. Two brothers died, one at 5, the other at 2 years. The patient herself began to be ill about the age of 15, after the onset of menstruation, which was followed by metrorrhagia, epistaxis, haemoptysis and chorea, with pains in the joints. The day after her marriage, at 19, she was attacked with similar symptoms, and again at 28 years, when she had her fourth confinement. She had five children, of whom four survived. The eldest, a girl, was haemophilic—no data. In 1869 she was again taken ill, and entered the hospital, where she was placed under Delmas, who was director of a hydrotherapeutic establishment and made a speciality of this form of treatment. A great part of Delmas's paper is taken up with the different forms of hydrotherapy applied to Pétronille Béron, whom we cannot admit as a bleeder. Under the name Jeanne Mérieux, aged 34, François Franck has described a case which, although differing in Christian name and surname, is identical with Pétronille Béron. Professor Sabrazès of Bordeaux, to whom we are greatly indebted for the history of these Bordeaux cases, considers that the cases of Franck and Delmas are one and the same. (See Bibl. Nos. 298, 317, 326.)

Fig. 476. *Hopff's Case.* The first case published under the name "Hämophilie." I. 1, and I. 2, II. 5, II. 6, stated not to have been bleeders. II. 1. died of haemorrhage following tooth extraction. II. 2, II. 3, bled to death from slight injuries to the head. II. 4, when a boy, bled copiously after a wound from a splinter of glass. Later was permanently lamed as the result of a contusion of the knee due to a fall from a horse. Complained of "gout," which left his arm stiff. When cupped for joint pains bled severely. As a result of "gout" a large mass formed in his thigh. Hopff was called to see him and found a hard inflamed swelling which softened later on. Spontaneous rupture taking place, a large quantity of brownish-red fluid was discharged, containing concretions. Suppuration set in with haemorrhage and the patient died. Autopsy not permitted. We are indebted to Prof. Borst of Munich for a complete abstract of this very inaccessible inaugural dissertation. (See Bibl. No. 53.)

Fig. 477. *Walker's Case.* Walker, of Spilsby (Lincoln), has briefly recorded the history of two male bleeders, with an incomplete history of haemophilia in two others in the same family. I. 1, a clergyman, died of heart disease aged 62. I. 2, died of senility aged 72. At 70 had an attack of haematemesis. I. 4, alive, aged 72. I. 3, healthy, living, aged 67[1]. II. 9, a clergyman, healthy. Suffered from headaches,

[1] *Owing to an oversight of the engraver the words* 1st Husband and 2nd Husband *in generation I. of Fig.* 477 *have been interchanged.*

which were relieved by epistaxis. He was known to Walker for 11 years before his marriage, as was also his wife, II. 10. II. 11, died young. II. 12, living, unmarried. II. 13, II. 14, twins. II. 15, died in infancy. II. 16, living, aged 48. III. 3, and III. 4, two miscarriages. III. 5, female, born 1861, healthy. III. 6, Charles G., born 1863. At 2 months bruises were first noticed and still occurred. They were often very large and so much blood was extravasated that his mucous membranes became blanched. Sometimes they appeared to develope spontaneously, whereas at other times they were due to traumata. Epistaxis occurred at 16 months for the first time, and shortly afterwards he fell, injuring his gums. The epistaxis ceased, but bleeding from the gums continued 14 days and nearly killed him. At the age of 2 he had a similar experience. At 6 an attack of scarlet fever was followed by an aphthous condition of the mouth and ten days' bleeding. At 7 he cut his finger at bed-time. The nurse tied it up and said nothing about it, but next morning the blood was through his bed and on the floor. At 8, haemorrhage from a bite of the tongue was fairly easily controlled. The tendency to bleed appeared to be getting less, and he had shown no joint lesions. III. 7, Edward, aged 7, born 1864. When one month old bruises were noticed, and had recurred ever since. Had scarlet fever at the same time as his brother, and since then had developed painful swellings of his joints. Bleedings from the tongue had occurred, lasting on one occasion for 14 days. III. 8, free, aged 6. III. 9, George, born 1868. Bruises occurred early, but were not so marked as in his brothers. He, too, had scarlet fever with suppurating glands, and from these and from the whole gingival surface he bled to death—aged 16 months. III. 10, a female, not affected, aged 2. III. 11, Gerald, aged 1. He showed a marked tendency to bruising, which was first observed when he was 10 days old. He had however cut five teeth without haemorrhage. III. 12, all healthy. (See Bibl. No. 334.)

Fig. 478. *Weigersheim's Case.* A short and valueless report of a communication given in the *Hufeland Gesellschaft* in Berlin, Jan. 25, 1878. A pedigree is appended with one or two isolated data. I. 1, "Itzig" (male?) died of bleeding when over 50—no details. II. 1, David, not a bleeder. II. 2, Louis, bled up to the age of 20. II. 3, Moses, "bled," subsequent history unknown. II. 4, Adolf, epistaxis, died of bleeding at the age of 52. II. 6, Rosalie, aged 62, not affected. II. 8, Hannchen, died at the age of 48, not a bleeder. II. 10, Caecilie, aged 56, "still bleeds." III. 1, Antonie, "bled," died at the age of 13. III. 2—4, healthy. III. 6, "H. L.," aged 21, not affected, sex not stated. III. 7, Emil, aged 18, "begins to bleed." (See Bibl. No. 393.)

Fig. 479. *Klein's Case.* I. 1, I. 2, not mentioned. I. 3, I. 4, died of cholera in 1866. II. 2, died of morbus cordis in 1898. II. 3, insane for some time and died of apoplexy at the age of 40. She and her three sisters, II. 4, II. 6, II. 8, were all said to have bruised easily, but to have shown no symptom of haemophilia. III. 1, K. B., aged 24, an architect—a marked bleeder. Haemophilic tendency discovered at his primary vaccination. When 1 year old, had colossal swelling of head from a fall. At 6, a small wound of tongue bled so profusely that it had to be cauterised several times before it would stop. At 10, fell on his nose and had great epistaxis and bleeding from the mouth. At 14, swelling of knee after a blow. Sitting on a stool with sharp edges produced swelling of thighs. Had had frequent superficial haemorrhages. After 16, bleedings and joint swellings became less, and he was now frequently attacked with severe "rheumatic" pains in the joints. Haemarthrosis into knee and elbow at 19 from cycling. One year later immense blood swellings in both groins. Eighteen months later carious tooth removed after great deliberation. At first bleeding was slight, but in three hours it was furious and lasted two days, when he was completely anaemic. He had haematuria twice. III. 4, a bleeder, died in his 14th year after an operation on his knee. III. 7, bled to death after tooth extraction at the age of 7. (See Bibl. No. 725.)

PLATE XLIII. *Grandidier's Cases.* Fig. 480. *Case I.* A Jewish bleeder family in Kassel. Originally the family came from Braunschweig, and at the time of Grandidier's publication, branches were living in Kassel and in Einbeck (Hanover). I. 2, Frau H., a Jewess, of Braunschweig. II. 2, Frau G. II. 3, Frau W. No information about them. III. 1, no information. III. 3, Frau H., not a bleeder, and died of some other disease. III. 5, Frau L., living first in Münden, later in Kassel (called Frau C. in Grandidier, *Die Haemophilie*, 1877, 2nd edit.). III. 7, Frau J., living in Einbeck. IV. 1, male, marked in Grandidier's pedigree as having bled to death after circumcision, not mentioned in text. IV. 2, female, alive. At the age of 2 almost bled to death from a leech bite, and always bled unusually freely after cuts, now healthy. IV. 3, a male, he had no umbilical haemorrhage, but bled to death after circumcision, being then 8 days old. Three doctors who were in attendance could not control the haemorrhage by any method of treatment. A full account of the medico-legal autopsy made by Grandidier on this boy is given in Grandidier, *Die Haemophilie*, 1st edit., Anhang, S. 151. IV. 4, female, healthy. IV. 5, bled to death after circumcision, the haemorrhage having lasted eight days. This was in 1826. A medico-legal enquiry was held on the matter as want of skill was alleged on the part of the operator. IV. 6, male, was not circumcised, the parents in the meantime having become Christians. He was grown up and healthy. His children (Grandidier, 2nd edit., 1877) healthy. IV. 9, Karl L.—a bleeder, not circumcised, aged 14. When 14 days old bruises first made their appearance upon him. At 1 year he developed a haematoma on his back without apparent cause. Dangerous haemorrhages after tooth extraction, leeches, and injuries

were incidents of his early youth. Later his joints became involved, especially his knees, which were repeatedly affected. In 1852, while a boy, he fell from a table on his knee, which swelled. After the swelling subsided contracture set in. His elbows were also frequently swollen. At a later period the tendency to spontaneous external haemorrhages diminished, although they were severe after injuries. Grandidier saw him with a vast hard haematoma involving half his face, the result of a blow by the head of another boy. His subsequent history is given by Grandidier 22 years later, in 1877 (*Die Haemophilie*, 2nd edit.), when he was 36 years of age. Whereas after 21 he no longer bled spontaneously, he was still liable to bruising after trivial injuries. At 33 years of age he had a considerable haemorrhage after the removal of a tooth, but no more joint lesions. He was twice married, and had children by each wife, but none were affected with his disease. IV. 11, and IV. 12, died young. IV. 13, died young according to Grandidier (1st edit.), married P. in Kassel (2nd edit.). IV. 15, married and lost sight of (Palestine), presumably the married sister in Smyrna, referred to by Grandidier in his 2nd edit. IV. 17, bled to death after circumcision, no details. V. 1, 2, 3, three healthy families, number and sex of individuals not stated. V. 4, male, since infancy an "exquisite" bleeder (*Die Haemophilie*, 2nd edit.). V. 7, family thought to be healthy, number unknown. Stated to be free from haemophilia in the 2nd edit. (See Bibl. Nos. 214 and 372).

Fig. 481. *Case II.* History of a Jewish family living in Bavaria and New York. Facts communicated by Henschel to Grandidier. I. 1, Frau A., of Windheim, in Franken, suffered from great haemorrhages and died at the age of 96. II. 1, Frau Fr., of Fürth, alive. III. 1, and 2, died of epistaxis, no details. III. 3, and III. 4, bled to death after circumcision. III. 5, male, bled badly after circumcision. Contracture of arm, ecchymoses frequent. III. 6, Frau S., of Nürnberg. From the account it is impossible to say definitely whether this person is a sister of II. 1, or a daughter. The latter is the more probable. IV. 1, male, bled to death from the lungs at the age of 21. Had suffered from epistaxis, haematemesis, haematuria. After injuries, bleeding was so great that it could scarcely be controlled. He had a swollen knee, contracture of an arm and numerous ecchymoses. IV. 2, and IV. 3, alive, bleeders, no data. IV. 4, alive, a bleeder with contracture of an arm. IV. 5, free. IV. 6, free. IV. 11, Frau S., in New York. IV. 13, had gastric ulcer. V. 1, and V. 2, healthy sons, number not stated. V. 3, not circumcised, aged 6. Suffered from kyphosis and extensive ecchymoses. Bled first at 9 months from a small wound in the upper lip. Later had numerous traumatic bleedings. V. 4, male, aged 2, healthy. (See Bibl. No. 268.)

Fig. 482. *Case III.* Case, observed by Merkel, in Riga, and communicated to Grandidier. I. 1—I. 4, no information. II. 2, and II. 3, healthy. III. 1, 2, 3, 5, not affected, sex not stated. III. 4, appeared healthy at birth. On second day an ulcer two inches square was found on each buttock, and blood welled out and continued to do so in spite of all treatment. Death supervened on the 11th day. The blood was finally very watery. On the eighth day some blood was seen in vomited milk. Bruises also occurred on the neck. Although this is described by Grandidier as haemophilia it seems to us very doubtful. The condition suggests purpura neonatorum with sepsis, and in the absence of a more complete history ought not to be classed as haemophilia. (See Bibl. No. 270.) We also reject the case communicated by Schulz, of Dorpat, to Grandidier, and described by him. There was no history of haemophilia in the family which belonged to Lübeck. The patient Julie E. was born in 1847, and from childhood bled from nose and right ear. Numerous ecchymoses occurred. Trifling injuries produced bruising. She had fever and pain in the splenic region, and died with symptoms of subarachnoid haemorrhage in 1860, aged 13. (See Bibl. No. 333.)

Fig. 483. *Case IV.* A most doubtful case of haemophilia, described by Grandidier in 1837, and not referred to again by him in any of his works till 40 years later, in 1877. I. 1, suffered from piles from which he died. II. 2, up to his 30th year bled spontaneously, and from wounds. III. 1, healthy. III. 2, described as a bleeder, no details. III. 3, also said to be a bleeder, the evidence being that she menstruated freely. III. 5, Conrad Neidhart, a peasant, of Hüttengesäss, near Hanau, aged 21. From early youth was asthmatic and bled copiously from mouth and nose. Early developed scrofulous glands which suppurated. After wounds there was no unusual bleeding. Haemorrhages usually occurred in spring and autumn, and were associated with palpitations and congestion of the head. Married at 20, but no children. III. 7, 8, 9, died before 30, often bled profusely. IV. 1—5, not haemophilic. IV. 6, asthmatic and cyanosed, died of fits at the age of 6 months. IV. 7, and 8, died of scarlet fever. IV. 9, menstruated at 8. (See Bibl. Nos. 100 and 372.)

Fig. 484. *Case V.* I. 1, and I. 2, not specifically mentioned, but presumably healthy. II. 2, and II. 3, were sisters, and both gave birth to bleeders. III. 1, a servant living at Alvesdorf, near Schöningen, alive, and married to III. 2, of whom there is no information. III. 3, brother of III. 2, bled to death from mouth and nose. III. 4, sister of III. 2, no information. III. 6, Jacob G., in Alvesdorf, bled to death in his 30th year from a small scratch on the neck. IV. 1, Heinrich M., aged 44. During his first year he was weakly, and later suffered greatly from rheumatic affection involving especially his feet. From early childhood he had spontaneous and traumatic haemorrhages from nose and gums, later from the urethra and bowel. The haemorrhages, which lasted two to three weeks, were frequently ushered in

by the sensation of the smell of fresh blood. The haemorrhages were so profuse that he became unconscious, and the blood was ultimately like water. In such conditions spontaneous arrest usually ensued. At the age of 3, he fell and bit his tongue and nearly died from haemorrhage. At 7, he frequently had epistaxis. At 19, he cut his thumb and bled three weeks. At the end of this time he applied a ligature to the arm, which caused the bleeding to stop. On the release of the ligature however he bled to the point of complete exhaustion for eight days, when it stopped. After the age of 31 haematuria occurred with violent renal colic. At 42 had bleeding from gums and urethra. At stool he passed more than a bucket full of blood, and then the haemorrhage stopped, "for," as he said, "I had no more." IV. 3, a bleeder, no details, except that he was living. (See Bibl. No. 214.)

Fig. 485. *Case VI.* The history of the family of a doctor living in a large town in N. Germany (afterwards stated to be Braunschweig). I. 1, and 2, died in old age. It is not stated whether they were the parents of II. 1, or II. 2, both of whom were healthy. II. 3, healthy. II. 4, died of peritonitis at the age of 40. III. 1, healthy children, sex and number not stated. III. 2, a doctor. Before 20 he had numerous scrofulous complaints, but afterwards was healthy. III. 3, menstruated early and profusely, and also bled from the anus. III. 4, a number of males, number not stated. All had "abdominal plethora." III. 5, a number of females, who were troubled with palpitations and flushings. IV. 1, male; at birth was strong and healthy. About the age of six months he developed over the false ribs on the R. side a painless swelling, which after going through the colours of a bruise, disappeared. At the same time, and without apparent cause, similar swellings appeared elsewhere. He could walk at the age of 1, but his buttocks were hard, black and swollen from bruising. At the same time, epistaxis started and frequently recurred. Spontaneous haemorrhage from the fraenum of the tongue. At 4, he fell and cut his head, and lost an enormous quantity of blood during eight days. He injured his finger and bled profusely, and a week later bled to death from a trivial injury. IV. 2, male; soon after birth he showed bruising and swelling of his scrotum, and 44 hours later was found dead in his cradle. The autopsy showed the abdomen and scrotum full of blood. IV. 3, male. At the age of 3, a haematoma showed itself over the false ribs, and this was followed shortly afterwards by similar swellings over the extremities. He had a scrofulous habitus and developed a dactylitis. Leech bites were followed by copious haemorrhage. At 4 he had epistaxis and bleeding from the mouth, both apparently without any injury. At 9, the epistaxis was very severe, and he had crops of ecchymoses. In 1842, when 13 years of age, he still showed a great tendency to epistaxis, haematomata and profuse haemorrhages from the most trivial injuries. On one occasion he had to get out of bed at night, and he fell and was found dying in the morning. At the autopsy the oesophagus was softened into a red confluent mass. During life he was of lively temperament and the picture of health. IV. 4, and 5, healthy. IV. 6, a female, married in 1855 and the mother of children, none of whom so far had developed haemophilia. (See Bibl. Nos. 69, 110 and 214.)

Fig. 486. *Case VII.* I. 2, Madame H., of Braunschweig (called Frau R. in Grandidier, 2nd edit. 1877), occasionally had epistaxis. II. 1, died after amputation of a leg. II. 2, died from small pox. II. 3, bled to death at the age of 19, and was regarded by Grandidier as a bleeder. At the age of 2 her mother found her almost dead from epistaxis, an incident which was repeated in the 5th and also in the 7th year. Menstruation appeared in the 13th year and recurred every fourth week. It was very profuse and lasted eight days. At the age of 19, in 1824 (1854, Grandidier, 2nd edit.), she began to menstruate 14 days before her time, and after a period of excitement she commenced to bleed from the nose, ears and eyes. At last the blood was merely a reddish fluid, and some days afterwards she died. During the haemorrhages she often had large ecchymoses on the arms and trunk. Apart from the absence of history of the haemophilic diathesis in this family we consider II. 3 a doubtful case of haemophilia. (See Bibl. Nos. 214 and 372.)

Fig. 487. *Case VIII.* This family was observed by Grandidier about 1854. I. 1, and I. 2, no data. II. 1, a manufacturer, in a good way, blond, strong and healthy. II. 2, his wife, slender, with white skin and black hair. Neither II. 1, nor II. 2, were bleeders. II. 3, the brother of II. 2, was however stated by II. 2, to have bled to death from a wound of the tongue; age not stated. III. 1, Georg L., aged 8, of weak nervous constitution. Two days after birth he had a foul smelling eruption on his skin. Was suckled by the mother. After weaning he became weak and was late in walking. He often fell and got bruises. At 3, fell and broke his right thigh; no unusual symptoms showed themselves however. At 4 he slightly injured his gum with a drumstick, and nearly bled to death in spite of the application of styptics. At 5, fell on his nose and bled 14 days. Shortly after, he fell on his forehead and developed a large ecchymosis. His knees were frequently swollen after falls. Had bruises almost always. III. 2, male, and III. 3, female, healthy. (See Bibl. No. 214.)

Fig. 488. *Case IX.* I. 1, living, healthy, no history of haemophilia in his family. I. 2, died of an acute disease at the age of 36. II. 1, Karl O., aged 18, a bookbinder's apprentice in Bieber (Hanau province). From childhood he suffered from spontaneous epistaxis and from ecchymoses, which occasionally involved both arms down to the finger tips. The haemorrhage after leech bites lasted four days. He had a furious haemorrhage after extraction of a tooth. Two of his brothers were bleeders and had joint

swellings, the other brothers and sisters being free from the complaint. This does not appear to us to be a very convincing case of haemophilia. (See Bibl. No. 214.)

Fig. 489. *Altstaedt's Case.* Altstaedt reports the history of a family very briefly, the data being given, partly in his text and partly in his *Ahnentafel.* I. 1—4, no data. I. 5, and I. 6, died in old age. I. 7, died old. I. 8, died very old. I. 9—12, no data. II. 2, died of apoplexy. II. 3, no data. II. 5, healthy family, number and sex not given. II. 6, no data. II. 7, frequently had epistaxis, died at the age of 55 of cardiac failure. II. 10, died of cancer. II. 11, died in childbed. III. 2, healthy. III. 4, presumably healthy. III. 5, and III. 6, unaffected. III. 7, Karl K., a bleeder, no details. III. 8, 9, 10, three brothers of III. 7, healthy. III. 11, healthy. III. 12—16, healthy. IV. 2, Willy, a bleeder, died of scarlet fever at the age of 6. IV. 3, Helmuth, aged 11, a bleeder, affected since his earliest childhood with subcutaneous haemorrhages from trivial injuries and with bleeding from the gums, bowel and occasionally from the urinary tract. Haemarthrosis frequent. Contracted scarlet fever, and during convalescence sustained a haemarthrosis of left middle finger. IV. 4, Otto, not a bleeder. IV. 5, Henry, aged 4, a bleeder, like IV. 3, but his joints had not been affected so often. He contracted scarlet fever at the same time as IV. 2, 3, and 4, and on seventh day of disease sustained a severe intestinal haemorrhage which lasted three days. IV. 6, Carla, healthy, aged 3. IV. 7, Hans, aged 4 months, had not shown any symptoms so far. IV. 9, marked in Altstaedt's pedigree as a female bleeder, no data, not mentioned in text. (See Bibl. No. 876.)

Fig. 490. *Grant's Case.* Six male bleeders in four generations. One of them, IV. 4, was reported by Dr Grant, who has kindly supplied for this article the additional data from which the pedigree has been constructed. The cases occurred in the Ballachulish district and Glencoe (Scotland). I. 1, stated by the family to have been a bleeder (no details). II. 1, 2, 4, 5, normal. III. 3, and III. 4, bleeders. Had copious haemorrhages from cuts, especially when young. III. 8, had haematomata and epistaxis, died of haemorrhage from bursting of a haematoma of the thigh, seen by Grant. III. 10, had epistaxis and haematomata. IV. 3, Grant's patient. Cut foot and bled very profusely. Oozing ultimately ceased after administration of ovarian tabloids. (See Bibl. Nos. 771 and 772.)

Fig. 491. *Lemp's Case.* III. 1, and 2, healthy. IV. 1, Francis Krick, 1833—1857. At 1 year old he bled from leech bites in the neck;—the bleeding was controlled by pressure after 12 hours. At $2\frac{1}{2}$ a large haematoma appeared on forehead from a fall. It was opened and bled for three days. At 3 there was dangerous bleeding from a "pin-point" wound on the margin of the tongue. Whenever he took food he vomited blood; the bleeding stopped on eleventh day, but patient was pulseless and thought to be dead. At 6 he slightly abraded his R. forearm; no bleeding, but the whole "antibrachium" became greatly inflamed and swollen, and he was left with "ankylosis" of the little finger. At 7 he bled for 13 days from a wounded head. At this time also he had an attack of haematuria. At 9 he knocked the L. side of his face so badly as to make him unconscious. A vast haematoma resulted, which in 12 days extended as far as the abdomen. At 13 his R. ear suppurated after 13 days' bleeding, the result of injury. At 17 he fell and got an haemarthrosis of R. knee. At 19 three days' bleeding from an injured hand. At 20 he fell and injured his hip. Eight days later a great swelling arose about his R. hip. He neglected it and became ill for 18 months and was left with a swollen thigh, disabled leg and deformity of the foot. He persisted in working while unwell and his thigh swelled up again enormously. After suffering great pain gangrene ensued and death. At the autopsy: the swelling was found to be due to great masses of blood clot disintegrating the muscles. No ruptured vessel was found. A similar haematoma was found occupying the position of the iliacus muscle. This case is of interest, in that the autopsy was conducted by Virchow. IV. 2, healthy. V. 1, living, aged 26. In good health but many years ago was subject to similar open and subcutaneous haemorrhages. He is described as the great grandson of the brother of the maternal grandfather ("solus pronepos fratris avi materni"). In addition to the foregoing, certain near "ascendants" of the mother (propinquos matris adscendentes) were subject to bleeding from the nose, lungs, stomach and anus. (See Bibl. No. 236.)

Fig. 492. *Baum's Case.* Blood investigations on two haemophilic brothers. No history of the disease in other members of the family. II. 1, Willy W., aged 10, bit his tongue at the age of $2\frac{1}{2}$ years and bled so long that the services of a doctor had to be requisitioned. In succeeding years he frequently bled from the gums and after tooth extraction. Epistaxis and bruises were frequent. For some years he had been under observation for haemarthrosis of the knee. He was admitted into the Klinik at Kiel for a swelling of the ankle, which had developed after playing. In the course of Baum's experiments venesection was performed, and the consequent haemorrhage lasted several days, soaking through the dressings. His brother, II. 2, aged 8, bled severely after an operation for phimosis at the age of 9 months. He was frequently bruised and had bled from the gums. While in the Klinik for investigation he cut his hand superficially with a piece of glass and bled for 24 hours. Venesection on this boy also produced serious bleeding for three days. Baum also describes a third case—a medical student, aged 20, from Westphalia. From infancy he had been subject to epistaxis. Cuts were followed by an abnormal degree of bleeding and bruises were very common. At the age of 10 slight swelling occurred in the L. ankle and later in the R. elbow. He had never been under the care of a doctor, however, till the present occasion, when he had

bled for two or three days after a tooth extraction. The maternal grandfather was stated to have once bled after tooth extraction and the mother was troubled with epistaxis. (See Bibl. Nos. 877 and 878.)

Fig. 493. *Treves' Case.* Treves gives a short account of a girl, V. 1, aged 6, the subject of great haemorrhage after tooth extraction. Information was obtained from the mother, IV. 10, and the grandmother, III. 7. A pedigree is given with a short statement as to whether the individuals were affected or not. The family had resided in Essex for several generations. Assuming that the cases described as bleeders were such it will be noted that affected males propagated the disease in five instances, viz. I. 3, II. 2, III. 1, III. 3, IV. 11. I. 1, and I. 2, stated to be healthy. I. 3, described as a bleeder, no details. II. 2, a bleeder. II. 3, a bleeder, not married. II. 4, healthy, also her children, III. 17. III. 1, III. 3, bleeders. IV. 1, bled to death. IV. 7, bled to death, not married. IV. 10, and IV. 11, were first cousins. IV. 11, had almost fatal haemorrhages on three occasions, twice from cuts on the fingers and once from tooth extraction. IV. 13, IV. 14, bleeders, not married, no details. IV. 24, bleeder. V. 1, Sir Frederick Treves's patient, Florence P., aged 6, admitted into the London Hospital, Jan. 26, 1885, for obstinate bleeding from the mouth. Two days previously the R. second molar had been removed without any considerable haemorrhage at first. Within one and a half hours, however, bleeding began, and the socket had to be plugged. On admission she was completely blanched and watery blood was issuing from her mouth. She had never bled before this nor had she ever suffered from ecchymoses. In the hospital she bled three times from the socket which had to be plugged. She was discharged on Feb. 8. She was of fair complexion with blue eyes and light hair, and her skin was singularly smooth and delicate. We have consulted the notes of this case in the London Hospital Registrar's records and find some discrepancies with the above. I. 3, is not stated to be a bleeder, nor is III. 3. The evidence that IV. 24 was a bleeder was considered doubtful. As Treves's description was published later, however, he no doubt had additional data. The family were living in Saffron Walden, Essex. (See Bibl. No. 510.) We have, as this goes to press, come in touch with this family again, and a pedigree bringing it up to date will be given at the end of this series.

Fig. 494. *Kinnicutt's Case.* A carefully recorded case of a severe example of haemophilia, V. 1, which the author considers may have arisen *de novo*. Of the first four generations it is stated that parents and grandparents of V. 1 had been known to Kinnicutt for many years. The father, paternal grandfather, great grandfather and great great grandfather were free from haemophilia. The mother and her direct ancestors for four generations were similarly free. "The grandmother on the paternal side was free from the disease and was a first cousin of the grandfather on the same side, *i.e.* the paternal great grandfather and the maternal great grandmother on the paternal side were brother and sister." Patient's father and grandmother on paternal side were diabetics. III. 2, 3, 6, not bleeders. V. 1, L. P., a severe case of haemophilia as shown by following evidence. At birth he was noticed to have a thin ivory-white skin. At the age of 5 months his mother noticed a swelling of his leg which was discoloured. Somewhat later a blackish lump as big as an egg appeared on his chest spontaneously and lasted several weeks. At 2 had epistaxis requiring plugging. From this time until he was 5 he was practically never free from black and blue spots on various parts of his body. At 5 he began to suffer from "inflammatory rheumatism," which compelled him to stay in bed for weeks at a time. An attack of epistaxis at 8 years of age almost cost him his life. His whole thigh swelled as a result of a blow. Next year, a coryza was followed by oozing of blood from his nose and this lasted for weeks. Then his left hand became swollen and nearly black. Dentition occurred at the usual time but was not associated with untoward haemorrhage. He was first seen by Kinnicutt in 1897 and frequently from then until his death in 1904. He was suffering from suggillations, and swelling of L. knee. Kinnicutt found him of slight physique with an unnaturally thin skin through which the veins were visible. A year later, another joint attack. In 1900, R. knee affected and severe haematuria. In 1902 during the summer and autumn large interstitial haemorrhages occurred in upper and lower extremities of the left side. In Oct. of the same year he was seized with severe pain in the R. half of abdomen and soon exhibited a deathly pallor. A large interstitial haematoma became palpable, occupying nearly the whole of the R. abdominal wall. Between 1902 and 1904 severe haematuria, effusion into L. elbow and a constant succession of effusions into both knee joints which thoroughly crippled him. In Feb. 1904, after walking on his crutches for a short distance he complained of sudden acute pain in the abdomen. He became blanched and showed all the symptoms of grave internal bleeding. At first no mass could be felt, but on the seventh day an intra-abdominal tumour was palpated in the R. inguinal region. Recovery was slow. In the summer of the same year he had a sudden recurrence of acute abdominal pain and died 24 hours later. No autopsy. (See Bibl. No. 790.)

PLATE XLIV. Fig. 495. *Wright's Case I.* Haydon family, living in London at 101, Princes Street, Edgware Road. The individuals studied by Wright were II. 12, III. 11—20, and IV. 2—12. I. 1, and I. 2, healthy. II. 1—5, five sons reported to have all died of haemorrhage. Order of these sons in succession not known. II. 6, Ward, married to II. 7, healthy. II. 8, Lacy, married to II. 9, both healthy. II. 10, Crockett, married to II. 11, both healthy. II. 12, Henry Gomm, aged 72, healthy, married to II. 13, who died of haemorrhage at the climacteric. III. 1—5, five males, normal. III. 6, and 7, two daughters, normal. III. 8, Norris, married to III. 9, both healthy. III. 10, healthy. III. 11, John Gomm, aged 41 in 1894, a bleeder, had bled all his life, and on many occasions had nearly lost his

life. Had been a patient in St Mary's Hospital. Knee stiff. In 1898 had two dangerous bleedings, one from cut on finger with a piece of glass, the other from the gums. Blood examination showed diminution of polynuclears and delayed coagulation time. III. 13, Welch, married. III. 14, Ellen Gomm, aged 40. III. 15, Haydon, healthy. III. 16, Emma Gomm, wife of III. 15, aged 36, suffered from floodings after confinements. Her blood count showed 4000 leucocytes per mm.^{3} and consisted of 42 °/$_\circ$ neutrophiles and 56 °/$_\circ$ lymphocytes and mononuclear leucocytes. III. 17, Bowditch, married to III. 18, Jane Gomm, aged 32. III. 18, had had floodings after confinements. Her blood showed a diminution in the total number of leucocytes with 46 °/$_\circ$ neutrophiles and 54 °/$_\circ$ lymphocytes. III. 19, King, married to III. 20, Kate Gomm, aged 27. III. 20, had menorrhagia and flooding. III. 21, daughter, unmarried. IV. 1, several sons. IV. 2, son, died in infancy. IV. 3, healthy. IV. 4, daughter, suffered from epistaxis. IV. 5, Jessie Haydon, aged 10½, healthy. IV. 6, Harry Haydon, aged 9, a bleeder. At 19 months bumped his forehead. A haematoma formed and it was opened. He bled for 14 days, being then in St Mary's Hospital. Afterwards he was repeatedly a patient at the hospital being admitted for epistaxis. Bled freely when milk teeth came out. Had swollen and stiff joints. Observed by Wright over a period of four years. Repeated examination of his blood showed diminution in total number of leucocytes and delayed coagulation time. IV. 7, Thomas Haydon, aged 7, a bleeder, but not so severely affected as IV. 6; haematomata and swollen joints were, however, marked symptoms. Had been twice in St Mary's Hospital with dangerous bleedings from wounds of tongue. IV. 8, Freddy, a bleeder, died at the age of 2. IV. 9, Fred Bowditch, aged 6, a bleeder. IV. 10, Ethel Bowditch, aged 2, healthy. IV. 11, Percy King, aged 3, a bleeder: had had frequent haematomata and at the age of 2 bled for four days from a cut on the tongue. He was treated in the West London Hospital and very nearly died. At 3 he bled till he was exsanguine from a scratch from a tooth. He ate plaster and mortar. IV. 12, girl, aged 1 year, healthy. (See Bibl. No. 908 with elaborations by the author for this article.)

Fig. 497. *Wright's Case II.* Cowdrey family, some of the members of which are living at Durley, Hampshire (England). I. 3, cousin of I. 2, known to have bled to death, although details are wanting. II. 1—6, six sons who died before reaching manhood. One of these, II. 1, bled to death when 18 years of age. Another known to have died after a street brawl. II. 9, 10, 11, 13, 14, 15, healthy. II. 16, P. Houghton, aged 70. II. 17, Caroline, wife of II. 16. III. 1, bled to death from a cut on his lip when 4 years old. III. 2, 3, healthy. III. 3, separated from III. 4 by 14 years' interval. III. 4—III. 9, healthy. III. 10, male, bled to death. III. 11, bled to death after the opening of a haematoma. III. 12, Cook, married to III. 13, Esther, aged 48, healthy. Seen by Wright, who examined her blood and found 12,300 leucocytes per mm.^{3} of which 60 °/$_\circ$ were neutrophiles and 37 °/$_\circ$ were lymphocytes. III. 14, Marcham, married to III. 15, Caroline, aged 46, healthy. III. 16, Glassport, married to III. 17, Elizabeth, aged 44, healthy. III. 18, Peter Houghton, was run over by a roller, and bled to death. III. 19, Richard, died at the age of 11, not a bleeder. III. 20, Harry, a bleeder, died from exhaustion after haemorrhage following a trivial cut on the knee. III. 21, Frank, a bleeder, had ecchymoses continually; ultimately died of consumption at the age of 22. III. 23, Jesse, was kicked on the mouth by a donkey and bled to death. III. 24, Edwin, a bleeder, is said to have died of cerebral haemorrhage—age not stated. III. 25, Kate Houghton, aged 28, healthy, frequently seen by Wright. Her blood showed 8400 leucocytes per mm.^{3}, consisting of 50 °/$_\circ$ neutrophiles and 48 °/$_\circ$ lymphocytes. III. 26, George Cowdrey, husband of III. 25, aged 45, known to Wright who examined his blood and found leucocytes 12,200 per mm.^{3}, 62 °/$_\circ$ neutrophiles and 35 °/$_\circ$ lymphocytes. III. 27, Wilfred, fell into a decline after loss of blood—a bleeder? IV. 1—5, healthy. IV. 6, bled to death at the age of 17. IV. 7, healthy. IV. 8, healthy. IV. 9, Frank, aged 11, not a bleeder. IV. 10—IV. 13, four daughters, named Lily (10), Ellen (7), Ethel (6) and Mary (5) Cowdrey respectively. They were all examined by Wright and with the exception of chilblains were found to be healthy. IV. 14, George Cowdrey, aged 3, a severe bleeder. Had always suffered from haematomata. Seen by Wright in 1893 and 1894. Nearly bled to death from a small wound in forehead. In 1893 had a swollen knee. In Feb. 1894 cut fraenum of lip and bled almost to complete exhaustion. In December of the same year bled profusely from cut of finger. IV. 15, Willie, aged 2½, healthy as yet. IV. 16, Jane, aged 1½ years, normal. (See Bibl. No. 908 elaborated by the author for this article.)

Fig. 521. *Wright's Case III.* Greenshields family. History of three male bleeders in two generations; family living in Parsons Green, London. Seen by Wright in 1894. I. 1, and I. 2, no information. II. 1—15, nine sons and six daughters, all of whom died young. Nothing is known of them except that some of them bled. II. 16, was hurt on the head and died as the result of an operation. II. 17, male, bleeder, had perpetual haematomata and haemorrhages. At the age of 19 he was struck on the leg by a cricket ball. Mortification set in necessitating amputation of the leg, and he bled to death. II. 18, W. S. Greenshields, a baker, 45 years of age. Had suffered from chilblains and latterly from an itching eruption of the skin. Not a bleeder nor of a bleeder family. II. 19, Mrs Janet Greenshields, wife of II. 18, aged 42. She is stated to have always menstruated profusely, and at the age of 27, 14 days after the birth of her eldest child, she had a flooding which nearly cost her her life. She also bled profusely after three miscarriages and had *post partum* haemorrhage after the birth of III. 5. Latterly she had menorrhagia, said to be due to a polypus of the uterus. Wright made an examination of her blood

(21/3/1894) and found 4400 leucocytes per mm.^{3} and composed of 1 °/$_0$ eosinophiles, 58 °/$_0$ neutrophiles, and 41 °/$_0$ lymphocytes including 6 °/$_0$ mononuclears. Her blood coagulation time was 6′45″. III. 1, girl, suffered from chilblains. III. 2, 3, 4, three miscarriages. III. 5, Willie Greenshields, a bleeder, born 1882. At the age of 1½ years he fell against a tin and a haematoma developed at the seat of injury. It was opened in the hospital and grave haemorrhage ensued. At 2½ was again in the hospital with a stiff joint. At 4 fell and bit his tongue. The haemorrhage was ultimately arrested by the actual cautery. From his fourth to his ninth year he had frequent haemorrhages. At 9½ he cut his tongue during a fall. Bleeding was at first slight but later very severe. It was temporarily arrested by perchloride of iron but restarted, and he was conveyed to the hospital where the tongue had to be clamped. This again produced a temporary arrest, but on the following evening the mother found the bed full of blood, and he was conveyed to St George's Hospital, where he bled for 15 days. Since then there had been no great external haemorrhages, but he frequently suffered from bruises, spontaneous ecchymoses and swellings of his joints. Wright carried out a long series of observations on his blood between 1894 and 1898, the general result showing a diminution in the total number of leucocytes with a low percentage of neutrophiles. The blood coagulation time was prolonged. III. 6, Maggie Greenshields, aged 10½, suffered from chilblains. Leucocytes 4250 per mm.^{3}, neutrophiles 55 °/$_0$, lymphocytes 40 °/$_0$. III. 7, Mary, aged 9, suffered from eczema. Leucocytes 8600 per mm.^{3}, 64 °/$_0$ neutrophiles, 33 °/$_0$ lymphocytes. Coagulation time 3′ 30″. III. 8, John, a bleeder. At 3 years of age fell and cut fraenum of upper lip and bled excessively. Between the ages of 3 and 6 he had frequent and copious haemorrhages from the gums and very severe haematomata. Joint swellings were also common. He bled to death from the bowel when 6 years old. III. 9, Thomas, not a bleeder, aged 6, seen by Wright, who found he had 10,200 leucocytes per mm.^{3} Two years later a count showed 7000 leucocytes consisting of 70 °/$_0$ neutrophiles and 28 °/$_0$ lymphocytes. Coagulation time 2′ 30″. III. 10, Florrie, aged 3½, not a bleeder. Leucocytes 8600 and consisting of 27 °/$_0$ neutrophiles and 70 °/$_0$ lymphocytes and mononuclears. (See Bibl. No. 908.)

Fig. 496. *E. B. Miles' Case.* Severe haemophilia in a doctor, III. 2, with similar affection in two brothers. I. 1—I. 4, had no history of haemophilia. II. 2, aged 89, alive and well. II. 3, his wife, aged 69, alive and well. III. 1, W. P. M., died at the age of 2½ from uncontrollable bleeding following a punctured wound of the pharynx. III. 2, E. B. M., a doctor, aged 49. Classical example of haemophilia. From his earliest years he was liable to bruise on the slightest provocation, and suffered from prolonged and nearly fatal haemorrhages on many occasions. Great bleeding followed extraction of teeth. He had innumerable attacks of swellings in his elbows, knees and ankles, leaving him partially crippled. He had to be very careful of himself all his life and in spite of this had been very frequently incapacitated. Subcutaneous haematomata had caused contractions in his arms. In later years he has not bled externally so much as formerly, but was still liable to large extravasations of blood under the skin from insignificant causes. Practically unable to follow his profession. In 1910 he was several weeks in bed with symptoms suggesting an haemorrhage into the cauda equina. III. 4, female, aged 48, not a bleeder, married, without issue. III. 6, female, aged 47, unmarried, not a bleeder. III. 7, female, died of meningitis at the age of 22. III. 8, female, single, aged 43, not a bleeder. III. 9, male, not affected, married, and had healthy family of five girls and two boys, IV. 4—IV. 10. III. 11, alive, aged 38, a typical bleeder like his brother, III. 2. IV. 1—3, perfectly healthy. IV. 1, female, aged 17. IV. 2, male, aged 8, had never bled. IV. 3, aged 1¼ years, had no symptoms of the disease. (See Bibl. No. 896.)

Fig. 498. *Ulrich's Case.* Short account of great haemorrhage following the application of leeches to a bleeder. No mention of ascendants. II. 1, male, aged 24, an apothecary, had previously suffered from grave haemorrhages from the most trivial cuts and from painful swellings of his joints. When one of his elbows became involved he permitted the application of three leeches on account of the intolerable pain and in spite of his previous experiences. Blood flowed in a continuous stream from the bites, and in spite of the application of many remedies including the actual cautery he became exsanguine on the thirteenth day and looked as if he would die, but at that time the bleeding spontaneously ceased. II. 2, the younger brother of II. 1, "had the same morbid tendency to haemorrhages"—no details. We are indebted for our abstract of this very inaccessible paper to Professor Ehrlich of Frankfurt. (See Bibl. No. 43.)

Fig. 499. *Darblade's Case.* The history of a weaver, III. 4, with alleged haemophilia in his ascendants. I. 1—3, healthy. I. 4, a female, had the disposition, no details. II. 2, healthy, died at the age of 63. II. 3, had uterine haemorrhage and lost much blood after confinements and died in one of these attacks. II. 4, brothers of II. 3, died of haemorrhage, number not given. III. 1—3, were bleeders; no details except that one bled to death after tooth extraction. III. 4, Louis R., a weaver, aged 57, living at Puteaux near Paris. During his first dentition he bled so badly that it was necessary to requisition the services of a doctor. He began to bleed spontaneously at the age of 12, being attacked with haematemesis, haematuria, haemoptysis and haemorrhage from the bowel. Epistaxis was infrequent. These haemorrhages lasted four or five days and left him very enfeebled. In 1858 he had grave bleeding from the bowel and was treated for it in the Charité. He bruised very easily, and after the application of a leech it was necessary to use the actual cautery before the bleeding could be arrested. In 1860 he was in the hôpital Beaujon bleeding from a slight wound on the head, and at a later period with copious haemorrhage from the urethra and bowels.

He himself had never had painful joints although his brothers were affected. III. 4, was the father of 14 children, IV. 1—7, eight of whom died young, none of them were, however, affected. A grandchild, sex not stated, had frequent haemorrhages from trivial injuries. A large part of Darblade's thesis is taken up with a general account of haemophilia. (See Bibl. No. 261.)

Fig. 500. *Groves' Case I.* Mr Groves, of Bristol, has described the case of a bleeder, IV. 2, and has published a pedigree of the family, who have lived for a long time at Kingswood near Bristol. The order of birth in our pedigree differs from that published by Groves, the alterations being made as a result of a correspondence with Mr Groves in Feb. 1909. I. 1, and I. 2, presumably healthy. II. 2, was well and healthy and had six children, viz. three girls and three boys. Of the latter, two were bleeders. II. 3, the brother of II. 2, was a "bruiser and bleeder," and died of bleeding from the extraction of a tooth. III. 1, Hannah, born 1867, died at the age of 14 of heart disease and dropsy. She was not a bleeder. III. 2, Mrs Webb, born 1869, mother of Groves' patient. She was presumably healthy. III. 4, Samuel, born 1871, a bleeder, died of epistaxis at the age of 17. III. 5, Edward, born 1873, not a bleeder; married and had six healthy children, three boys and three girls. III. 7, Mrs Curtis, born 1879, healthy. III. 9, Harry, born 1883, a bleeder, died aged 6 from the bleeding from a finger which had been crushed in a door. IV. 1, bleeder, bled to death when 4 months old, no details. IV. 2, William Webb, over 16 years of age, a bleeder who had had almost every variety of haemorrhage. In childhood he was constantly bruising and bleeding. At 11 he fell and cut his lip and bled for a week; at or about the same time he hit his R. elbow and sustained a large swollen bruise extending down his arm. A few days later he noticed that all his fingers became "clawed," *i.e.* they were bent up into the palm. A doctor bound his arm up in splints for four weeks and at the end of that time his forearm was weak and shrivelled and supination was impossible and had continued so. At 13 years he was hit on the knee by a stone; great swelling of the knee ensued and had recurred frequently since. At 15, and again at 16, he had haematuria lasting three weeks and one week respectively. The day before he was seen by Groves he had slipped off his bicycle, but alighted on his feet in such a manner as to give them a slight twist or strain. A large hard and painful swelling developed over the R. thigh, probably a deep-seated haematoma under the quadriceps cruris muscle. Both knee joints grated when moved and both ankles were stiff and painful. Mr Groves informs us that he subsequently developed phthisis but the haemoptysis was not unusually severe. IV. 3, male, not a bleeder. IV. 4—9, three males and three females, ages 14, 12, 10, 6, 5, 3, all healthy and alive except IV. 8, who died of measles. IV. 10, cousin of IV. 2, Harry Curtis, now 5 years old—a bleeder. When three days old he had severe bleeding from the umbilicus which continued more or less for three weeks. At the age of 2 he fell and cut his lip and bled for three days, a similar accident happening at the age of 5. Local styptics and the cautery only made matters worse, and he was admitted into the infirmary, where he remained ten days. Fourteen days before his nose bled for three days without stopping, and at the same time without any known cause he had pain and swelling in the right knee and ankle joints. (See Bibl. No. 834.)

Fig. 502. *Groves' Case II.* The history of two bleeders, brothers (IV. 2, IV. 3), who belonged to a family of bleeders. I. 2, healthy, had two healthy daughters, both of whom bore bleeders. III. 1—4, four healthy males. III. 5, male bleeder, died of epistaxis when 6 years old. III. 6, male bleeder, died of epistaxis at the age of 10. III. 7, Mrs H., healthy, had had four sons and six daughters; three of the sons being bleeders. III. 9, 11, 13, 15, 17, 19, healthy females all married, no record of families. III. 21, unmarried but healthy. III. 22, a bleeder, often had haemorrhages. At 24 years of age he slightly injured his gum with a nail. He was a shoemaker, and kept the nails in his mouth as is the custom. Bleeding ensued, and he bled to death on the 20th day in the Royal Infirmary, Bristol. IV. 1, male, healthy. IV. 2, male, bleeder, Samuel H., now 18 years old. He had the usual history of inordinate bleeding from trivial cuts and wounds, together with frequent joint troubles in his knees. Both knees were normal on inspection and measurement, but round the edges of the articular cartilages some nodular thickening could be felt, and on moving the joints cracking could be heard and grating felt. IV. 3, Cleophas H., aged 14, a bleeder. Sustained many haemorrhages when young. At the age of 9 he bit his lip and had to be taken to the Children's Hospital, where he was "strapped down in bed" for three weeks, no doubt to keep his hands from his mouth. At 7 he became entangled in a parliamentary election crowd, and in order to extricate him his father pulled him by the left arm. This caused great bruising, swelling, and stiffness of the elbow and wrist, and when the swelling disappeared he was left with a wasted limb. The left knee grated, and was two inches bigger in circumference than the right. IV. 4, aged 6, a bleeder; his mother giving a long account of bleeding from cuts, large and frequent bruises, and swollen knee joints. IV. 5—10, six healthy females. (See Bibl. No. 834.)

Fig. 501. *Reinert's Case.* Reinert has recorded the history of a family living in the neighbourhood of Osnabrück. I. 3, and I. 4, named S., were living at the beginning of the 19th century. Both reached old age, but Reinert was ignorant whether either was a bleeder. They had three sons and two daughters. II. 1, Heinrich S., died in 1818, being then about 30 years of age, of a blow on the head. The seat of injury was blue and a few drops of blood came from his ear. Symptoms of cerebral compression set in, and he died 23 hours after receiving the injury. II. 2, his wife, not a bleeder. Her parents, I. 1 and I. 2,

were also healthy. After the death of II. 1, she married II. 3, and had by him a healthy family. II. 5, Gerhard S., II. 7, Wilhelm S., II. 9, Marie S., presumably not affected. It is stated, however, that all three had numerous descendants, but they could not be traced. In a footnote, however, Reinert (p. 11) adds that Dr W. informed him that among them several haemorrhages had occurred. II. 11, Dorothea S., healthy. III. 1, male, died soon after birth. III. 2, stated to have existed, but no information. III. 4, Caroline, aged 50, according to Reinert, a bleeder. She had dark hair and brown eyes, and as a child was healthy. In her 7th year after a tooth extraction she bled off and on for 14 days, the haemorrhage ultimately ceasing spontaneously. She recovered quickly, but after that time was anaemic. In her 14th year some leeches were applied to her neck on account of hoarseness. The haemorrhage was so great that it took her some weeks to regain her strength. Charpie was applied both times, but without success. She menstruated regularly but profusely, the period lasting 5—8 days, and being accompanied by headache. Otherwise she was strong and healthy. She had had eight children, but no information is given as to her condition during confinements. III. 5, Marie, aged 53, apparently older than her sister. During her childhood she also bled like her sister after a tooth extraction, and her menstrual condition was similar. After a miscarriage she had a very bad haemorrhage; according to Reinert she was a bleeder. III. 7—11, healthy. III. 12, 13, 14, for the most part healthy (see II. 5—II. 9). III. 15, male. In youth he was bitten on the finger by a rat, and the haemorrhage which ensued could only be controlled by a stitch. In his pedigree Reinert marks him as a doubtful bleeder. III. 18, and 19, females, died young. With regard to III. 20, III. 21, and III. 22, Reinert states (p. 7) that "all were married and had numerous descendants," but on p. 10 in his pedigree he says that, of the six daughters of Dorothea, one daughter was married. We have followed his pedigree III. 24, Engel, not a bleeder, married firstly to III. 23, and secondly to III. 25. IV. 1, died at the age of 2; IV. 2, at the age of 9, both of croup ("an der Bräune"). IV. 3, died of umbilical haemorrhage. IV. 4, the oldest living son, aged 26. At the age of 5 he had a small boil behind the L. ear. It burst by itself and commenced to bleed. A doctor was sent for and he was able to stop it. At 9 he bled, with remissions, for three days, after the removal of a carious tooth. At 11 he stabbed himself accidentally with a sharp-pointed knife above the knee. Blood came out in a fine stream. The application of a small piece of "Feuerschwamm" and of a ligature soon arrested the haemorrhage. Later he received a small wound on the inner aspect of the lower lip, near the art. coronaria. The blood clotted well, and a stitch finally arrested it. Three years before, after he had been walking briskly during the cold weather, a painful swelling of the R. knee set in but went down in three weeks. Reinert considered IV. 4 to be a bleeder. IV. 5, bled badly after the most trivial injuries. He had twice had carious teeth removed. The blood which clotted well came slowly, and stopped after plugging with lint, but restarted and lasted three days, leaving him blanched. Considered by Reinert to be a bleeder. IV. 6—8, healthy. IV. 9, and 10, healthy. IV. 11, Wilhelm, considered by Reinert to be a bleeder. He twice had a tooth extracted, being followed the first time by seven days', and the second time by nine days', haemorrhage. When about 18, while trying to fire a rocket, something went up his nose. He bled three-quarters of an hour and then collapsed. After this, especially at night, he used to bleed from the gums, and a scorbutic condition of the mouth developed. Latterly he bled for two days from some leech bites on his leg. IV. 12—14, healthy. IV. 15, female, tuberculous, died of haemoptysis and epistaxis; figured in Reinert's chart as a bleeder. Reinert himself says that there was no other history of haemorrhage in this person, but that her one and only haemorrhage "perhaps" points to heredity. IV. 19, Gerhard, often had spontaneous epistaxis. Had injured himself several times, and on each occasion a stitch was required to stop the bleeding. At the age of 15 he died as the result of being thrown out of a cart on to his head. IV. 20, died of meningitis, aged 10. IV. 22, and IV. 24, two healthy females. IV. 25, Wilhelm, aged 20, half-brother of IV. 19. Up to the age of 5 he appeared unhealthy and suffered from skin eruptions. At 6 persistent epistaxis. At 14 he cut his thumb; the bleeding soon stopped of itself but several days later began again, and it was necessary to insert a stitch before it ultimately stopped. Considered by Reinert to be a bleeder. V. 2, aged 9; owing to his "lively temperament," had occasionally received injuries. During the year 1869 he fell on his forehead, and on another occasion another school boy hit him on the head with an inkpot and stitches were required both times, as also for a cut on his thumb. Reinert remarks on the absence of ecchymoses in all the cases. The evidence of haemophilia in the majority of Reinert's cases is unconvincing. (See Bibl. No. 309.) On p. 11 Reinert describes a typical bleeder, a boy of high Hanoverian family, but does not mention his family history. This boy has also been referred to by Cammann (see Bibl. No. 324) and Strohmeyer (see Bibl. No. 275).

Fig. 503. *Donkersloot's Case.* Three very doubtful cases of haemophilia in males. I. 1, aged 60, died of metrorrhagia. I. 2, healthy. II. 1, aged 20, died of epistaxis, brought about by an apple which fell from a tree and struck him on the nose. II. 2, contracted pleurisy and was venesected in the arm; great haemorrhage into the tissues, gangrene, amputation; death from haemorrhage. II. 3, a small boy, lost 12 ounces of blood during an operation for ingrown toe nail. Donkersloot also refers to the case of three sisters, who suffered from epistaxis and great haemorrhage after tooth extraction. (See Bibl. No. 182.)

Fig. 504. *W. R. Steiner's Case.* Alleged haemophilia in a negress with unconvincing family history.

I. 1, Nancy P., a Scotchwoman, married to I. 2, a Guinea negro. II. 1, John Thomas T., an Irishman, married to II. 2, who is described as having been a bleeder from early life till her death, which took place in 1898. With the idea that she had too much blood the doctor used cupping or leeching twice a year. II. 1, and II. 2, had 14 children, ten males and four females (generation III, but IV. 1, the source of information, could only name eleven of them, viz. seven males and four females). III. 1, Susan Emily, died young. III. 2, died young. III. 3, James Isaac, bleeder, died young; no actual data. III. 4, Thomas Henry S., healthy. III. 5, Sarah Anne, "bleeder," the evidence being that she bled from the nose occasionally. She died, aged 60, in 1898 of asthma, dropsy, renal and cardiac disease. III. 6, Thomas S., second husband of III. 5. III. 7, Mose, died young. III. 8, Mose, stated to have been a bleeder, died of haemorrhage from mouth and nose, aged 34, in 1895. He had suffered from early childhood. III. 10, William, died young. III. 11, William, a bleeder, no data. III. 12, Laura H. III. 13, Geo. Washington, a bleeder, no data. III. 14, Victoria. III. 15, Mary Lizzie, a bleeder, no data[1]. III. 16, Robert R. III. 17, John Thomas, died aged 57. IV. 1, Alverta S., a "bleeder," the evidence being that she frequently bled from the nose till she was 16 years old, but had had no attacks afterwards. IV. 3, died young. IV. 4, 5, 6, not affected. IV. 7, IV. 8, died young. V. 1, V. 2, had occasional attacks of epistaxis and bled considerably from the slightest cuts and bruises. V. 3, Maud Estelle, a bleeder, died young; no data. V. 4, Alverta, aged 14, a dark-skinned negro girl, Dr Steiner's patient. Bled from trivial scratches from early childhood. No great amount of blood lost. No epistaxis till present illness, which began $2\frac{1}{2}$ months before, when she woke up bleeding at the nose; gums swollen, but no bleeding. Some purpuric spots developed while in the hospital. No joint affections. Discharged in fourteen days greatly improved. (See Bibl. No. 733.)

PLATE XLV. Fig. 505. *Port's Case.* A shortly described account of three brothers. I. 1, unaffected; I. 2, thought the tendency came from her mother. She, I. 2, had had severe "floodings," and bled much when one of her teeth was drawn. II. 1, 2, and 3, died young unaffected. II. 4, died of haemorrhage from a bitten tongue at the age of 2. II. 5, at the age of 4 died from a slight scalp wound. II. 6, aged 16. He began to bleed from the nose at the age of 3 and nearly died of it at 5. At the age of 10, he had a molar extracted and bled several days. Some time later, he had smallpox without a tendency to haemorrhage. During the last three years he had had repeated swelling of elbows and knees, and an exploratory puncture into two joints, made shortly after the onset, disclosed pure blood. II. 7, aged 10, weak, but not affected. (See Bibl. No. 463.)

Fig. 506. *Kercksig's Case.* History of a tailor, II. 1, with alleged haemophilia in a brother, II. 2. I. 1, and I. 2, no history. II. 1, Kercksig's patient, aged 32, a tailor, of slender build and fine white skin, had suffered from childhood from haemorrhages, especially from the gums, nose, and bowels. Enormous masses of stinking blood were vomited or passed *per rectum.* He bled till he was exsanguine and no remedy appeared to exert the slightest beneficial effect. Very trivial injuries produced large suggillations on his skin, and a prick from his needle could only be arrested by acupressure. Death took place at the age of 32, from several violent intestinal haemorrhages. II. 2, suffered from haemophilia, no data. II. 3—II. 6, unaffected. We are indebted for the abstract of this very inaccessible publication to Professor Ehrlich, of Frankfurt. (See Bibl. No. 140.)

Fig. 507. *Bjørkman and Liedbeck's Case.* History of two brothers, Mellgren by name, living in Sweden. Bjørkman described the one case, II. 1, and Liedbeck the other, II. 2. Our abstract was made by Professor Karl Petrén, of Lund, and the cases were regarded by him as genuine haemophilia, a view in which we concur. I. 1, suffered from epistaxis, and died of pulmonary consumption. I. 2, no information. II. 1, a boy 12 years old, shown to the Swedish Society of Physicians in 1846, by Bjørkman. From birth onwards he was very liable to haematemesis and bled profusely from trivial injuries. In 1842 he had a swollen ankle for which leeches were applied. The haemorrhage following was so great that it could not be arrested for eleven days. In the following year leeches were again applied for a traumatic swelling of the eyelid, and he bled for eight or nine days. In 1844 he very nearly bled to death from haematemesis induced by swimming. In 1846 he received a blow on the forehead and bled twelve days. His final illness is described in Bjørkman's second communication. A tooth was quite loose and the patient extracted it with his fingers. Blood commenced to flow from the socket, and he bled to death on the ninth day. Towards the end the blood was very watery. In the intervals between his haemorrhages II. 1 was perfectly healthy. The autopsy showed great anaemia and an extravasation of blood under the skin of one thigh. II. 2, Hans Victor Mellgren, described by Liedbeck as being 16 years of age in 1847, although Bjørkman describes him as the younger brother of II. 1, who in 1846 was 12. II. 2, according to his own statement and that of others, was frequently troubled with violent epistaxis, and sustained enormous effusions of blood after most trivial injuries. For example, on one occasion a slight knock on the orbit caused such an extravasation of blood that he was unable to open his eyes for a week or two. In 1846 he was under Liedbeck for a swelling of a knee. On August 12, 1847 (misprinted 12/2/1847 in one part of the paper), he received a bruise over the temporal muscle, the exact cause of the bruise being undetermined. After the accident different witnesses alleged that he walked to the town but on his

[1] Owing to an oversight III. 15 is not marked as a bleeder on the plate.

way home he was seen to stagger like a drunken man. On the 14th, he fell into coma and died on the 16th. The autopsy, made by Liedbeck, showed that the whole of the temporal muscle was involved in a sanguineous extravasation. Effusion of clotted blood was also found round the pons Varolii, the optic nerves, and the right hemisphere of the cerebellum. There was no evidence of fracture of the skull. The right knee joint contained synovia mixed with blood. No other evidence of disease. III. 3, the youngest brother, not a bleeder. (Liedbeck.) (See Bibl. Nos. 161 and 173.)

PLATE XLV. Fig. 508. *Grusche's Case.* History of a family of bleeders, two of whom, IV. 25, IV. 26, were in the surgical clinic in Halle. I. 1, Riel, not affected. I. 2, Frau Riel, not affected. They had three boys and two girls, one of the boys being a bleeder. II. 1, Karl Riel, not affected. II. 3, Wilhelmina Riel, normal, married II. 4, Exner, also healthy. II. 5, Dorothea Riel, normal. II. 7, August Riel, healthy. II. 9, Wilhelm Riel, a bleeder, and bled to death from the gums when 11 years of age. III. 1, many children, healthy. III. 2, Wilhelm Exner, bleeder, and bled to death from the gums at the age of 2. III. 4, Gustav Exner, normal, married, and had twelve children, IV. 2—IV. 13, all of whom were healthy. III. 5, Pauline Exner, healthy, married III. 6, Stockmar. III. 7, Anna Exner, healthy, married III. 8, Hoetzel, also healthy. IV. 1, many children, healthy. IV. 14—23, Stockmar family. IV. 14—18, died young, but showed no symptoms of haemophilia. IV. 19, not a bleeder. IV. 20, IV. 21, IV. 23, three male bleeders, no information. IV. 24, Martha Hoetzel, died at the age of 3 months. She was younger than Anna, IV. 27, but in Grusche's chart she is represented as older. IV. 25, Paul Hoetzel, a bleeder, aged 15, a locksmith in Halle. His parents early observed that bruises were produced by the slightest injuries and trifling wounds produced severe and prolonged haemorrhages. On several occasions he almost bled to death. Bleedings occurred from gums and nose, and often lasted for eight days before they were arrested. At 2, he received a minute scratch on the tongue and bled for a week. Six months later an attack of epistaxis lasted twelve days; after another interval of six months he bled for eleven days from the gums. At the age of 9 he knocked his head against a bedstead during the night and sustained a great bruise. On the fourth day the lesion started to bleed and continued for fourteen days. At 10 he bled for fourteen days from a tooth extraction, and again at 12 and at 13. Swollen joints and rheumatic pains now presented themselves, the joints affected being the ankles, knees, hips, and shoulders. At 15 he knocked the end of the middle finger of the left hand against an iron post. Swelling and bruising ensued, but not much blood was lost at the time. He incised the swelling himself, and a quantity of dark-red, half-clotted blood came away and as oozing continued, he came to the clinic, where the swollen finger was dressed, but the bandages were still bloody for nine days. IV. 26, Walther Hoetzel, aged 9, a bleeder, suffered almost in exactly the same way as his brother, IV. 25. He first bled at 9 months from an almost invisible injury to his gums, the bleeding lasting eighteen days. At 5 he almost bled to death from a wound of the head, caused by a piece of glass. At 7 he bled for nine days after tooth extraction, and had several bad attacks of epistaxis. According to his mother the blood once soaked through the mattress to the floor. At 6 he broke one forearm and at 7 the other. On both occasions great swelling and discoloration. Swellings of joints were also present. At 9 an empty cask fell on his left shoulder and knocked him down against a stone, which injured his R. hip. He was conveyed to the hospital suffering from haematomata of the abdominal muscles and the infraspinous fossa of the scapula. During his stay in the clinic absorption of the blood took place. IV. 27, Anna Hoetzel, aged 12, living, healthy. V. 1, 2, 3, 4, numerous children in four families. VI. 1, family, healthy. In addition to this family Grusche describes six other isolated cases of bleeding. These were as follows:—(1) Carl Keller, aged 31, a thatcher, of Cöthen, admitted with left inguinal hernia, for which he was operated upon. Great swelling of penis occurred, and wound became like a haematoma and had to be opened up. Diffuse blood swellings on thighs and buttocks. He subsequently gave a history of bleeding from trifling injuries. No history of haemophilia in the family. (2) Hermann Richert, aged 40, a painter, with a history almost identical with that in Case I. No family history of haemophilia. (3) Carl Opitz, seaman, aged 57, with haematoma of rectus abdominis muscle. Until he was 15 he had epistaxis, and bled easily. A sister died of excessive menstruation. (4)-Franz Putzing, tailor, aged 29. Septic wound on forearm caused by a piece of glass. Great bleeding. No family history of bleeding. (5) Oscar Bormann, aged 23, tailor, suffering from haematoma of right iliac fossa and bleeding from the mouth. Parents healthy. (6) Arthur Winkler, aged 24, carpenter, cut his hand with a circular saw; considerable bleeding. No family history. None of these six cases bear the stamp of haemophilia. (See Bibl. No. 742.)

Fig. 509. *Schliemann's Case.* Schliemann has published a case, IV. 2, which was observed in the hospital in Würzburg in 1831. II. 1, aged 16, died from haemorrhage following a blow on the nose. He had not previously shown any symptoms. II. 2, had suffered with some joint affection for many years before her death. III. 1, died of phthisis. III. 2, healthy. III. 3, and III. 4, sisters and brothers of III. 2. IV. 1, male, slipped and injured his tongue and bled to death. IV. 2, Jörg Weber, aged 13, scrofulous and debilitated. Soon after birth his body was observed to be covered with livid raised spots. These disappeared in fourteen days, but recurred till he was 11 years old. Contusions produced severe ecchymoses. He showed bloody stripes on his nates when beaten at school. He once slipped and struck

his forehead. A haematoma formed and burst, and he nearly bled to death. At the age of 11 he had a six days' haemorrhage after tooth extraction. At 13 painful swelling of left elbow. IV. 3, 4, 5, died of convulsions soon after birth. IV. 6, and 7, healthy, grown up. V. 1, aged 2, a male, had a very painful swelling of the scrotum. It burst spontaneously and discharged blood and matter. (See Bibl. No. 68.)

Fig. 510. *Rave's Case.* This is one of the earliest recorded cases of haemophilia, although Grandidier (1st edit. p. 110) was inclined to regard it as a transitional form, between true haemophilia and scurvy. Rave himself, II. 2, and three of his brothers were affected, whereas his sisters were free. I. 1, was healthy. I. 2, was scorbutic. II. 1, severely affected. In early life two of his teeth fell out and he almost bled to death, and the same fate befell him in his 16th year. The blood streamed from his mouth in spite of all remedies. One and a half years later he died of haemoptysis. Blood taken from his veins scarcely looked like blood. II. 2, Rave, suffered from his 5th to the 18th year of what was regarded as scurvy. The most trivial blow or fall produced a bruise, and he suffered pain and stiffness of the joints. II. 3, bled to death from a tooth in his 9th year. II. 4, bled to death from a tooth in his 7th year. II. 5, Rave's sisters, not affected or only in very slight degree. (See Bibl. No. 14.)

Fig. 511. *Hertzka's Case.* A case was described by Hertzka in 1880. I. 1, phthisical. I. 2, healthy. II. 1, died at the age of 7 months, not affected. II. 2, aged 9. At the age of 11 months bruises appeared over the whole body. Epistaxis occurred at the end of the first year. These symptoms continued till 7, when the knee joint became inflamed. Later, internal bleeding and haematemesis occurred. The ecchymoses now became infrequent, but a cerebral haemorrhage resulted from a fall. A year later, the left knee became distended with blood. II. 4, died at the age of $2\frac{1}{2}$ of phthisis. II. 5, aged 7 months, was unaffected. (See Bibl. Nos. 411 and 412.)

Fig. 512. *Simon's Case I.* The following somewhat doubtful case occurred in a French family residing in France and America. Some of the cases are probably not haemophilia, but the account is not precise enough for certain diagnosis. This family is the same as that described by Delmas (see Bibl. No. 298 and Pedigree No. 474). Simon refers to Delmas' paper as follows: "One of these latter cases has been the subject of a communication by M. Delmas....His account was necessarily incomplete, the patient being only on his way through Bordeaux. We will present the complete history...." The points of difference observable between the accounts of Delmas and Simon, some of them of a serious nature, are interesting in that they demonstrate the danger of including any but well described families in a collection of material destined to be the basis for mathematical calculations. II. 1, M. S., was alleged to be haemophilic. He emigrated to America at the age of 18, and married there. He died of epilepsy at the age of 70. His brother, A. S., II. 3, also married in America, and died of old age at 94. III. 1—3, died young of "tumeurs blanches." III. 4—7, were healthy. III. 8, alleged to be haemophilic, died of epistaxis after a shrub had fallen on his head at the age of 14. III. 9, had gout. III. 10, was haemophilic after the age of 11, he died at 48 of epilepsy. III. 12—19, consisted of three males and four females: some were rheumatic, others healthy. IV. 12, and 13, were both haemophilic. V. 1. Referring to III. 1—7, Simon says "Ses petits-fils le sont" bleeders. "Pas d'autres détails." V. 2, living, her twin sister born dead. V. 4, born dead. V. 5, miscarriage. V. 6, healthy. V. 7, 8, 9, triplets miscarried. V. 10, C. C., bled three days from a cut upper lip. At the age of $4\frac{1}{2}$ he had epistaxis lasting four weeks. Rheumatic pains in the knees with periarticular ecchymoses which were very painful. Bad bleeding while cutting his permanent teeth. Died of haemorrhage at the age of 10. His brother, A. C., V. 11, died at the age of 1, of intestinal haemorrhage, the result of whooping cough. V. 12, healthy. V. 13, T. C., came to France at the age of 3. On the boat he had his first haemophilic attack. This consisted of "crises" preceded by shivering. He was easily bruised and the bruises were extremely painful. Morphine injections produced no ill-effects. A small boil bled for fifteen days. He had attacks of arthritis without fever: one knee became anchylosed. At the age of 10 he had epileptiform convulsions and at the same time the L. knee became swollen and tender. Later he lost the tendency to arthritis, and the epilepsy was controlled by bromides. V. 14, healthy. V. 15, L. L. His first manifestation of haemophilia was at the age of 3. He fell down, tore the fraenum of the upper lip and bled for twelve days. He also slightly scraped his hand and bled for ten days. He had attacks of arthritis similar to V. 13. At the age of 7 he bled for three days from a cut thumb. (See Bibl. No. 350.)

Fig. 513. *Simon's Case II.* The second doubtful case is in a French family. II. 1, healthy. II. 2, attacks of "catalepsy" had been observed. II. 3, aged 45. In his youth he had epistaxis, ecchymoses and rheumatic pains. His first bad bleeding was at the age of 26 while serving in the cavalry. He bit his tongue and bled three days. He served in the Franco-German War without mishap and all tendency to haemorrhage ceased. III. 1, healthy. III. 2, G. D., aged $12\frac{1}{2}$. At the age of 6 months he slightly injured his thumb and bled for $2\frac{1}{2}$ days. Measles attacked him at the age of $2\frac{1}{2}$. Three months later ecchymosis started, and a small abscess at the angle of the jaw had to be opened; it bled for ten minutes. At 3 he bit his tongue and there was "violent haemorrhage" and when he cut a tooth the bleeding was "terrible." Death was imminent when the haemorrhage stopped. At the age of 5 he visited London, and sustained great pain and swelling of the hand after

it had been shaken by an Englishman. The soot of the London atmosphere produced violent asthma, and he was forced to return to Paris. At 6 he had an haemorrhage from the bowel. Joint pains and epistaxis. (See Bibl. No. 350.)

Fig. 514. *Wijmans' Case I.* A haemophilic family in Holland. I. 1, and I. 2, nothing known with certainty. II. 1, and II. 3, said to have been bleeders. II. 5, E. J. L., died at the age of 62. She was often venesected: not a bleeder. II. 6, J. B., husband of II. 5, healthy, no haemophilia in his family. III. 1, and III. 2, two male bleeders, died of haemophilia. III. 3, five children. III. 4, said to have been a bleeder. III. 6, died of pulmonary disease. III. 7, a bleeder. He showed ecchymoses, haematuria, melaena, haemorrhage in knee joint, and died suddenly in his 7th year. III. 9, healthy, but plethoric, aged 41. III. 10, H. K., husband of III. 9, strong healthy man, no haemophilia in his family. III. 11—13, three girls, suffered from pulmonary disease. IV. 1—5, family of III. 9, and III. 10. IV. 3, died suddenly when 3 years old, not a bleeder. IV. 4, a bleeder. IV. 5, Wijmans' patient, 11 months old. He looked healthy and had cut several teeth. In several places, especially on his legs, fluctuating swellings and ecchymoses. At the age of 8 months he bled eight days from the mouth as the result of a fall. When seen by Wijmans, he had had a fall on his chin and had bled severely from the tongue for a day and a half. The blood was clear and watery and trickled away slowly.

Fig. 515. *Wijmans' Case II.* I. 1, and I. 2, not haemophilic, nor was the disease known in their ascendants. II. 1, died of *post partum* haemorrhage, but she was not haemophilic. II. 10, a male bleeder, bled to death when $1\frac{1}{2}$ years old. II. 11, a bleeder. Suffered especially from epistaxis. At 9 years of age he fell on his head and suddenly died shortly afterwards (internal haemorrhage?). III. 1, and III. 2, two bleeders, both dead. III. 4, a bleeder, died of the disease. III. 5, a bleeder died of angina diphtheritica. III. 6, Wijmans' patient, first showed the symptoms of haemophilia when 14 days old. Was 7 years of age when seen by Wijmans. (See Bibl. No. 258.)

Fig. 516. *Otto's Case.* John C. Otto, a physician of Philadelphia, was the first to give an intelligent account of haemophilia in 1803. In a short communication in the *Medical Repository of New York* (1803, Vol. VI. p. 1), he described in general terms the family of a woman named Smith—with certain descendants by name Shepard. This is the family frequently referred to in the literature as the "Smith-Shepard" family, of Plymouth, New Hampshire. At the end of his paper, Otto also records that Dr Rush had informed him that he had twice been consulted upon this disease, once by a family in York, and secondly by one in Northampton county, and Dr Rush also favoured Otto with an account which he (Rush) had received some years before from Mr Boardley of a family in Maryland. This account is given by Otto in inverted commas, being evidently the communication sent to Rush by Boardley. The history of this Boardley-Rush-Otto Case (see below) is both interesting and amusing from a medico-historical standpoint. As the publication of Otto is somewhat inaccessible and as it has very frequently been misquoted we give it in some detail following the exact original. The alleged relationship of the Smith-Shepard and the Appleton-Swain family has arisen apparently from Hay's remark that "a person by the name of Appleton married a Smith of Haverhill, supposed to be the Mrs Smith mentioned by Dr Otto." (See Bibl. No. 25.) Otto's account of the Smith-Shepard family is as follows: "About seventy or eighty years ago a woman of the name of Smith settled in the vicinity of Plymouth, New Hampshire, and transmitted the following idiosyncrasy to her descendants. 'It is one' she observed 'to which the family is unfortunately subject and has been the source not only of great solicitude, but frequently the cause of death.' If the least scratch is made on the skin of some of them, as mortal a haemorrhagy will eventually ensue as if the largest wound is inflicted. The divided parts in some instances have had the appearance of uniting and have shown a kind disposition to heal and in others cicatrization has almost been perfect, when, generally about a week after the injury, an haemorrhagy takes place from the whole surface of the wound and continues several days and is then succeeded by effusions of serous fluid. The strength and the spirits of the person become rapidly prostrate, the countenance assumes a pale and ghastly appearance the pulse loses its force and is increased in frequency and death from mere debility then soon closes the scene.....So assured are the members of this family of the terrible consequences of the least wound, that they will not suffer themselves to be bled on any consideration, having lost a relation by not being able to stop the discharge occasioned by this operation.....A few years since the sulphate of soda was accidently found to be completely curative of the haemorrhages I have described. An ordinary purging dose administered two or three days in succession generally stops them. The prescription being known to the whole family application is rarely made to a physician. The sulphate of soda has constantly succeeded when administered, but the prescription being in the possession of the Shepard family, the descendants of Smith and the cases that have been attended by physicians not being very numerous, it is impossible to ascertain the various states of the system in which it has been given, or to form any correct conclusions respecting its manner of acting. It is a surprising circumstance that the males only are subject to this strange affection and that all of them are not liable to it. Some persons who are curious suppose they can distinguish the bleeders (for this is the name given to them) even in infancy, but as yet the characteristic marks are not ascertained sufficiently definite. Although the females are exempt

they are still capable of transmitting it to their male children as is evidenced by its introduction and other instances, an account of which I have from the Hon. Judge Livemore, who was polite enough to communicate to me many particulars upon this subject. This fact is confirmed by Drs Rogers and Porter, gentlemen of character residing in the neighbourhood.....When the cases become more numerous it may perhaps be found that the female sex is not entirely exempt, but as far as my knowledge extends there has not been an instance of their being attacked.....The persons subject to this haemorrhagic disposition are remarkably healthy. No age is exempt nor does any one appear to be particularly liable to it. The situation of their residence is not favourable to scorbutic affections or disease in general. They live like the inhabitants of the country upon solid and nutritious food and when arrived at manhood are athletic, of florid complexion and extremely irascible." It would appear that Otto did not know any of the members of this family himself, although his description of the diathesis is very definite. It is to be regretted that no account exists of the two families known to Rush. The case referred to in the literature as Otto's, Coxe's, Boardley's and Rush's, is, as was said above, both interesting and amusing. It would appear that Mr Boardley or Bordley made a communication to Dr Rush on the sons of a certain "A. B." (Benjamin Binny?). Rush, in turn communicated it to Otto, in whose paper it was published. It would then appear that at the request of Otto, Mr Boardley who had "not been inquisitive enough for particulars" in the first instance, communicated with one John Coats. The latter after consulting Dr Allen and Dr Martin came in touch with "Mr Walter Binny" who resided on the farm formerly occupied by "B. Binny," and obtained the necessary information; the correspondence of all of these being furnished by Dr Otto to the editor of the *Philadelphia Medical Museum*, by name John Redman Coxe. In Otto's account of the Boardley-Rush case it says: "A. B. of Maryland has had six children, four of whom have died of a loss of blood, from the most trifling scratches or bruises. A small pebble [!] fell on the nail of the forefinger of the last of them when at play being a year or two old. In a short time the blood issued from the end of that finger until he bled to death. Two of the brothers living are going the same way. Their surviving sister shows not the least disposition to that threatening disorder although scratched or wounded. The father gave me this account two days since but I was not inquisitive enough for particulars." In the *Philadelphia Medical Museum*, however, the editor (Coxe) puts a different complexion upon this interesting history, as can be seen in the following correspondence of additional information which he procured through the late Mr Bordley.

EASTON, *May* 31, 1803.

Dear Sir, ...I was not myself acquainted with the history of the cases you mention but with a view of aiding your benevolent intentions I consulted Dr Allen the oldest practitioner in the neighbourhood, Dr Martin, and extended my inquiries to Choptauk Bridge, Dorset and Queen Anne's County but in vain, until our present court when I met Mr Walter Binny a juryman who is perhaps better acquainted with the circumstances than any other person in this country. Mr W. is nearly related to Mr Benjamin Binny who I presume gave you the information which was the subject of your letter. He now occupies the very farm upon which Benjamin resided. He knows nothing of the minutiae of the business but is certain of the fact that all the male children of B. Binny fell victims to fatal haemorrhages from the slightest causes as the puncture of a pin the cut of a knife. A daughter of his is now living who has occasionally experienced those little disasters we are all liable to and with the usual effects. Either three or four boys died from trifling injuries notwithstanding the assistance of the best informed gentlemen in medicine. Yours etc., JOHN COATS.

P.S. Since writing above I have had a second interview with Mr Binny: result you will find in enclosed letter. The only male who did not immediately die of loss of blood fell a victim to the consequence by dying of the dropsy in early life.

June 1, 1803.

Dear Sir, I have made inquiry respecting the loss of the three sons of Benj. Binny. The first (II. 1) that died with the loss of blood was occasioned by the kick of a colt over the eyebrow. The second (II. 2) was lost by a blister being raised on the fore finger from the fall of a brick (see "small pebble" above) from a negro child when they were at play. The third (II. 3) was cut over the eye by the swing of a gate as he was passiug through. Physicians attended each of them but to no purpose as all their skill could not stop the blood. A fourth (II. 4) was very subject to bleed at the nose though he did not die of the same. The females were not subject to the same complaint.

Yours, CHAS. W. BINNY.

From the above it seems that "A. B." of Otto's paper was merely a symbol for Benjamin Binny (I. 1) and that Mr Walter Binny (a juryman) is identical with Charles W. Binny. Such is the history of Otto's case! We give a pedigree founded on the data and leave it for what it is worth. For 100 years writers have quoted this case. How few can have looked into the published facts! (See Bibl. No. 17.)

Fig. 517. *Bramwell's Case.* In 1907, Byrom Bramwell published a chart of a family, prefaced by a few remarks. The man, IV. 3, aged 37, was admitted into the Royal Infirmary, Edinburgh, with epistaxis, and while he was in the hospital haematuria developed. When an infant he nearly died from the removal of a small tumour behind the ear. He had also bled from the gums after tooth extraction; on one occasion had had epistaxis and on another haematuria. I. 1, II. 1, II. 4, III. 3, III. 6, and V. 11, are stated to have been affected with haemophilia, I. 1; II. 1 (aged 30); II. 4 (aged 68); III. 6 (aged 40) having died of it. In the absence of further data, an independent opinion cannot be formed. (See Bibl. No. 828.)

Fig. 518. *Page's Case.* The history of a bleeder, IV. 5, in St Mary's Hospital, London. The family history was worked out by Mr Davis (the dresser), but it is stated that he did not see the various members of the family. Generation I. no data, except that I. 3, and I. 4, were not related. II. 3, said to be a bleeder "although in what precise way it was not known." II. 3, and II. 4, were not related. III. 1, 2, 5, 7, 8, 9, males, all dead, but it is not known whether they were bleeders. III. 10, a bleeder, alive, no data. III. 3, 6, 11, 12, females, alive, all bleeders, no data. III. 3, aged 60. IV. 2, 3, 4, stated to be bleeders, "if a history of severe epistaxis and tooth bleeding is sufficient to establish this fact." IV. 5, aged 22, a male, admitted into the hospital with extensive swelling of L. thigh extending from the knee to the groin and evidently consisting of extravasated blood. He had had epistaxis, and haemorrhage after tooth extraction. He had also bled furiously when an abscess was opened. His knee was swollen, probably haemarthrosis. He was seven weeks in the hospital. V. 1, and 2, healthy. (See Bibl. No. 518.)

Fig. 519. *J. H. C(ampani)'s Case.* The following history is shortly described in *Guy's Hospital Gazette* over the initials J. H. C. I. 1, N. I. 3, W. II. 1, Honor J. II. 3, Bessie J. "When young a bad bruise followed the slightest injury. This tendency has decreased with advancing age." II. 5, James N. II. 6, Mary W. II. 7, Annabel Q. III. 2, "who is slightly lame from some knee trouble which began in childhood." This family is said to resent inquiries. III. 5, Grace C. "When young had a dangerous tendency to haemorrhage. There was excessive *post partum* haemorrhage when she gave birth to her daughter." III. 9, Richard N., unaffected, alleged to have propagated the disease to his son. III. 10, not mentioned. III. 12 and 13, unaffected. III. 14, "a son, who in childhood bled profusely from slightest lesions. He does not appear to have had any joint trouble." Apparently dead. IV. 1, Georgina C., "bit her tongue when 10 years old. It was some weeks before the bleeding was entirely suspended." IV. 3, Bernard N., at 7 months he fell down and opened a small artery on the deep aspect of the upper lip. He bled for fourteen days. Epistaxis was at times severe, especially when he had whooping cough. "Almost incredible bruising" followed the slightest knock. He had had swellings of one elbow and both ankles. At the age of 3 he fell downstairs and hurt his right knee, and on the next day the joint was swollen, remaining so with slight exacerbations for one year when he was admitted to Guy's Hospital. The knee was then in a position of "triple displacement" with fluctuation. Formalin was injected into it and no bleeding followed the puncture. He was again in this hospital at the age of 5, for trouble in the same knee, which had again become flexed. The matter placed between inverted commas is the whole information supplied in each case. (See Bibl. No. 882.)

Fig. 520. *Young's Case.* Gen. I. Individuals not specifically mentioned but presumably normal. II. 2, 3, 6, presumably normal. II. 7, not a bleeder, but of a bleeder family. II. 8, died from haemorrhage after tooth extraction, aged 24. II. 9, at 16 months fell and cut the tip of his tongue and bled to death. III. 2, presumably healthy. III. 3, not a bleeder. III. 4, bled to death after slight injury to finger. III. 5, bled for several days after tooth extraction and died from subsequent enteritis, aged 17 years. IV. 1, Dr Young's patient, aged 13 months. Started bleeding from a minute point just above two recently cut incisors. Contusions and extravasations of blood all over his body. (See Bibl. No. 549.)

PLATE XLVI. Fig. 521 (see p. 314).

Fig. 522. *Finlayson's Case.* Finlayson published the history of a family living mostly in Lancashire. The information was said to be of unusual reliability. He was supplied with many names and other details which however he did not include in his publication. His chart is also printed in a manner so unusual that an element of ambiguity is introduced. The couple in the first generation had one son, II. 9, a bleeder, and three daughters who propagated the disease to four of their sons. One of these three daughters, II. 2, also had two daughters, III. 2, and III. 4, who transmitted haemophilia to four of their sons. No male, bleeder or unaffected member, propagated the condition. I. 1, and 2, unaffected. In the family of II. 1, there was no tendency to haemorrhage. II. 9, was the first bleeder known in the family. He died at the age of 30. III. 1, stated that his family was not affected. III. 10, was a bleeder; he died at the age of 26. III. 11, 12, and 14, were also bleeders. III. 17, unaffected children of some of the males in generation II. IV. 1, William W., aged 8. Born one month prematurely; feet first. At birth he had a large discoloured mark on his brow. While still a baby, his joints started to be affected. Swellings came up very quickly, sometimes with some discoloration of the skin. Trauma was at times recognised as the cause, but frequently they were inexplicable.

During the attacks the patient was a little feverish and fretful. No disability remained at first. At the age of 18 months ecchymoses first appeared. On at least four occasions the haemorrhage was severe. The toe nail was broken by a piece of slate and the blood flowed freely. The haemorrhage was arrested by a doctor, but it subsequently broke out again. On another occasion he bled for a fortnight from a ruptured fraenum of the upper lip. Again there was excessive haemorrhage from an injury to the brow. He had never exhibited any spontaneous external haemorrhages. In 1882, he was seen at the Glasgow Western Infirmary. Numerous arthritic attacks occurred while he was under observation, chiefly in the right knee and elbow. There was no elevation of the general temperature. Alterations in the joints were detected. He was lame. IV. 2, aged 6, a bleeder. IV. 3, was 20 years old. IV. 8, and 9, were stated to have been bleeders. IV. 11, a large family unaffected. (See Bibl. No. 445.)

Fig. 523. *Milner's Case.* Two haemophilic brothers with incomplete history of haemophilia in the family. II. 3, a maternal uncle, had the disease, dying from the effects of repeated haemorrhages and joint affections, the nature of which was diagnosed "rheumatism with complications." III. 1, "H." aged 14. First showed the haemophilic tendency at 5 or 6 months of age, after being circumcised. On this occasion he nearly bled to death. Milner saw him a few months later when he bled from a cut on his lip, an injury which he again sustained a few weeks later. On the second occasion be bled three weeks. Since 1896 his history had been a long succession of cuts, bruises and joint affections, a blow on the soft tissues producing a large haematoma. The gums bled every time a tooth became loose and epistaxis was frequent. His joints were first involved in 1903, the hip being the primary site affected. Later both the knees and the R. elbow became affected. Last winter he had severe haematuria. III. 2, "C.," presented symptoms similar to his brother, III. 1, and had haematuria twice. Milner describes a third case of great haemorrhage after opening an abdominal abscess in a boy aged 6, but he regarded this as an example of the "sporadic" type of the disease. (See Bibl. No. 898.)

Fig. 524. *Larrabee's Case.* We are unable to find adequate evidence of haemophilia in any of Larrabee's cases. The author himself attended the infant VII. 3, and there can be no doubt that neither this child nor her ascendants were haemophilic. With regard to the other side of the family, the evidence must surely have been supplied by someone little acquainted with the facts. The family resided in Boston, U.S.A. II. 1, David L., left England for America about 1757. There was a tradition that his brother, Kirvin, II. 3, bled to death. III. 2, Thaddeus L. III. 3, Lucy L. III. 4, Ephraim L. III. 5, James L., the individual alleged to have been related as shown in the chart. IV. 5, David H. L., died at the age of 62 of cerebral haemorrhage. IV. 7, Angeline L., died at the age of 35 after the removal of a tumour. IV. 9, died on the first day of life of haemorrhage from the nose and rectum. IV. 11, Louisa L., violent epistaxis throughout life. Died at 42, three days after a sudden haemorrhage from the uterus. IV. 13, was probably a daughter of III. 5. V. 1, a large family unaffected. V. 3, Nellie A. L., suffered from severe epistaxis a few weeks after birth. After the birth of her first child she "flowed for six weeks." Severe *post partum* haemorrhage after the second delivery. Recently a trivial scalp wound produced a haemorrhage of some severity. She is alive, aged 52. V. 4, Louisa L., severe epistaxis in first few weeks of life, afterwards unaffected. Some trouble with her knees in her last few years. Died of cancer at 45. V. 5, Henry L., died at the age of 21 days of haemorrhage from the umbilicus, nose, skin and ears. V. 6, Charles P. V. 9, L., a grandson or great grandson of III. 5. V. 11, A. L., a descendant of III. 5, his wife V. 12, living, a widow. VI. 2, Edwin L., had two severe haemorrhages as a boy from cuts, the scars of which were in the vicinity of arteries. Otherwise unaffected. VI. 3, aged 19, died of phthisis, with epistaxis and haemoptysis. VI. 4, died at 3 weeks of uncontrollable epistaxis. VI. 5, died at the age of 25. For three years before her death, she had a disease of the knees resembling tuberculosis. VI. 6, died at 18 of haemorrhage after tooth extraction. VI. 7, died at 7 of haemorrhage from a cut hand. VI. 10, L., a descendant of III. 5, alleged to have bled to death. VI. 12, unaffected. VI. 13, died in early childhood of haemorrhage from a trivial cut on lip. VI. 14, and 17, haemorrhage following slight cuts. Sometimes these healed promptly, at other times a painful dark coloured mass appeared. VII. 1, and 2, twins, miscarried at 6½ months. VII. 3, Dorothy, umbilical haemorrhage, purpura, bleeding from a scratch on the scalp, nose, rectum, finger nails, vagina and the unbroken skin, death on 5th day; no autopsy; no suspicion of syphilis. VII. 4, and 5, living and recognised as bleeders. They bled profusely from the slightest scratch. VII. 7, and 8, "two sons who are bleeders." VII. 10, died a few hours after birth with purpura and a large black swelling on the scalp. (See Bibl. No. 811.)

Fig. 525. *Coste's Case.* In Coste's thesis two cases are described, the one previously published by Gayet (see Bibl. No. 626), the other an original one, of somewhat doubtful character. I. 1, male, was affected from his earliest years with rebellious epistaxis which however diminished as he grew older. He died at the age of 52 after a slight fall, although the actual circumstances could not be ascertained. I. 2, healthy. II. 1, bled repeatedly from the nose. II. 2, Coste's patient in the Hôpital des Enfants-Malades under Lannelongue. He was normal up to the age of 10, except that he suffered from epistaxis. In June 1899 he was admitted into the hospital with a large swelling occupying the postero-internal surface of the left thigh. It was considered to be an abscess and was punctured, but it proved to be a haematoma. After

it healed he was discharged, but was readmitted five months later in a profoundly anaemic condition and covered with ecchymoses. His left thigh was also enormously swollen from a black and blue diffuse mass said to be the result of an injury. Recovery took place but he continued to have ecchymoses. (See Bibl. No. 715.)

Fig. 526. *Löns' Case I.* Male bleeder, aged 12. His mother, II. 3, said to have repeatedly suffered from epistaxis and bleeding from the gums. This was also the case with his grandfather, but whether paternal or maternal is not stated. III. 1, residing at Wernigerode, now aged 12. He had suffered all his life from severe bleedings following trivial injuries. He had bled for days after tooth extraction and had also suffered much from epistaxis. A haematoma covering the whole head resulted from being pushed against a wall. Two small incisions were made into it, but the haemorrhage was so severe that he had to be taken to the surgical clinic at Halle, where he remained for seven weeks. Two sisters, III. 2, III. 3, were not affected.

Fig. 527. *Löns' Case II.* Haemophilia in two generations. I. 1—4, no data. II. 2, and II. 3, normal. II. 4, markedly haemophilic, no data. III. 1, male, aged 22, residing at Zwickau. From his earliest years he was a bleeder. Went to work at the age of 14, but suffered severely with swellings of his knees and groins; admitted into hospital with diffuse swelling over R. trochanter.

Fig. 528. *Löns' Case III.* Four male bleeders in a family, living in Halle, and belonging to a bleeder family in Sangerhausen. I. 1—I. 4, no data. II. 1, and II. 2, not affected. II. 4, said to have been a bleeder and to have bled after tooth extraction. His brother, II. 6, was also a bleeder, and died of haemorrhage after an operation. III. 2, healthy. III. 3, female, said to have frequently suffered from haemorrhage—no data. IV. 1, bled to death from an incision made into a lump on the head. IV. 2, aged 3, bled for prolonged periods in his 2nd and 3rd years. He fell and cut his forehead. Bleeding at first was not severe, but later it became so serious that he was conveyed to the clinic in Halle, where a wound 2½ cm. long was found with infiltrated edges and partly scabbed over. Several large haematomata up to 4 cm. in diameter were also present over his R. temple. IV. 3, 4, 5, not affected.

Fig. 529. *Löns' Case IV.* I. 2, female, said to be a bleeder—no data. II. 1, male, bleeder. Suffered serious haemorrhages from slight injuries. A swelling of inflammatory nature appeared on his L. thumb. It was incised but the haemorrhage was so great that he had to be conveyed to the clinic in Halle. The wound was found to be dirty and was opened up. This was followed by severe haemorrhage, and epistaxis set in. II. 2, also said to have been a bleeder; no data. The evidence of haemophilia in the four cases seems to us quite insufficient. (See Bibl. No. 631.)

Fig. 530. *Dequevauviller's Case I.* (Obs. 7.) In his thesis for the degree of doctor (1844) Dequevauviller records nine cases of which only the last three are possibly to be regarded as haemophilia, viz. his observations 7, 8, and 9. A pedigree for obs. 7 only, has been constructed. Severe bleeding after the application of leeches to the perineum. The family history is short and incomplete, all that is reported being that the parents of the patient had four children, two of whom were dark like their mother, and showed the haemophilic tendency, two were blonde like the father and were not affected. Whether the patient, II. 1, is included in the four is not apparent from the description. II. 1, M. L., aged 22, a patient under Ricord in the Hôpital du Midi for phimosis. He had been subject to epistaxis of great severity on several occasions and had bled copiously after tooth extraction. The bleeding following leech bites was so serious as to necessitate the use of the *ferrum candens*. On three occasions he had been venesected and each time difficulty was encountered before the bleeding could be stopped. The slightest injuries produced haemorrhages and ecchymoses. After seeing the patient, Ricord ordered that 25 leeches should be applied to his perineum. On account of his past haemophilic history the patient demurred but his objections did not weigh with M. Ricord and the leeches were applied. The punctures bled all day until the whole of his genitals were enveloped in an enormous clot, from beneath which blood trickled. Various remedies, heroic and otherwise, were tried before the bleeding was arrested.

Dequevauviller's Case II. (Obs. 8.) A boy of 14 fell, and was attacked with severe epistaxis lasting eight days. After two years, another attack. The knowledge of heredity in this case was supplied to Dequevauviller by Dr Herrgott, who asserted that the boy came of a large family, the male part of which were affected with the disease. The parents and the daughters were free. No other details.

Dequevauviller's Case III. (Obs. 9.) Dr Herrgott also recounted to Dequevauviller the history of one of his college friends born at Türkheim in Upper Rhine. He had always shown the haemorrhagic tendency, the slightest injuries producing severe ecchymoses. Epistaxis was frequent, and at the age of 14 his left knee became involved. Twelve leeches were applied, but it was five days before the bleeding from their bites could be stopped. "All the boys of the family except the youngest are subject to frequent epistaxis and to rebellious haemorrhages from slight injuries." It was noted that whenever the above-mentioned patient took wine, a haemorrhage shortly followed. Dequevauviller's six other observations concern fairly severe bleedings either after operations (septic?) or in the course of typhoid. In any case they have nothing to do with haemophilia. (See Bibl. Nos. 142 and 143.)

Fig. 531. *Parkes Weber's Case.* The history of a German waiter, Hermann Z., III. 8, aged 23. Short account with pedigree of other members including females alleged to have been affected. I. 1, and I. 2, named Seidner, no information. II. 2, born 1848, died 1903, she "suffered from severe bleeding at her

confinements, and would not let a tooth be drawn for fear of haemorrhage." She was figured by Parkes Weber as a bleeder; in our opinion on insufficient grounds. II. 3, sister of II. 2, could not have teeth extracted for fear of haemorrhage and had severe bleeding after every confinement; figured as a bleeder by Parkes Weber. III. 1, male, aged 40, "had severe and persistent bleeding when he had a tooth drawn." III. 2, born 1872, died 1902, not a bleeder. III. 3, female, born 1875, figured as a bleeder; no definite data, although this is probably the sister who suffered bleeding at two confinements and dared not allow a tooth to be extracted for fear of haemorrhage. III. 4, female, born 1878, died 1879, not affected. III. 5, female, born 1880, not affected. III. 6, born 1883, died 1893, "apparently as a result of weakness following repeated attacks of epistaxis." III. 7, male, born 1885, not affected. III. 8, had severe haemorrhage from a bite of the tongue when 3 or 4 years old, and from a wound on forehead at 5. He also had epistaxis and bleeding from the gums. Some evidence of haematuria on one occasion. When 10 he had a swelling of his R. knee, and heard his mother tell the doctor that he was a bleeder. During the last two years he had suffered from spontaneous haematomata, due to slight traumatism, in both extremities and in both inguinal regions, and he had also had haemophilic swellings, in both knee joints and in left shoulder joint. In 1907, grave haemorrhage after tooth extraction, and in 1908 a large haematoma of left thigh. Blood examination on Nov. 13, 1908, showed lymphocytes 24·2 %, intermediates 4·8 %, large hyalines, 9·8 %, neutrophiles, 58·2 %, eosinophiles, 2·4 %, mast cells, ·6 %. Coagulation time variable III. 10, still born. III. 11, "tended to bleed excessively, from the gums." (See Bibl. No. 906.)

Fig. 532. *J. N. Hughes' Case.* Hughes records the case of Mr W., IV. 1, aged 37 years, who for the last four years had been in a crippled state, the result of chronic rheumatism. His parents stated that from infancy he had been subject to pain, swelling and stiffness of the joints. He was also in a remarkable degree the subject of haemorrhages not only from a single organ but from all. He had had at different times attacks of haematemesis, haemoptysis, haematuria, epistaxis, and haemorrhages from the gums and anus. Considerable extravasations of blood had at one time or another occurred in almost every part of his body without any appreciable cause whatever. Mr W.'s grandfather was subject to rheumatism from childhood, and was always known to bleed freely from any wound, however small. He also had frequent ecchymoses, epistaxis, haematuria, and finally died of haematemesis caused by a fall from a fence at the age of 45. His only brother was remarkably predisposed to haemorrhage and rheumatism, and died of the former. His sisters (six in number) were robust and healthy women, most of them having lived to an advanced age. The mother of Mr W. was quite a healthy woman and free from rheumatic or haemorrhagic affection. Mr W.'s sisters were all healthy. His brothers (eight) had all died, none of them arriving at the age of 20 years. Most of them were subject to haemorrhage and rheumatism. So notorious was the predisposition in the male members of this family to haemorrhage (the father excepted) that slight wounds had always been looked upon as dangerous in whatever part received. Three out of the eight brothers deceased died of haemorrhage, one from a cut on the tongue, another from a wound in the cheek, and the third of haematemesis. The nephews of Mr W. were likewise much disposed to haemorrhage and its concomitant rheumatism. There were among them several cripples, and some had died from loss of blood. On the other hand, the nieces were healthy and not at all inclined to either affection. In short, so far as the history of this truly unfortunate family could be traced, the males had invariably been found to inherit a strong predisposition to the disease in question, while not a solitary case had been known to occur in any female member of it. The hereditary character of the haemorrhage prevalent in the family of Mr W., and which obtained amongst his ancestors in the maternal line, will scarcely be doubted by any when it is considered that the predisposition thereto had developed itself satisfactorily in at least three successive generations. (See Bibl. No. 65.) In a second paper Hughes gives without any particulars the history of another family the facts being "well authenticated" and obtained from "several intelligent and highly respectable members of the family." "1st, that spitting, vomiting and purging of blood, bloody urine, bleeding at the nose, extravasations of blood among the muscles and integuments of the body generally, especially of the extremities producing dark discolorations and swellings attended frequently, after a few days' continuance, with pain and stiffness and obstinate haemorrhage from very inconsiderable incisions on whatever part of the body they are made, have been exceedingly common among the male members of the connection. 2nd, that the haemorrhage whenever it has manifested itself has been invariably attended with rheumatism to a greater or less extent. 3rd, that the slightest sprains or contusions have generally been followed by rheumatism of the part. 4th, that the majority of the males who have arrived at old age have been disabled by rheumatism. 5th, that on the approach of old age the tendency to haemorrhage has been less manifest. 6th, that a considerable number of the males have died in infancy and childhood. 7th, that deaths immediately from the loss of blood have been frequent, several resulting from the employment of the lancet, some from accidental wounds, others from various internal haemorrhages and two of the members simply from the application of blisters—'the blisters' in the language of my informant drawing 'blood instead of water.' 8th, that of the two diseases, haemorrhage and rheumatism, the former has always maintained the priority. 9th, that the females, though in no instance sufferers from the predisposition,

have nevertheless transmitted it to their offspring. And 10th, that the predisposition in question can be satisfactorily traced as far back as the fourth or fifth generation." It is to be regretted that this accurate summary is not illustrated by an account of the individual members of the family. (See Bibl. No. 71.)

Fig. 533. *Oliver's Case I.* History of a male, V. 10, aged 18, bleeding after tooth extraction. A pedigree is appended with very scanty information regarding other members of the family. Generations I. and II. no information. III. 2, wife of III. 4, who was not a bleeder. III. 3, said to have "bled to death on the road to Waterloo as the result of an accident." IV. 2, female, healthy. IV. 3, bled to death, at the age of 10 months, from lancing of the gum; no other information. IV. 4, died of phthisis. IV. 5, IV. 7, not bleeders. IV. 10, "bled to death, injury"; no particulars. IV. 11, not a bleeder. V. 5, "bled to death, injury." V. 6, "bled to death, aged 31 years, seven days after extraction of a tooth." V. 7, "bled to death, aged 27 years, had haematuria and purpura." V. 9, not a bleeder. V. 10, Dr Oliver's patient—a male, aged 18, iron founder. He had not suffered from haemorrhage in infancy. Ten years before, he was cut on the L. thumb and bled for a fortnight. With this exception he had never had any severe haemorrhage till he came to Newcastle-on-Tyne Infirmary suffering from toothache. As the haemorrhage continued six hours after extraction of the tooth he had to be admitted as an in-patient. The bleeding continued for 48 hours and recurred at intervals even afterwards. He had never had swollen joints. V. 11, aged 17, normal. VI. 2, a male, aged $4\frac{1}{2}$, "a bleeder," no details. VI. 3, female, aged $2\frac{1}{2}$, "suffers from profuse epistaxis."

Fig. 536. *Oliver's Case II.* I. 1—I. 4, not specifically referred to. II. 2, and II. 3, not bleeders. II. 4, a male, "used to bleed a great deal from the nose and ultimately died after a vomiting of blood," no other details. III. 1, male, aged 33, a groom, patient of Dr Oliver. When quite a child a leech was applied to his side and the bleeding which ensued could scarcely be checked. At the age of 9 a cut on the outer side of the R. eye bled for weeks. Very severe haemorrhage which required plugging came from the socket of a tooth which had been extracted when he was 14. At 16 he bled for a month from a cut between the thumb and R. index finger. At 19 he was an out-patient at the Newcastle-on-Tyne Infirmary. He frequently had epistaxis which was difficult to arrest. At 20 and at 30 years he had haematuria lasting a week. His joints, especially knees and elbows, had frequently swelled. In May 1886 he had pain in his side and was treated by some medicine which made his gums sore. Bleeding started from the mouth and from the kidneys and he was treated in the hospital. Some weeks after discharge he was re-admitted with swelling of the knee joints and haemorrhage from the gums. Six or seven weeks after his second discharge from the hospital he was thrown violently against a door while attempting to separate the combatants in a public house brawl. He was taken to the hospital, but the house surgeon, knowing nothing of his antecedents, did not think it necessary to detain him. He died, however, on the following day. The autopsy revealed a bulging, over the three upper costal cartilages, which on reflecting the skin proved to be an extravasation of blood in the right pectoral muscle. A large basinful of blood also flowed from his chest. The right pleural cavity contained about two pints of dark coloured fluid and several clots as large as a closed fist. III. 2, sister of III. 1, is "said to have a tendency to bleed," no details. IV. 1, son of III. 2, had "frequent spontaneous haemorrhages which are with great difficulty controlled." (See Bibl. No. 507.)

Fig. 534. *Fournier's Case.* This doubtful case was published in 1851. It refers chiefly to F. M. of Faujeux, II. 4. He was healthy till the age of 2, when blue spots began to appear on his legs accompanied by malaise. At the age of 4 these became more frequent. Epistaxis occurred and the haemorrhage from wounds was somewhat difficult to arrest. A blackish tumour of malignant aspect was removed from his scalp. The small wound was converted into a large ulcer by styptics and bled for three weeks. Fournier first saw him at the age of 9, when he had numerous ecchymoses due to trauma. After leeching, the blood could not be stopped for six days. At 11, he developed a lesion of the knee joint which was left distended with fluid. "At 11, the epistaxis and cutaneous swellings might be said to have disappeared," together with all tendency to haemorrhage. The knee, however, still contained fluid. His parents, I. 1, and 2, were healthy, though in the case of I. 2, the slightest cut produced haemorrhage somewhat difficult to stop. II. 1, died, at the age of 10, of intestinal haemorrhage, "symptomatic of purpura haemorrhagica." The two sisters were unaffected, II. 2, being twenty-two years old, and II. 3, eighteen. (See Bibl. No. 192.)

Fig. 535. *Liston and Kendrick's Case.* History of a family living in Norfolk. III. 1, was described by Kendrick, the other individuals being described by Liston. The evidence of haemophilia is not convincing. I. 1, bled to death from mouth and nose. II. 2, not affected. III. 1, Mr P., aged 23, member of a numerous family in Norfolk. He had an upper molar removed and bled to an extreme degree of anaemia for nine days. At the age of 17 or 18 he had bled for 14 days from a similar cause. III. 2, S. P., a farmer, aged 32—the source of Liston's information. He bled profusely from a small wart which had been cut from his hip when he was 19. Some weeks before, he fell and hurt his back and loins. Liston ordered leeches but the haemorrhage was so great that syncope appeared. III. 3—7, four brothers who bled much after tooth extraction. III. 8—12, all free from haemorrhagic diathesis, although three of them were very unhealthy. (See Bibl. Nos. 63 and 107.)

Fig. 537. *Hernandez's Case.* I. 1, 2, 3, 4, no information. II. 2, died of some inflammatory process in the chest. II. 3, alive and well. III. 1, was subject to frequent attacks of epistaxis, often copious. III. 2, was asphyxiated by charcoal. III. 3, Carré, now 27 years old. At age of 3 as the result of a fall, left knee became inflamed. At 6, fell and cut his tongue in several places; great haemorrhage. At 8, a wound of scalp bled profusely. At 20, violent haemorrhage after tooth extraction. Later a swelling of thumb was cut into and bled for a long time. Two years later was in the Hôpital de la Pitié under Verneuil for a swelling over lateral half of abdomen. Was discharged, but returned bleeding from a carious tooth. In addition to the foregoing two other cases are described as follows:—Obs. II. Taken from Grenaudier's thesis (see Bibl. No. 447). Case of leukaemia, with repeated epistaxis, profound cachexia, death in eight months. The patient, a soldier, Goiret by name, never had haemorrhage till he was 21. Obs. III. Case of epistaxis due to tumour of nasal fossae. Removal of tumour by M. Pean; death from haemorrhage. In his conclusions Hernandez tries to make out that haemophilia is not an entity and that it differs in no respect from other haemorrhages—a conclusion which is obviously incorrect. (See Bibl. No. 475.)

Fig. 538. *Gould's Case.* All that is known of this family is the pedigree exhibited by Gould at a Medical Society. In this pedigree, II. 1, III. 1, III. 6, III. 11, III. 16, III. 23, III. 24, IV. 4, are marked as bleeders without any details. IV. 1, a bleeder, was stated to be rheumatic. IV. 5, bled to death. IV. 10, and 11, were twin bleeders. Gould remarks that they were nearly all more or less subject to rheumatism, and that no females were affected. I. 1, Mr Yeaton, and IV. 2, and 3, stated to be unaffected. (See Bibl. No. 233.)

Plate XLVII. Fig. 539. *Newcombe's Case.* History of a haemophilic family in the North of England (probably Northumberland). I. 1, died young, no history. I. 2, was burned to death, no other details. I. 3, died at the age of 85. I. 4, died young of tuberculosis of the lungs. None of these were known to have any taint of the disease. II. 2, died in a fit at the age of 50. II. 3, alive, aged 86. Had "a tendency to bleedings in her younger days, and even now her skin bruises easily." III. 1, dead, no information. III. 2, alive, aged 50, moderately healthy but of a very neurotic temperament, had no symptoms of the diathesis, but after her confinements she alleged that she had always had a most severe loss, so much so, indeed, as more than once to endanger her life. III. 3, was troubled all his life with the disease, haemorrhage and swellings of the joints, and bled to death from the bowels at the age of 17. III. 4, had hip joint disease. III. 5, no information. III. 6, husband of III. 5, died of phthisis. III. 7, no information. IV. 1, John T., a bleeder, aged 32, patient of Newcombe, had suffered all his life. The slightest scratch would bleed from a day to three or four weeks. Bleedings from nose, gums, kidneys, bowels. Swelling of joints frequent, and induced by the slightest accident, such as a sprain or a blow. At first the joint swelled and was extremely painful for three or four days. Later, absorption of the exudate in the joint would take place. In many of the joints after frequent attacks swellings remain, causing some deformity and stiffness. Latterly a hard exquisitely painful swelling of the arm appeared just over the axillary artery, and caused by the sleeve of his coat being too tight. His whole life has been one of misery and danger, and had not the mother nursed him with the greatest care and assiduity he would never have reached manhood. In appearance he was anaemic, spare and of light complexion, very nervous and excitable, and except for haemophilia was healthy. IV. 2, Fred T., aged 29, a bleeder with a history identical with IV. 1, his brother. IV. 3, not a bleeder. IV. 4, has a most profuse catamenial flow. IV. 5, sex not stated, died of haemorrhage at the age of 4; no details. IV. 9, and IV. 10, stated to have been bleeders, sex and details not given. Newcombe adds that "the females of the family seem to have almost escaped the disease themselves but transmitted it to their offspring." (See Bibl. No. 377.)

Fig. 540. *Heath's Case.* Accompanying a short account of the two boys, IV. 2, and 4, is an extensive pedigree explained by a few notes. I. 1, James Argent, a bleeder, is reported to have had a bleeding father and uncles. II. 1—3, one living. II. 4, 6, 8, and 10, presumably married, as it is said that some of their sons, III. 1, were bleeders. II. 12, Matilda. II. 13, Chas. Pearce. III. 2, Charles, died at the age of 21 of haemorrhage from the bowels. III. 3, James. III. 4, William, aged 22, frequently bled. III. 5, Emma. III. 7, Matilda. III. 9, Alice. III. 10, F. Weedon. III. 11, Charlotte. III. 12, T. Barber. IV. 2, Charles, aged 12. He bled from the slightest scratch or blow. Came to University College Hospital with a double row of teeth; none of the milk teeth had been removed from fear of haemorrhage. These were all extracted. No haemorrhage, nor did he bleed again. [Eight years later however (Sept. 1876) he cut his leg. There was considerable destruction of skin and a tendency for the granulations to bleed. *Vide* Heath, in discussion on Jenner, Bibl. No. 362.] IV. 3, Francis, died at 3. IV. 4, James, aged 8. At the age of 3, he attended Westminster Hospital, for bleeding from a cut temple. The blood was easily arrested. Previously he had bled from the slightest scratch, but afterwards remained entirely free. IV. 5, Charlotte, died at 14 months. IV. 6, Alice, died at 5 months. IV. 7, Emma. IV. 8, aged 3 months. We fail to find any evidence of haemophilia in these cases. (See Bibl. No. 300.)

Fig. 541. *Masters' Case.* Dr J. L. Masters, of Indianapolis, has kindly communicated for this article the history of a bleeder family with which he has long been acquainted. It has not previously

been published. The family live in Indianapolis, U.S.A. I. 1., was a bleeder, although it is now impossible to get details of his history. II. 2, not affected. III. 2, male, now aged 50, a doctor of medicine. He began to show symptoms while still a child, the slightest injury being followed by haemorrhage which was so great as to induce syncope. At 12, he fell and knocked out two of his teeth. The haemorrhage was so great that he was considered to be dead. A doctor placed a cold looking-glass over his mouth, when it was found that he still breathed. At 22 he almost bled to death after the removal of a tooth. As a child, epistaxis was frequent and severe. Minute cuts on the fingers bled almost to the point of syncope. Dr Masters adds that in the case of II. 2, bleeding immediately after a wound was not so severe. Often some time would elapse, the wound being nearly healed when the haemorrhage burst forth. There was, in fact, a tendency to secondary rather than primary haemorrhage. For instance, a small cut on the scalp bled little at the time, but in a week became alarming. On another occasion he injured his big toe, and although there was no haemorrhage to speak of at first the blood spurted out about a week afterwards. He had been sitting on a chair resting the foot [presumably with his foot on the mantelpiece] when he felt something trickling down his face. It proved to be blood squirting with every heart-beat from underneath the toe nail. With advancing years the tendency to haemorrhage has diminished, although even now at the age of 50 spontaneous ecchymoses are frequently met with on the body. III. 4, brother of III. 2, a bleeder, but not in so severe a degree. IV. 1, and IV. 2, sons of III. 2, are bleeders. As, however, the greatest care has been taken of them the opportunities for haemorrhage have not been great. Epistaxis is, however, very frequent and difficult to arrest. II. 2, the mother of III. 2, is also stated to have been a bleeder in early life, but no data are given. She is alive, aged 78. At 72 she had a cerebral haemorrhage with hemiplegia. The eldest sister of III. 2 has a boy who is also a bleeder. IV. 6, the other children, five in number, being healthy. (See Bibl. No. 895.)

Fig. 542. *Rachford's Case.* History of a family living in Campbell County, Ky., six miles south of Newport. Not connected with any known bleeder family. II. 6, Charles Denner, a native of Bavaria, aged 56. He emigrated to America when a young man. Married II. 7, a native of Campbell County. She died in 1882 of peritonitis. I. 3, had never heard of haemophilia, and was quite sure that none of his immediate ancestors had it. II. 1—5, four brothers and one sister of Charles Denner. They were healthy and did not know of haemophilia in the family. II. 6, and II. 7, had nine children in the following order: III. 2, Lizzie Denner, not a bleeder; married III. 1 (of healthy stock), and had three boys, the youngest of whom was haemophilic. III. 3, Charles D., a bleeder. At 2½ years of age, bled to death from under surface of tongue in spite of medical skill. No actual wound could be found. III. 4, Henry D. III. 5, Kate D., not bleeders. Both died of "summer complaint," aged 15 and 12 months respectively. III. 6, Harry, not a bleeder, died of peritonitis, aged 19. III. 7, Martin, aged 16, always well and strong. III. 8, George D., a bleeder. At 7, bled five days from a small wound of the scalp. At 14, bled twenty-six hours from under surface of tongue, although no actual wound could be detected. Elbows swollen and discoloured. Ecchymoses. III. 9, Sallie, and III. 10, Willie, aged 12 and 10 respectively, not haemophilic. IV. 3, aged 2 years, had painful swellings of joints and blue spots. Twice had had dangerous haemorrhages from slight wounds of scalp. In a letter from Professor Rachford (May 1909) he adds that he is unacquainted with the subsequent history of this family. (See Bibl. No. 591.)

Fig. 543. *Van der Scheer's Case I.* I. 1, and I. 2, healthy. II. 1, G. v. d. W., healthy, as was also his wife, II. 2, J. H. She had three brothers and five sisters, all of whom were liable to epistaxis. One brother, II. 3, died of phthisis; one sister, II. 6, was drowned. III. 1, L. v. d. W., 35 years old, plethoric. Suffered from rheumatism and epistaxis. Menstruated profusely and during confinements had haemorrhages of varying degrees of intensity. III. 2, healthy. III. 3, healthy. III. 5, suffered from asthma. IV. 1, a bleeder. Epistaxis at the age of 6 months and frequently afterwards, many subcutaneous ecchymoses. Lost a tooth at the age of 2 and nearly bled to death. Six months later he wounded his tongue and bled to death 2½ days later, having lost a colossal amount of blood. Everything was tried, including the *ferrum candens*, but without avail. IV. 22, aged 11, had great haemorrhages, after trivial wounds, ecchymoses and haemorrhagic extravasations in the skin and joints. IV. 3, aged 7, a bleeder, had epistaxis, suggillations, and free haemorrhages from the skin. IV. 1, 2, 3, also showed a hypoplastic development of the penis. IV. 4, aged 5, and IV. 5, aged 3, both healthy. IV. 6, and 7, both scrofulous. Sex not stated. IV. 8, healthy.

Fig. 544. *Van der Scheer's Case II.* History of a man who bled to death into the spinal canal during coitus. I. 1, alcoholic. I. 2, affected several times with scorbutus. II. 1, and II. 2, both had profuse menstruation. II. 3, E. A., a bleeder. At 4 he sustained an enormous traumatic haemorrhage, and again at 7 after having slightly cut his finger. Bled for three weeks from a trivial knock on the nose with a small stone. At 10, his knee became swollen, and four leeches were applied, leading to almost fatal haemorrhage. He continued to suffer great losses of blood, after slight injuries, and rheumatic pains. He married a healthy woman, II. 4, when he was 28, and she bore him two daughters. During coitus when 30 years old he was suddenly seized with pain in his back, and died paraplegic and comatose next day. An autopsy revealed the whole spinal canal filled with blood. III. 1, girl, died of hydrocephalus during her 1st year. Van der Scheer also refers to a third case—a boy, B. J., who died of cholera in his 8th year. At 4 he had epistaxis, haematemesis, and subcutaneous ecchymosis, after an

injury to his head. His father was weakly and rheumatic, his mother healthy. Doubtful case. (See Bibl. No. 187.)

Fig. 545. *Hubbard's Case.* In Hubbard's original publication reference is made to a boy, aged 4, who bled profusely from a scratch on the forehead. Haemophilia was alleged in the grandmother, II. 3, who bled severely after tooth extraction, and in three maternal uncles. Dr Hubbard has supplied us with additional information from which our pedigree is constructed. I. 1—4, healthy. III. 1, a bleeder, had severe haemorrhages after trivial injuries. His joints, especially knees and ankles, were frequently affected. He had severe bleeding from an injury, to his hand, which necessitated amputation. Eight years later he died of internal haemorrhage. III. 2, was a bleeder, affected like III. 1, and bled to death. III. 3, nearly bled to death after tooth extraction, and ultimately died of haemorrhage. III. 4, 6, 8, three daughters of II. 2, and II. 3, were healthy. Two of them bore bleeders. IV. 1, bled to death. IV. 2, bled from the mouth and died. IV. 3, almost bled to death from a bruise on the hip. IV. 4—7, healthy. IV. 8, bled to death from a cut on the foot. The actual evidence of haemophilia apart from death by bleeding is meagre in this case. (See Bibl. No. 534.)

Fig. 546. *Lafargue-Laborie Case.* Case of a bleeder, IV. 5, seen by Lafargue, in Lisfranc's clinic. An indefinite history of other bleeders in the family was supplied by his mother, III. 2. I. 1—4, no information. II. 3, presumably normal. II. 4, an uncle of III. 2, whether paternal or maternal is not stated. III. 2, said that II. 4 died of haemorrhage, but she was unable to give any particulars. III. 2, alive, aged 75. III. 1, alive. III. 3, brother of III. 2, had haemorrhages into his joints, and died in the Charité at the age of 17 after the extraction of a tooth. IV. 1, fourteen children, not bleeders. They all died before attaining the age of 3. IV. 2, died from haemorrhage from the vulva, aged 6 weeks. IV. 3, had epistaxis and severe bleedings from trivial wounds. He died of haemorrhage from a wound of the head after the formation of an enormous haematoma. IV. 4, had haemorrhages from slight wounds and died from the effect of a cut on the calf at the age of 17. He was admitted into the Hôtel-Dieu, where it was found necessary to ligate the femoral artery. Result, secondary haemorrhage and death. IV. 5, Lafargue's patient, Laroche, a compositor, aged 41, in the Hôpital de la Pitié. Five days previous to admission he had struck his side against a key in a door. A large swelling made its appearance which was evidently a haematoma. As a child he had repeatedly suffered from epistaxis. At 25 spontaneous haemorrhages occurred from the gums and lasted for weeks. Formerly he was troubled with swellings of his joints. Several times leeches had been applied, but the closure of their bites necessitated the free use of the *ferrum candens* as the haemorrhage was so profuse. At the age of 34 he had haematuria. During his stay in the hospital it was noticed that he bruised on the slightest pressure. Laroche was also the subject of a communication by Laborie (Bibl. No. 94), but his account is not so complete in detail as that of Lafargue. (See Bibl. No. 89.)

Fig. 547. *M'Caw's Case.* I. 1, and I. 2, no information. II. 3, a bleeder, "bled to death from cut on lip." No other data. III. 3, affected with haemophilia, but "seemed to grow out of it." No particulars. IV. 1, aged 8, admitted into Queen St. Hospital for Sick Children, Belfast, with epistaxis, which had lasted eight days. III. 2, said he first developed symptoms of the disease at 15 months, when a large ecchymosis formed on his brow as the result of a fall. A doctor cut into it, and profuse haemorrhage followed. In his 2nd year he fell and nipped his tongue, and bled for eight days, the haemorrhage ultimately ceasing when he was exhausted. Five months later he fell and got haemarthrosis of knee, which, after recovery, recurred six months later. On one occasion he cut his index finger and bled for a week. Hurt his elbow, which swelled and blackened; then epistaxis lasting eight days. Plucked out a loose tooth, which bled for seven days. (See Bibl. No. 506.)

Fig. 548. *Acland's Case.* History of a boy, aged 7, who bled to death in St Thomas's Hospital, London. Some evidence of haemophilia in two brothers and in father's maternal grandfather. I. 1, male, is said to have been found insensible from loss of blood from epistaxis on more than one occasion. According to III. 3, he wore a blood stone as a charm. II. 1—5, healthy. III. 2, and 3, healthy. IV. 1, W. C., aged 7, injured his tongue with a piece of slate pencil, and bled to death on the ninth day. An autopsy failed to reveal any abnormality except some rounded cellular masses of indefinite nature in the thymus gland. IV. 2, suffered from epistaxis. IV. 3, had petechiae, bruises, and haemorrhages from the gums. On three occasions he had bled from the bowel. IV. 4—6, three girls, all healthy. (See Bibl. No. 487.)

Fig. 549. *Förster's Case I.* Two families are referred to in Förster's article. In the first there may be a slight example of haemophilia in III. 2, whose cousin, III. 3, of sex not stated, suffered from blue spots. II. 1, was healthy and well off. His wife was also healthy, though menstruation was profuse. III. 1, represents an abortion in the third month. III. 2, was a "breech" presentation, and five months later a bruise was found on his buttocks. Subsequently other bruises appeared more often on the extremities. At the age of 2 he fell down and bled much from a wound, hardly visible, of the gum; while at 4, after a similar accident, the left elbow became much swollen, bruised, and stiff. An injury, of the left upper eyelid at the age of 5, inflicted by an umbrella, also produced bruising and swelling. He was then 7, and complained of pains in the joints.

Fig. 550. *Förster's Case II.* The second pedigree presents a number of persons not mentioned in the text. The account seeks to show haemophilia occurring in cousins (sister's children), but we do not ourselves accept the diagnosis. The children were mostly wretched and "scrofulous," and their sufferings appear to us to be due probably to infective processes. II. 1, a family in which there had been instances of cousins marrying, but no tendency to haemorrhage. III. 3, very "scrofulous" and anaemic; also suffered from menorrhagia. III. 4, healthy. III. 5, recollected the fact that she used to have ecchymoses. III. 6, healthy. IV. 1, 2, 3, 4, and 6, unaffected, but anaemic and "scrofulous." IV. 5, at the age of 3 weeks, bleeding occurred from the navel. Later, ecchymoses appeared chiefly on the legs, and haemorrhage from the gums and lips. At the age of 3 bleeding occurred from a swelling on the under aspect of the tongue, which projected from the mouth. The haemorrhage was arrested, but death followed with convulsions. *Post mortem*, a haemorrhage was found in the brain. IV. 7, was born in 1866. He was healthy at birth, but when being bathed on the 4th day, blood was noticed issuing from the cord. This was stopped by proximal ligature, but later, after separation, haemorrhage started from the insertion. Jaundice was profound, and death followed on the 11th day of life. IV. 8, born two years later, also bled from the umbilicus, and the bleeding continued slightly for three weeks after the separation of the cord. Slight haemorrhage from the gums was produced by sucking the bottle. No bleeding took place after slight wounds. There was no tendency to rickets, but colds and cough were common. At 17 months ecchymoses first appeared, and at 18 months he had typhoid fever. Two and a quarter years later he began to complain of his right arm, and suddenly one night an enormous swelling of the right shoulder occurred. A few days later he had a diffuse attack of urticaria, and the shoulder was found to have suppurated. Death followed. In the case of IV. 9, born in 1869, there was no umbilical haemorrhage. During the third week some blood was found in the stools. At the age of 13 months "Grindkopf," with swelling of glands and catarrh. When 3½ years old he was very feeble—ecchymoses and petechiae, but no bleeding after slight wounds. At the time of writing he was 7 years old, and fairly healthy. IV. 10, born in 1872, discharged watery blood from the umbilicus. Fever set in, and death followed on the 21st day with diarrhoea. IV. 11, unaffected but unhealthy. IV. 12, was born in 1875, and after separation of the cord, haemorrhage started from the ulcerated umbilicus. At the age of 5 months, after a period of restlessness and thirst, a crop of purpuric spots appeared. Three months later, a little blood was found in the stools and urine and two drops came from the eyes; at this time there was a sudden painful swelling of the left elbow, with discoloration of the skin. On another occasion there was a slight haemorrhage from the lips, and a burn on the hand suppurated. The child was thin, pale, and slightly rickety. Death at 12 months from pneumonia. The *post mortem* revealed tuberculosis. (See Bibl. No. 388.)

Fig. 551. *Salomon's Case.* Salomon, of Hildesheim, has described a case which, with Canstatt, we consider as doubtful. Lange regarded it as haemophilia. I. 1, normal. I. 2, female, aged 62. In her youth she often had uncontrollable epistaxis. After the application by Salomon of two leeches to her chest the bleeding did not stop for eight days. II. 2, a female, big and strong. In her youth she had epistaxis. Years before she experienced difficulty in stopping the bleeding from small wounds. II. 3, normal. II. 4, R. von L., aged 25, a ship captain. Frequently had severe epistaxis. Salomon was called to see him in 1829, as he was bleeding from the gums near one of the lower molars. In spite of all treatment the haemorrhage continued with remissions for five weeks, and several times the patient was given up for dead. II. 5, male, had epistaxis. Salomon drew one of his teeth, but the bleeding necessitated the actual cautery. III. 1, large family, no data. (See Bibl. No. 85.)

Fig. 552. *Wilmot's Case.* History of a bleeder, III. 2, with short account of similar condition in two cousins. I. 1, and I. 2, no data. II. 1, aged 35, not a bleeder. II. 2, small active woman, not affected with haemophilia. Had not lost excessive quantities of blood during confinements. II. 3, sister of II. 2, no data. II. 4, husband of II. 3, healthy. III. 1, healthy boy. III. 2, a bleeder, seen at the age of 4 by Wilmot, as he had been bleeding from the palate behind the incisors for four days. Haemorrhage was with difficulty quelled. Two months later a swelling appeared over the right parietal bone. It suppurated and burst, and great haemorrhage occurred. Later he fell and divided the lingual artery, which had to be tied. For seven months after, he remained well, but then bled almost to death from the gums. Four months later he was seized with pain and swelling of the calf of the left leg, which became black and yellow. Still later, epistaxis and large swelling of thigh. III. 3, and 4, healthy. III. 5, bled to death from the gums when 4 months old. III. 6, bled to death from an incision into an ecchymosis produced by a fall. (See Bibl. No. 126.)

Fig. 553. *W. Jenner's Case.* I. 3, and 4, died of short illnesses. II. 2, died of "nervous fever." II. 4, normal. No miscarriages. Source of information. II. 5, and 6, healthy. III. 1, healthy. III. 2, profuse catamenia, but healthy. III. 3, Thomas V., aged 13, printer's boy. Mother noticed the tendency to bleeding at 6, when he had very severe haemorrhage after a cut on the palate. Permanent teeth appeared late: double row of incisors. One row extracted, followed by great bleeding. For the last seven years he was constantly losing blood from nose, mouth, and once from gums. Never free from ecchymoses. His wrists, knees, and ankles were frequently swollen, without trauma. Left knee permanently affected. Came to University College Hospital, London, with alleged bleeding from bowel.

While in hospital he passed clots *per rectum*. Died twenty-four hours after admission. *Post mortem*, large bruise down two-thirds of forearm. Ecchymoses, especially on the right thigh. Here there was a large haematoma, beneath the deep fascia, full of firm clot. Left knee joint full of purple blood. Cartilages reticulated. Lower 14 in. of large bowel full of blood clot, which at one place was adherent to the mucous membrane. Jenner considered it to be true haemophilia. III. 4, aged 10. At 3 he bled profusely from a cut on the palm. After scarlet fever he had haemorrhages from nose and mouth, which were often spontaneous. They usually came on when he was looking his best. III. 5, aged 7, healthy. (See Bibl. No. 362.)

Fig. 554. *Hope's Case.* Family living in Freemantle, Western Australia, and formerly in South Australia. I. 3, not a bleeder. I. 4, drowned; was not a bleeder. II. 3, source of information respecting II. 4, 5, 6, 7, whom she stated were bleeders. II. 4, John B., always a bleeder; nearly bled to death from the bite of a rat. During his convalescence he died, aged 12, as a result of a carriage accident. II. 5, William B., bled to death at or immediately after a lithotomy operation, aged 6. II. 6, alive, aged 24. Had epistaxis; nearly bled to death after tooth extraction. II. 7, aged 20, bled at nose. III. 1—4, healthy. III. 6, healthy so far—a child. III. 5, Albert C. Crewe, aged 6, had bled since he was a child. Last year cut his lip as result of a fall, and bled two days. Epistaxis severe. On July 13, 1888, received punctured wound of thigh just above knee. Great swelling of thigh. Knee left stiff. Attempts to straighten it led to a large blood tumour in popliteal space. Ultimate recovery. (See Bibl. No. 531.)

Fig. 555. *Maixner's Case.* Professor Maixner, of Prag, has sent us the short account of two brothers, bleeders, III. 4, and III. 6. Another brother, III. 7, bled to death from a trivial wound. A brother of the mother was also a typical bleeder. In Maixner's patients the haemorrhages occurred most frequently into the subcutaneous tissues and joints. It will be noted that a normal healthy brother was born between Maixner's two patients. Blood examination by Dr Hynek showed the coagulation time of the blood to be normal and that the clot separated itself very easily from the walls of the capillary into which it was drawn. (See Bibl. No. 894.)

Fig. 556. *Price's Case.* Two male bleeders in a family of four children. I. 1, 2, both died of phthisis, aged 30 and 37 respectively. I. 3, 4, not bleeders according to II. 3. II. 2 died of phthisis, aged 34. II. 3, said he often bled profusely from the nose. II. 2, and II. 3, not related. III. 1, died in six days; cause not stated. III. 2, had severe haemorrhage after circumcision when 2 days old. At 1 month a blood tumour formed on his wrist, and he was frequently bruised. When he began to walk, his ankles, elbows, and knees swelled. At 6 or 7 years had a bruise extending from ankle to thigh. Sometimes he was quite crippled with his swollen joints. Admitted into Westminster Hospital oozing from the mouth. Left ankle swollen and tender with a large ecchymosis on its outer side. Similar ecchymoses on left elbow, right ankle, and leg. Blood coagulated slowly. On recovery, he went to convalescent home, and there his knee swelled. Haematuria also developed. Aged 13. II. 3, circumcised when 2 days old, and bled freely. Haemorrhage from gums during teething. Frequently lost much blood from small wounds; joints had been swollen and discoloured. (See Bibl. No. 839.)

Fig. 557. *Payne's Case.* Haemophilia occurring in North Carolina and affecting three brothers and a maternal uncle. I. 1, 2, 3, 4, no information. II. 1, a blacksmith, strong and healthy, lived to a ripe old age; was gouty, and suffered from eczema (note by Dr Payne). II. 2, alive. II. 3, a bleeder. II. 4, other members of the family, healthy. III. 1, aged 18, frequently the subject of haemorrhages from trifling injuries. Haemorrhages especially prone to come on ten days to two weeks after the injury; bled profusely from wound on head caused by the spur of a chicken rooster; all his life he suffered from epistaxis. Was thrown from a waggon and had an enormous haematoma. A kick from a gun produced a lump as large as an hen's egg. Epistaxis, requiring plugging. While riding a horse he was thrown, striking the pit of his stomach across a rail fence; vomited blood, but was soon able to be at work again; about a fortnight later he passed a large quantity of blood *per rectum*, which continued. Everything was tried to stop it, but without success, and he died forty hours later. No autopsy. It is stated that in spite of terrible losses of blood he had great recuperative powers. III. 2, aged 17, also regarded as a bleeder like his brother, the haemorrhage occurring especially some days after the injury. He was also an epileptic. III. 3, a weak, nervous boy, aged 15, had troublesome haemorrhages from slight wounds, and always required styptics and plugging of the cavity after the extraction of a tooth. III. 4, not a bleeder. III. 5, healthy. In a personal communication Dr Payne informs us that ultimately all the three brothers died of haemorrhage following trivial injuries. (See Bibl. No. 508.)

Fig. 558. *Besserer's Case.* I. 1, and I. 2, normal. II. 1, a bleeder, bled to death from a small wound at the age of 9 months. II. 2, a bleeder, died at the age of 5 years apparently from meningitis or sepsis. Autopsy showed hypertrophy of left ventricle. II. 4, 5, 6, III. 1, all healthy. (See Bibl. No. 154.)

Fig. 559. *Cadet de Gassicourt's Case.* This is the case of a haemophilic boy, II. 2, aged 13½, who while lying in the Hôpital Sainte-Eugénie in 1876, was the subject of a discourse by Cadet de Gassicourt. His father, I. 1, who is himself described as haemophilic because of frequent attacks of epistaxis, said

of his son on one occasion of epistaxis, "You are like me!" The mother, I. 2, was unaffected. The boy himself from the age of 2 or 3 was subject to epistaxis, bled readily from the smallest wound and bruised with great facility. On one occasion while at the kindergarten at the age of 5 or 6, he had such a bad attack of epistaxis, that the parents were summoned. He remained pale and anaemic for twelve months after. On this occasion he was admitted to hospital for haematuria following on a strain of a moderate character. He was found to be covered with ecchymoses and had a blood blister the size of a pea on his gums. Further, in hospital, he had two attacks of epistaxis and blood was found in his stools. On examination, his heart and lungs were found to be normal. There was no fever and his appetite was unaffected. A sister, II. 3, also suffered from epistaxis, but another brother, II. 4, was unaffected. II. 5, other sisters. (See Bibl. No. 359.)

Fig. 560. *Waterhouse's Case.* Waterhouse of Pontypridd (Glamorgan) has described four cases of haemophilia occurring in two generations. I. 1, showed no haemorrhagic tendency. II. 2, not a bleeder, although she had several attacks of intractable menorrhagia. II. 2—II. 5, four females, scrofulous but not haemophilic. II. 6, not a bleeder, drowned at the age of 18. II. 7, II. 8, II. 9, three male bleeders, details given only of II. 9. II. 7, died, aged 8. II. 8, died, aged 6. II. 9, in Waterhouse's first account was described as aged 5, intelligent and quick; his trouble was said to be congenital and manifesting itself in oedema of the eyelids and obstinate haemorrhage from trivial causes. Ecchymoses were seldom absent. Elbows, wrists and ankles became swollen and painful. At 4 he fell against a bed post and loosened three teeth from which he bled for six days. On the onset of exhaustion, the haemorrhage ceased spontaneously and he rapidly recovered. At 5 he fell off a stove and bruised his head. A large swelling appeared extending from the lower jaw to the coronal suture. In Waterhouse's second account, he is said to have subsequently developed a swelling round his right ankle with purpuric spots in various parts. III. 1, brought to Waterhouse in 1870, having fallen against a chair. Considerable extravasation of blood had taken place into the cellular tissue of the forehead. Later, he fell and injured the mucous membrane of his lip. Profuse bleeding ensued and lasted 24 hours in spite of styptics. Purpuric spots also appeared. (See Bibl. Nos. 313 and 314.)

PLATE XLVIII. Fig. 561. *Nettleship's Case.* Mr Edward Nettleship has furnished us with data supplied to him by IV. 58, a medical man in whose family haemophilia occurred. The family is a well known one and has contained many distinguished representatives. On the male side it can be traced back continuously through sixteen generations. Our first generation is the eleventh in direct descent. I. 1, died in 1788. By his wife he had three sons and six daughters, one of whom, II. 16, who died of old age, was the maternal grandmother of the first bleeders. II. 2, was born in 1770 and died in 1846. II. 4, married her first cousin, II. 5. II. 17, died of cancer of the stomach at the age of 43. II. 18, and II. 19, both died of old age. III. 1, was born in 1802 and died in 1879. III. 7, died in advanced life in 1846. III. 16, born in 1813, died 1881. III. 15, 17, 18, 19, four wives of III. 16. III. 30, female, died in 1906 of cancer of the breast, aged 79. III. 31, fairly healthy female, died of pleurisy. III. 33, healthy, died in New Zealand of a cause unknown. III. 38, married abroad. III. 40, died young. III. 41, died many years ago: cause unknown. III. 43, died young. III. 45, healthy, married when old. III. 46, healthy female, unmarried. IV. 1—37, so far as is known these presented no signs of haemophilia. IV. 38, alive and well. IV. 39, healthy. IV. 41, healthy female, alive. IV. 43, healthy female, alive, unmarried. IV. 44, healthy male, alive. IV. 45—50, all healthy, living. IV. 49, 50, unmarried. IV. 51, healthy. IV. 53, bleeder. At an early date it was seen that he was not as other children. He was easily bruised and suffered from mysterious swellings of his joints which medical men diagnosed as rheumatism. If wounded he bled profusely. In spite of frequent joint attacks he was able to attend a boarding school. At 21, he nearly bled to death after tooth extraction. He was engaged in business and did well. The only active exercise he was able to take was rowing and he died of intramuscular haemorrhage while engaged in this sport. IV. 54, quite healthy. IV. 55—57, died in infancy. IV. 58, source of information; a healthy medical man, not married. IV. 59, a bleeder, "A.G." considered to have been the most severely affected of all. When a baby it was seen that he had the same defect as IV. 53, but to a much greater degree. The slightest injury was followed by a huge bruise. The gums would bleed without apparent cause. Swelling of the joints was frequent and he was not expected to live long. The slightest cut bled profusely. When a young man he nearly bled to death from a wound of the hand from a fish hook. Between the ages of 25 and 40 he had frequent bleedings from the rectum and into the muscles of the thigh. He was seldom without a bruise in some part of the body. He once had an haemorrhage into the substance of the lung and was frequently in a home with joint attacks. Once his shoulder joint suppurated and the pus was allowed to come by itself to the surface. When 30—35 years old he was able to go to Switzerland, but at any altitude over 5000 feet haemorrhage from the rectum started. The last few years of his life were spent at the sea side in a bath chair. He died of haemorrhage from the rectum at the age of 46. Bleeding into muscles or joints was always attended by severe pain and fever. Absorption of blood and recovery to health was usually rapid. IV. 60, healthy female, married 40 years. IV. 62, a bleeder of a milder type than IV. 59. At school he was able to play hockey, although at an early age he had joint attacks and

bled immoderately after slight injuries. At school he nearly died of haemorrhage from the bowels. He was given up as a hopeless case. This was however his only serious attack until the age of 16, when he died of bleeding into the pelvic muscles after a slight injury. The haematoma which formed, suppurated and burst into the rectum. IV. 63, died of phthisis. IV. 64, alive, unmarried. IV. 65, alive. IV. 67, died from an accident, details not stated. IV. 68, alive, unmarried. IV. 69, dead, cause of death not stated. IV. 70, died young, cause of death unknown. IV. 72, 73, alive. IV. 74, IV. 75, no information. V. 1—8, believed to be healthy. V. 9, alive. V. 10, born 1861, alive. V. 25—29, alive and well, unmarried. V. 30, and V. 31, healthy families. V. 33, very healthy, married five years. V. 34, a moderate bleeder but diagnosed as such at an early age. He is able to carry on his business effectively but has to take precautions against occasional immobility resulting from joint attacks. With increasing years the attacks are less frequent however. He is married. He was seen by one of us (W. B.) in March 1910. V. 36, is a bleeder of a type similar to V. 34. He has joint attacks and bleeds from various places, but is able to attend to his work with fair regularity. V. 38, healthy, married in 1906. V. 40, unmarried female, healthy. V. 41, a bleeder and was recognised as such at a very early age. He bled to death, internal haemorrhage and from the gums at the age of 12. V. 42—45, healthy, unmarried. V. 46, healthy, unmarried. V. 47—50, four healthy males, unmarried. VI. 1, born 1909. VI. 2, healthy boy. VI. 3, unaffected female. VI. 4, very healthy female, aged $2\frac{1}{8}$ years, born in India. We are indebted to Mr Nettleship and IV. 58 for permission to record this hitherto unpublished case. (See Bibl. No. 900.)

Fig. 562. *Zoege von Manteuffel's Case.* Description of III. 4, a Jew living in Dorpat. A pedigree with bleeders marked upon it is given but without any details. I. 1, Jossel Kahn. II. 1, David Rohloff. II. 2, Sarah Kahn. II. 3, Kosselowitz, married to II. 4, Jette Kahn. II. 5, Babes, married to II. 6, Mina Kahn. II. 7, several sons. III. 1, Joseph Rohloff, a bleeder, aged 27. III. 2, Bernhardt R., aged 23, a bleeder. He bled for weeks from a small wound of lip. III. 3, Samuel R., aged 12, twin with III. 4, Levi R., who was brought to Zoege von Manteuffel bleeding from the socket of the left lower molar tooth. Upon its removal two days before, violent bleeding had ensued almost immediately. At first it was controlled, but it started again and was very severe. A sample of blood was examined by Alexander Schmidt and he recommended the use of "zymoplasma" which in combination with cocaine ultimately caused the flow of blood to cease. He had bled seriously on one occasion after the removal of an incisor, and on another occasion for ten weeks from a wound on the head. III. 5, Abram R., a bleeder. III. 6, female, not a bleeder. III. 8, Rebecca Kosselowitz, marked in pedigree as a bleeder, no details. III. 9, Jakob K., a bleeder. III. 10, a bleeder. III. 11, not affected. III. 12, healthy family. IV. 1, a bleeder, no details. (See Bibl. No. 607.)

Fig. 563. *Müller's Case.* History of a male adult, III. 2, with symptoms of internal haemorrhage and short history of previous haemorrhages. Several members of the family stated to have been bleeders without the exhibition of data to justify the diagnosis. I. 1, suffered from the haemorrhagic diathesis. II. 2, II. 3, bleeders. II. 4, male, healthy. III. 2, as a child bled severely from small wounds, and had severe epistaxis 4—6 times a year. At the age of 12 he had haematemesis and pain in abdomen and subsequently five similar attacks. Admitted into Stephans-Hospital in Reichenberg, in April 1909, with pain in right epigastrium, melaena and haematemesis. III. 3, sister of III. 2, stated to be a bleeder. IV. 1, male, and IV. 2, female, said to be bleeders. (See Bibl. No. 899.)

Fig. 564. *Nesterovski's Case.* Case of alleged haemophilia, V. 4, manifesting itself after circumcision. Eight other members were described as bleeders. Two of these died from haemorrhage after circumcision and the rest of the male children suffered severely at that operation. (See Bibl. No. 659.)

Fig. 565. *Kerr's Case.* An American case from Denver, Colorado. Gen. I., healthy so far as is known. II. 2, healthy. II. 3, aged 43 years, not a bleeder. II. 4, wife of II. 2, aged 34, had never bled profusely at any time, not even in connection with four miscarriages (III. 7). II. 5, female, aged 32, healthy. II. 6, male bleeder, bled to death from an operation at the age of 17. II. 7, male, died at the age of 14, not haemophilic. III. 1, girl aged 13, always healthy. III. 2, Kerr's case, male, aged 10, presumably of white race, a bleeder. Livid spots appeared on his extremities at the age of 5 months. At $2\frac{1}{2}$ years he bled from nose and mouth for five days, being attacked in like manner at $3\frac{1}{2}$, $4\frac{1}{2}$, and 8 years. In Jan. 1899, oozing of blood from abrasion of the gum, pain and swelling of right knee and elbow, and "ecchymotic copper coloured spots on left forearm." Knee swollen for months. III. 3, aged 8, recently had some haemorrhage from the bowel, but had not had other haemorrhages. III. 4, died shortly after birth from umbilical haemorrhage. III. 5, died of inanition at the age of 6 months; had one attack of bleeding. III. 6, male, aged 10 months. Bled severely after circumcision performed when he was 4 months old; ecchymoses in legs and arms, with underlying movable masses (haematomata?); over 5th rib a similar bruised area. III. 7, four miscarriages 4—9 weeks after conceptions. (See Bibl. No. 711.)

Fig. 566. *Fussell's Case.* Haemophilia in two brothers, IV. 1, and IV. 2, living in Camden, New Jersey. It is stated that they belonged to a bleeder family not previously reported, although evidence of haemophilia is not very definite in the ascent. I. 1, and I. 2, no information. II. 2, "subject to

haemorrhage and when young was found dead from haemorrhage from some unknown point." II. 3, died of phthisis, aged 45. II. 5, and 6, no information. III. 1, healthy. III. 2, subject to epistaxis when young. III. 3, healthy, aged 40. IV. 1, E. C., aged 10, native of U.S.A., a bleeder. When a baby, was cut with a piece of glass and bled for a long time. At 3 had epistaxis. At 5 bled from small cut on forehead. Bandages were soaked with blood. Stitches inserted. Haemorrhage from stitches. Bled for a week after tooth extraction. Large subcutaneous extravasation of blood over right side of chest. IV. 2, H. C., aged 4. At 11 months fell out of bed and struck his head upon which a haematoma developed "the size of a water bucket." At 1 year, fell and knocked out a tooth, haemorrhage from socket for a week. At 2 bled for ten days from a cut on forehead. Subcutaneous haemorrhage on inner aspect of left arm; remains of old subcutaneous haemorrhages on legs. Joints normal. After one fall a large haematoma developed reaching from the axilla to the margin of the ribs and laterally extending from the nipple line to the scapula. Its level was one inch above that of the skin. (See Bibl. No. 656.)

Fig. 567. *Nash's Case.* I. 1, 2, 3, 4, not specifically mentioned. II. 2, alive. II. 3, died of pleurisy. III. 1, died from haemorrhage after a cut received during an "epileptic" fit. III. 2, 3, 4, died from haemorrhage the result of accidents. No details. III. 5, A. B., aged 28. Had epistaxis at the age of 3. Bled for seven days after tooth extraction at age of 12. At age of 25 cut forehead and bled four days. Admitted into hospital with marked bruising over great trochanter and extending to middle of thigh. Received injury to olecranon and developed an abscess. Considerable bleeding followed a small incision for evacuation of pus. Readmitted five months later with haemarthrosis of right elbow, and one month later had haemorrhages into both knee joints. Had had seven attacks of epistaxis and once bled from bowel but only for two days. III. 6, and 7, dead; cause of death unknown. III. 8, 9, 10, healthy. (See Bibl. No. 686.)

Fig. 568. *D'Arcy Power's Case.* D'Arcy Power has described a very severe case of haemophilia, III. 1, with joint lesions. A brother died of bleeding. I. 1, died of rheumatic gout. I. 2, no history. I. 3, died of influenza at the age of 70. I. 4, no information. II. 2, not a bleeder. II. 3, not a bleeder. II. 4, died young of "bleeding from the head." II. 5, died of phthisis. III. 1, a bleeder. Since the age of 2 he was always liable to swellings of the joints, the attacks being preceded by pain, restlessness at night and a peculiar increase in the size of the small blood vessels on the cheeks. He was first seen by Power in 1895 being then 7 years old. He had fallen down a stair and his knee had swelled. Aspiration was performed and dark treacle-like blood evacuated. Again in 1895 he had oozing from the gums after the extraction of a tooth and his left elbow was swollen. Later an immense subcutaneous extravasation of blood occurred in his left popliteal space and extended as low as the ankle. In 1896 right elbow swollen. In 1897 again in the Victoria Hospital for Children, with bruise of face associated with great swelling. A new house surgeon cut into it and only dark fluid blood came out. The boy pulled off the bandages in the night and lost a deal of blood. Readmitted bleeding from a cut lip which had to be cauterised. Now aged 19, and lame from defective movements of both knees. Left elbow bent. Knuckle of index finger swollen. III. 2, in 1895 aged 14 months, bruised his knees and elbows from crawling. In 1896 had severe epistaxis. Bled to death at the age of 11 after having had three teeth extracted. (See Bibl. No. 868.)

Fig. 569. *Mosonyi's Case.* A Hungarian family with three doubtful bleeders. I. 1, died of pneumonia one month after his marriage to his cousin, I. 2, who was healthy. Three years later she married I. 3, who lived to old age. By her first husband, I. 1, I. 2 had one healthy daughter, Veronica, II. 2, and by her second husband seven daughters and three sons. Two of the sons died when a month old, the other bled to death from the nose at the age of 6. Four of the daughters died young. Three were alive and healthy. III. 1, 2, 3, died of an unknown cause. III. 4, 5, healthy. III. 6, aged 6, an alleged bleeder, the evidence being that he bled for two days from a very trifling injury. III. 7, aged 2, suffered from severe and prolonged epistaxis very difficult to control. We are indebted to Prof. Otto Pertik of Budapest for our abstract. (See Bibl. No. 865.)

Fig. 570. *J. C. Hutchison's Case.* I. 1, according to II. 2, suffered in the same way as III. 1, and died from haemorrhage following a slight cut. The tendency existed in the family (males) for three or four generations. II. 2, had a tendency to haemorrhage. III. 1, Willie Clarke, aged about 9, was just starting to walk when he fell and sustained an immense bloody tumour of scalp. This was left alone and was disappearing when he again fell and bruised the other side of his head making another tumour as large as the first. Frequent epistaxis. Once bled from a small wound on dorsum of tongue and Hutchison was called in. Styptics and cautery were of no avail but acupressure stopped it. Puffiness and tenderness of joints, especially knees, from slightest injury, sometimes spontaneously. In one of these attacks, Hutchison punctured the joint and withdrew a syringe full of blood and serum. III. 2, born 1871. Delivered by Hutchison. At 3 months III. 1 threw a piece of slate at him, producing a scalp wound, of which he died in a few hours from uncontrollable haemorrhage. III. 3, and 4, unaffected. (See Bibl. No. 374.)

Fig. 571. *Barlow's Case.* A male bleeder; no other cases of haemophilia in the family. I. 1—I. 4,

healthy. II. 2, died of consumption at the age of 45. II. 3, wife of II. 2, lost much blood during her labours, but not at other times. II. 4, sixteen brothers and sisters of II. 3. III. 1, four children, dead in infancy. III. 2, and III. 3, consumptive. III. 6, 7, 8, healthy. III. 4, a bleeder. Was healthy as a baby but about the age of 2 years, was noticed to bruise easily on his arms, legs and thighs. Often "frightful places" as big as a 5/- piece would come out upon him spontaneously. At 12½ had epistaxis and bled nineteen hours. Tooth extraction, however, was not followed by severe haemorrhage, although he suffered from spontaneous haemorrhages from the gums. At 21 he passed blood from the bowel. Joints were affected for the first time at the age of 25, when both his knees became involved. During the last five years he had had three joint attacks, for one of which he was treated in Charing Cross Hospital for a month. At the age of 29 he was examined by Barlow, who found him suffering from haematuria without apparent cause. On one of his legs he had a bruise the size of a shilling. This led to enquiry with regard to his history, when the above facts were elicited. (See Bibl. No. 358.)

Fig. 572. *Tamme Beth's Case.* A supposed case of haemophilia in a female. I. 1, and I. 2, healthy. II. 1, and II. 2, both suffered from epistaxis. II. 3, a brother of II. 2, vomited blood several times. III. 1, a peasant girl, aged 17, healthy but "not with the physique usually seen in peasant girls." She menstruated normally and was healthy up to the age of 17. About this time she had an attack of pleurisy which was followed by a whitlow on the right hand. It bled for a long time and rendered her anaemic. A doctor made an incision and removed a piece of dead bone: this was followed by copious haemorrhage which neither astringents, caustics, nor the cautery could stop. Ultimately the phalanx was amputated, but after apparent healing had occurred, haemorrhage broke out in the scar, and various means, including phlebotomy, were tried till the patient was exhausted. She then complained of headache and pain in the chest with a sense of oppression. Resort was had to animal magnetism after which the bleeding stopped. Nine months later she was again in the hospital in Gröningen with a quartan ague. Venesection was performed thirteen times. She had not menstruated for eighteen weeks, but the menses now started and continued for ten weeks on end. She was again seized with pleurisy, and was bled another eleven times. Later, blood began to flow from the scar on her forefinger again, and also from the middle and ring fingers. She was again mesmerised and ultimately recovered. III. 2, had epistaxis. III. 3, died of epistaxis. III. 4—9, healthy. The above history presents no analogy to genuine haemophilia: Professor Wenckebach, to whom we are indebted for our abstract of Tamme Beth's very inaccessible paper, attributes the condition to secondary anaemia in a hysterical girl. (See Bibl. No. 56.)

Fig. 573. *Heymann's Case.* Case occurring in one of the most important inland families in Palembang, Sumatra. II. 1, died at the age of 56. II. 2, "the mother was a bleeder." II. 3, died from haemorrhage after circumcision, which according to custom was carried out about puberty. III. 1, died from trifling cut in upper lip. III. 2, died from injury to finger. III. 3, died from spontaneous haemorrhage from gums. III. 4, aged 13, nearly bled to death after circumcision, but was saved by a European doctor. There was no condition in the family known which might account for these incidents. None of these cases seem to have been seen by Heymann, whose account is very short and of little value. (See Bibl. No. 242.)

Fig. 574. *Greig Smith's Case I.* The following family was described by Greig Smith in 1884, Henry Skelton in 1887, and Shaw in 1897. Skelton was the family physician and Shaw elaborated the facts and brought them up to date. Where the accounts differ, we have in most cases followed Shaw. I. 1, died middle-aged of asthma. II. 1, all died young. II. 2, healthy adult. II. 3, died of a gastric ulcer. II. 4, asthmatic; she always lost a lot of blood at her deliveries. III. 1, Alfred P., aged 30, was born in London, and at 17 months, was in St Thomas's Hospital with swelling of the left knee. He was told that the skin burst and the wound was kept open one month. At the age of 5 he was in the Bristol Hospital for Sick Children with the knee in the same condition, and again at 7 with the same joint affected in the Bristol General Hospital. At 11 he was struck by a stone on the inside of his left knee. The joint was very much swollen, and he was detained four months. During this time the right knee and elbow were aspirated and blood was withdrawn from both joints. (Photo in Shaw's paper.) Later he had attacks of haematemesis lasting two months. At 13 he suffered much from epistaxis. At 14 he was seen by Skelton with haematuria. A year later he had bleeding from the mouth and swollen joints. At the age of 17 he was again in hospital under Greig Smith with uncontrollable haemorrhage from a scratch on the ball of his thumb. The styptics tried for three weeks had produced a slough the size of a florin. The haemorrhage stopped in five days. A fortnight later while in bed, at night, he woke up with his left elbow very painful. It was full of fluid. The joint was normal again in three days. Six weeks later he had haemorrhage from the gums. This recurred at the ages of 18 and 20, in which year he had smallpox without haemorrhage. At 21, he was said to have had an attack of rheumatic fever. At 28, haemorrhage occurred for one week after tooth extraction. At 30, he was thin and cadaverous. His teeth were very bad, his gums spongy and liable to bleed. The urine contained blood. Osteoarthritic changes (X ray in Shaw's paper) were present in the knees. Ecchymoses followed a hypodermic puncture, but the collection of blood samples produced no haemorrhage. Throughout his life haemorrhages were preceded by an "aura": twitching of the eyelids two or three days before. Pains and swelling of the larger joints had

been preceded by a sensation of heat. III. 2, at the age of 16 months fell from a perambulator and developed an abscess on the vertex. This being incised, the child bled to death. III. 3, Ernest C. P., younger than III. 2. At 17 months bled to death from blood welling out of the gums or tooth sockets. III. 4—8, unaffected and grown up. (See Bibl. Nos. 483, 524 and 664.)

Fig. 575. *Greig Smith's Case II.* II. 3, died in childhood of haemorrhage from a bitten tongue. II. 4, died 24 hours after tooth extraction. III. 1, aged 12, a bleeder, who suffered from haemorrhages from mouth and nose and also extravasations under the skin. III. 2, seen at the age of 15 months, after bleeding 12 days from superficial abrasions on the fingers and palm, the result of a fall. The child was never free from bruises, but had never bled before. No signs of any constitutional disease. (See Bibl. No. 483.)

Fig. 576. *Holloway's Case.* Family living in America, of German descent on maternal side. I. 1, and I. 2, healthy. I. 3, and I. 4, Germans, not related. I. 4, alive, but unable to give any information of her ancestors. Her own family consisted of five daughters and three sons, II. 3—10, all of whom with one exception were healthy. The exception was one girl, who had joint swellings, but these were regarded as rheumatic. III. 1, Frank, aged 29, a bleeder; first manifested the symptoms of the disease in infancy, when he was frequently bruised and suffered haemorrhages from trivial injuries. His first serious bleeding was at 2 when he cut his tongue with a tooth. He became exsanguine and unconscious from loss of blood. At 7 years very severe epistaxis; at 9 he bled to unconsciousness from wound of finger. He bled when shedding his teeth. One of them was extracted and he nearly bled to death. At 25 he had great oozing from a cut on the hand. He had on many occasions had swollen joints and bruises coming on spontaneously or as the result of slight injuries. III. 2, Charles, a bleeder, first serious haemorrhage during shedding of temporary teeth. Second, great haemorrhage was from a carbuncle on the elbow, which was incised and he bled to death. III. 3, and 4, girls, had never shown any tendency to bleed. III. 5, had "swellings," but never bled externally till he shed his milk teeth. At 16 sustained a great haemorrhage from slight scalp wound. Had many attacks of epistaxis lasting with intermissions for weeks. During the last three years had swellings of joints and haemorrhages into the skin. III. 6, girl, not a bleeder. (See Bibl. No. 494.)

Fig. 577. *Speidel's Case.* Case of fatal haemorrhage from a small wound in the scalp of a boy aged 5. Indefinite history in an aunt's son and in three "Vetter" the exact relationship of whom is not stated. I. 1, and 2, II. 1, 2, 3, 4, no information. III. 1, P. S., a boy, aged 5, received a small wound on the head from a stone. Bled profusely at first and later, in spite of pressure and styptics. For a short time the bleeding was controlled, but ultimately it recurred and he bled to death on the fifth day. Some suspicion of foul play having arisen the body was exhumed two days after burial and an autopsy made. Extreme anaemia of all the organs but no evidence of injury except the small wound on the scalp. At the inquest it was elicited that he came of a bleeder family, the evidence being that a cousin, III. 3, bled to death from an ulcer on the scalp at the age of 5, and three cousins (Vetter), aged 9, 17, and 25 respectively, bled to death from small wounds (one wound being from cutting a sausage). It is expressly stated that the females of the family were free from haemophilia. Speidel also describes a case of great haemorrhage in a girl after the extraction of a tooth, but says there was no history in the family. (See Bibl. No. 525.)

Fig. 578. *Finger's Case.* History of a boy, III. 4, the son of a colleague of Finger, of Lemberg. Maternal grandmother, I. 4, a Jewess. I. 1, and I. 2, healthy; likewise I. 3, and I. 4. II. 2, a doctor of German origin (Schlesien, according to Grandidier). II. 3, a Pole, died of pulmonary tuberculosis. II. 4, and II. 5, brothers and sisters, alive and well, known to Finger. III. 1, female, aged 18, had tuberculosis. III. 2, healthy. III. 3, aged 10, ill developed but otherwise healthy, had epistaxis. III. 4, male, aged 6, slenderly built, had been affected for three years with recurrent haemorrhages from nose, tonsils and gums. Suggillations and bruises constantly occurred spontaneously, also swellings of joints, especially knees and ankles. This case was also communicated by Finger to Grandidier (see Bibl. No. 267, p. 330), who stated that it had not been published, although Finger's account appeared a year before. (See Bibl. No. 254.)

Fig. 579. *Osborne's Case.* A case of relatively little value published in 1835. II. 1, when 9 years old was cupped for "some affection of the knee joint." After he returned home from the hospital, the haemorrhage restarted, but was allowed to continue as being beneficial. He collapsed, and though assistance was then obtained, he died on the following day. II. 2, John Gamble, aged 13, was seen by the author with inflammation of the capsule of the left shoulder joint. Two leeches were applied, and haemorrhage lasted for three days. II. 3, fell down and struck his temple on the corner of a table at the age of 6. Uncontrollable haemorrhage occurred and death followed. II. 4, no haemorrhage at childbirth. III. 1, no information. (See Bibl. No. 90.)

PLATE XLIX. Fig. 580. *Cousins' Case.* In 1869 Cousins published a case of haemophilia in a boy, II. 2, with a remark that a brother, II. 3, had "manifested in a less degree a tendency to constitutional bleeding, and several members of the family have a rheumatic tendency." Cousins' patient, W. B., II. 2, aged 16, was the subject of profuse bleeding from early life, starting at the age of 10 months. The haemorrhages were mainly nasal, pulmonary, intestinal and subcutaneous. He had had pains in the

limbs and at about the age of 15 he had chronic rheumatism of the right knee and elbow. At the age of 16 he was accidentally pushed down. He struck his forehead over the eyebrow and a few hours later had an attack of epilepsy. The whole of the left side of his face and forehead was swollen with blood. He vomited, suffered great pain, had frequent fits, but ultimately recovered. Not long after, he struck his forehead in another fit and died unconscious. *Post mortem*, an extensive blood extravasation was found at the base of his brain. (See Bibl. No. 307.)

Figs. 581 and 582. *Muir's Cases.* In 1906 Muir published a few details of cases of haemophilia occurring in certain related "Cape Colonial" families. In this paper he confined himself to one line of descent containing 27 bleeders, "merely mentioning that there are others equally marked with disaster and tragedy." On examination of the pedigree it will be seen that the affected persons are male only, and that the disease is propagated by the unaffected daughters, with one (excluding the doubtful, I. 1) exception. This exception is III. 6, who apparently was responsible for all the subsequent cases. "Intermarriage was, however, common in these days," but was denied in this case.

Case I. I. 1, Pieter Willem C——, born 1783, "his is the first clear case and he died of epistaxis in 1852, aged 69 years." I. 2, his wife, Anna Susanna J. II. 6, Elsie Maria C. II. 8, this woman "started a haemophilic line with numerous subsequent cases." II. 9, five males, unaffected. III. 1, and 2, unaffected. III. 3, 4, and 5, "died of haemophilia." III. 6, Pieter Willem de B., died of haemorrhage from an adze cut. III. 7, Johanna de B.; said to have been of healthy stock and without consanguinity. III. 8, *vide* II. 8. IV. 2, Elsie de B. IV. 3, Maria de B. IV. 4, Johannes H. IV. 5, unaffected. V. 1, and 2, died of haemorrhage. V. 3, at the age of 5 years while playing "Klippertjes" sustained a minute abrasion on the toe and succumbed to the bleeding after nine days. V. 4, unaffected. V. 6, Mrs J., living. V. 8, Mrs E., living. V. 10, Mrs A., living. V. 12, Mrs M., living. V. 13, living. VI. 1, a year before his death, he bled seriously from the gums after eating mealies on the cob. He died after nine days' haemorrhage from "an almost invisible" axe wound on the head. VI. 2, died at the age of 2 years of a five days' haemorrhage from an injured toe. VI. 3, unaffected. VI. 5, Mrs C. B., stated that "doctors can do nothing for the bleeding disease." VI. 8, unaffected. VI. 9, died of bleeding. VI. 10, "has had severe haemorrhages and is bruised easily." VI. 14, aged 2 years, died of haemorrhage lasting 13 hours, after incision into an "abscess." VI. 15, aged 7 years, injured his heel with an axe and died in 10 days. VI. 16, died in 1896 at the age of 28 years, 24 hours after an "abscess" at the site of an old fracture had been incised. VI. 17, bled for 10 days from under the upper lip. VI. 18, several died very young and some have undergone tooth extraction without unusual consequences. VI. 19, Mrs J. C. VI. 22, Mrs J. van S. VI. 25, Mrs B. VI. 29, aged 25, a cripple from joint disease. The first bleeder seen by Muir. Ten days before, he had fallen from his horse and had bruised his hip. Muir found the signs of an abscess and was about to incise it, when he was told of the family liability. He, however, punctured the swelling and drew off blood. In one month the swelling was completely absorbed. VI. 30, also a cripple from haemophilic joint disease. VI. 31, and 32, unaffected. VII. 1, died at the age of 13 months of haemorrhage from the fraenum of the upper lip, injured by a fall from the nurse's arms. VII. 2, died shortly after birth of a "tremendous haemorrhage from the lower bowel." VII. 3, brought to Muir while suffering from haemorrhage from the fraenum of the upper lip. The bleeding was stopped by adrenalin in five minutes. VII. 4, "bleeder," bled "from the mouth on one occasion." VII. 5, "bleeder." Developed bruises from the slightest cause. VII. 6, bleeder. VII. 7, "haemophilic." VII. 8, died of haemorrhage from a finger, cut by a stone. VII. 9, bleeder. VII. 10, unaffected. VII. 11, unaffected. It will be noticed that the author in his title refers to "eight generations of haemophilia in South Africa"; we are, however, able to detect seven only. In addition to the above cases, another family, Case II., is described which is related to them in a manner not specified.

Case II. I. 2, nearly 80 years old: source of information. II. 1, Carel, died at the age of 9 "from a fall and a scratch on the lip." II. 2, Cornelis, died of epistaxis after bumping his nose at play. II. 3, Frederick, fell off a ladder and ruptured the fraenum of the upper lip. Died of the resulting haemorrhage at the age of 5 years. II. 4, Jacobus. At the age of 5 he fell on his face and died of epistaxis. II. 5, Daniel, aged 25, bleeder: he died of epistaxis spontaneous in onset. II. 6, 7, and 8, unaffected. II. 9, and 10, unmarried. (See Bibl. No. 816.)

Fig. 583. *Consbruch's Case.* This is one of the oldest reported instances of haemophilia, and was described by Consbruch of Bielefeld in 1810. According to Nasse (1820) it is "unquestionably the same family" as that described by the author of the *Medicinische Ephemeriden*, Chemnitz, 1793, p. 267 (see Bibl. No. 13); the family living at Ravens-berg near Bielefeld in the Teutoburger Wald. Consbruch was known to Nasse, and the latter (p. 399) speaks of having known one of the bleeders in the family. This would be II. 4, or III. 2, and Nasse states that he died later of haematemesis. I. 1, and I. 2, were healthy, and had several children, the females being healthy. One female, II. 2, is specially mentioned. II. 4, a bleeder. In early youth he was subject to dangerous bleedings and still was not quite free. His joints were also affected and at the end of his attacks he showed ecchymoses. III. 1, male, bled to death from a small wound. III. 2, bled only in spring and autumn, either from nose or from small wounds. If he escaped either or both of these, his joints swelled. On one occasion he wounded his arm and

compression had to be kept up for weeks. III. 3, other brothers presumed to exist from the context. Although there is no doubt, from various data, that the author of the *Medicinische Ephemeriden* is Consbruch, and that the two accounts refer to the same family, it will be seen from the following complete translation of the former paper that there are points of difference. "On the 4th of November I was called to the country to see a boy of 11 years, who two days before had slightly cut his thumb. All medical applications proved unable to stem the haemorrhage, and before I was able to arrive he had bled to death. A brother of this boy had, a few years before, died from the results of a slight cut, while several *brothers* of the mother had similarly met their end. All females in this family are, so far as I am aware, free from this unhappy idiosyncrasy. The brothers and sisters of the mother, her children and the woman herself have very fine transparent skin, showing the blood-vessels beneath, and raven black hair. The women menstruate normally and are perfectly healthy. The males are extraordinarily liable to bleed from the nose, when the blood may readily become abundant and hurry them to the grave. As regards its physiology and pathology, this is a curious circumstance, which I am unable fully to explain." (See Bibl. Nos. 13, 22 and 35.)

Fig. 584. *Froelich's Case.* In an important paper on haemophilic joints Froelich reported four cases in one of which there was a certain amount of evidence of a family tendency to bleeding. It was the case first communicated to him by Dr Treff of Gondrecourt, and referred to the history of a bleeder, living in Grand (Vosges). This man, aged 20, was however subsequently seen by Froelich (see Bibl. No. 787). I. 1, stated to have been a bleeder and to have suffered from rheumatism, no details. II. 1, and II. 2, healthy. III. 1, the patient, aged 20, a tinsmith. At the age of 3 the right great toe swelled, and this was followed by swellings of the knees, elbows, and ankles. Between his joint attacks he had frequent epistaxis and considerable haemorrhages from trifling wounds. At 14 the right hip joint became swollen and painful, a recurrence taking place three years later. In 1901 he fell from a roof and sustained a compound fracture of the forearm bones, with great haemorrhage. Against Froelich's advice he served as a soldier, and in 1903 his right hip became involved. Thinking it was an osteomyelitis a military surgeon incised it and patient bled to death in 36 hours. The operator informed Froelich that there was extravasation of blood in the quadriceps, adductors and joint capsule of the thigh, the internal iliac fossa being filled with blood. III. 2, male, died of epistaxis at the age of 12. Froelich also described three other cases: (1) a male, aged 7, with swellings of both knee joints: his mother was well, his father had died of general paralysis of the insane; (2) male, aged 9, with ecchymoses and pains in joints; enlargement of right knee. Father well, mother had haemoptysis, and a brother of patient had frequent attacks of epistaxis; (3) girl, aged 4, who bruised from slight injuries and who was lame from a swelling in right thigh in Scarpa's triangle. In the *Zeitschrift für orthopaedische Chirurgie* (see Bibl. No. 787) it is stated that she fell on the crista iliaca and got a haematoma as large as a hen's egg. The father was arthritic; mother "slightly haemophilic" with ecchymoses and *post partum* haemorrhage. The uncle had epistaxis badly. (See Bibl. Nos. 768 and 769.)

Fig. 585. *Jacob's Case.* History of a family residing formerly in Newark and now in Nottingham. Careful enquiry into the family history by the late Dr W. H. Ransom, of Nottingham, failed to reveal a family tendency to the disease. III. 1, and III. 4, were long under treatment by Dr Ransom and were also observed by Sir William Jenner. I. 1, died of softening of the brain and senile decay. I. 2, and 3, no information. I. 4, died of a "stroke." II. 1, died of consumption. II. 2, and II. 3, alive. II. 4, aged 56, living. II. 5, aged 55, living. II. 6, died of Bright's disease, asthma, and eczema. II. 7, dead, no information. II. 8, died of fever in Florida. II. 9, dead; no data. II. 10, alive. III. 1, aged 34, was free from haemophilia until the age of 4. After a blow on the head he showed signs of the disease in the form of haemorrhages, bruises and affections of his joints. At the time of reporting he was crippled in both knee joints and in the elbow joints. These joints had been very hard and painful. He had had scarlet fever, measles, whooping cough and appendicitis, and had suffered from haemorrhage from the bowel and haematemesis. III. 2, aged 32, suffered from violent epistaxis and other haemorrhages. He was very easily bruised and had had much trouble with swellings in his joints. He was a nervous and sensitive man and although not of robust constitution his recuperative powers were very great. Attacks of bleeding occurred at relatively long intervals. III. 4, aged 28, affected very similarly to III. 2, being easily bruised and subject to great haemorrhages. His joints were much affected: a very delicate man. III. 5, not affected. III. 6, not affected. III. 7, four or five children who died in infancy. (See Bibl. No. 888.)

Fig. 586. *Theodor Hirsch's Case.* In 1895 Hirsch published a case of haemophilia observed in Hoffa's clinic. This is manifestly the same as the first case described by H. Goelt four years later (see Bibl. No. 698), and between these two dates the patient had not again been under observation. The two accounts are practically identical. The patient, III. 6, Georg H., aged 25, a law student from W. (Würzburg), had a brother, III. 1, who bled to death at the age of 7 after a fall on the nose, and an uncle, II. 3, "who also suffered from haemorrhages into the knee joint." II. 1, 2, 4, and 5, were all healthy and unaffected. III. 2, died in infancy of a cause not stated. III. 3, died of cholera, while III. 4, 5, and 7 were healthy. III. 6, himself bled badly during the second dentition and once his life was despaired of. Epistaxis was frequent. The first joint lesion occurred at 13, an haemarthrosis of the left knee.

A recurrence in the next year kept him long in bed, and ever since similar conditions in other joints were almost constantly present. At 17 years, after being thrown from a horse, his knee was contracted for three months, and this same condition was produced by a false step later in the year. At the age of 18 his knee was straightened under anaesthesia by von Bergmann and the pain was excessive. He wore retentive apparatus afterwards. Similar accidents occurred till he was 21, when he bled for three weeks after tooth extraction. Under anaesthesia, the thermo-cautery was applied, and the stage of excitation during induction produced bilateral haemarthroses of the knees. When he was 22, haematuria occurred, and blood extravasation in the gluteal region after a carriage accident. When examined in hospital the scars of previous bandages were found on him and gross changes in the joints (fully described). Haemarthrosis of the knee occurred while under observation, and also an acute abdominal attack diagnosed as internal haemorrhage and peritonitis. The condition of his limbs was much bettered on his discharge. Gocht showed three photographs of this man. (See Bibl. No. 628.)

Fig. 587. *Faber's Case.* Faber published a case in 1899 of a bleeder, III. 5, in Denmark who had one brother, III. 6, affected, but no other relative in the history as it appears. II. 2, "is a sufferer from haemophilia" in a manner not specified. II. 4, and 5, were afflicted with rheumatic affections of the joints. III. 1—4, were unaffected. III. 5, the patient under observation, was aged 20, and from the first year of his life "had shown the ordinary symptoms of haemophilia." The joint disease started in his sixth year, and in the course of time many joints had been affected with resulting deformity. The course of the attacks and the physical condition of the joints as described by the author do not differ from the usual. It is stated, however, that the X ray examination revealed no osteophytes and only slight effacement of the normal structure. The brother, III. 6, before-mentioned, died at the age of 6 of epistaxis and is stated to have suffered from the same disease as III. 5. (See Bibl. No. 696.)

Fig. 588. *Moses' Case.* History of a bleeder family living in Greifswald. I. 1, and I. 2, no information. I. 3, alive, not a bleeder. II. 2, died of phthisis. According to III. 3, he suffered from painful swellings of the feet with bluish discoloration which disappeared after a time. II. 3, wife of II. 2, alive and well. III. 1, female, aged 18, suffered frequently from copious epistaxis which always lasted a considerable time. III. 2, male, also subject to epistaxis in severe form and presented joint swellings. III. 3, Hugo Giertz, suffered from his earliest years with epistaxis and swellings of the joints. Extensive bruises were also very frequent especially on his back and breast. On several occasions he became unconscious after epistaxis, but his recovery was observed to be very rapid. At the age of 12 he cut his hand on the flexor aspect with a piece of glass. This was followed by violent haemorrhage for which he was admitted into the surgical clinic in Greifswald. No bleeding point could be found. The wound was plugged and the bleeding was thus temporarily arrested. The right arm became greatly swollen, and as a subcutaneous haemorrhage was suspected, the bandages were removed. This showed a wound about 20 cm. long (2·0 cm.?) from which there was great parenchymatous haemorrhage. A vein was ligatured and the wound again plugged. Next day the whole bed was soaked with blood. Ultimately by the use of Penghawer Djambie and compresses the bleeding ceased. Some months later he was again seen bleeding copiously from the gums, and necessitating his readmission into the clinic. A large haematoma was also present in his left leg and extending from the knee to the ankle. Haemarthrosis of ankle joint. After six days the bleeding from the mouth ceased and he rapidly recovered, so that he was able to take his discharge on the 18th day. A few months later his leg swelled and discoloration appeared over the malleoli. Shortly afterwards he fell on his head on a stone and a large haematoma followed accompanied by haemorrhage. A month later he cut his finger with a knife, but the haemorrhage which ensued was slight. It may be remarked that the conditions under which this boy lived were extremely unhygienic, and he frequently had to go without food for days. III. 7, a female, died of tuberculous meningitis; not a bleeder. III. 9, a bleeder, dead; no details. (See Bibl. No. 589.)

M'Causland's Cases. In a general paper on haemophilia M'Causland refers briefly to seven cases, in two of which no history of the disease could be traced in the ascent.

Fig. 589. *Case I.* This is of interest in so far that the patient, III. 5, who was a law student, made a careful enquiry into the family history, but was unable to find any evidence of the disease within the preceding three generations. His two brothers, III. 1 and III. 3, were healthy, and had healthy families. Both his sisters were healthy, and one of them, III. 6, was married and had a healthy family. The case occurred in the practice of Dr Gillman Morehead, to whom we are indebted for our particulars. III. 5, aged 30, an Irish law student, had infantile paralysis, and was lame in the right leg. He gave a history of bleeding from small wounds. Morehead attended him in 1904 for a large haematoma extending from the middle of the posterior surface of the thigh to well above the iliac crest. It was induced by sitting down suddenly on the gunwale of a boat. Later he bled from the mouth. His teeth were very decayed, but no dentist had ventured to extract them. The haemorrhage started after a mercurial course, and could not be stopped. Under nitrous oxide it was decided to remove a tooth. Six hours after, bleeding started and lasted for nine days, when it spontaneously stopped. During this period he had a spontaneous haemorrhage into the left elbow joint. A hypodermic injection of morphine into the right forearm resulted in a subcutaneous haemorrhage extending from the right wrist to the pectoral region. A year later

he received a cut from the stem of a pipe he was smoking, and death ensued, partly from haemorrhage and partly from asphyxia, the result of extravasation into the substance of the tongue. Since the pedigree was engraved we have obtained the additional information that I. 4 had healthy brothers and sisters, and that the three families in the fourth generation, marked with a query in the pedigree, consisted of one daughter, two daughters and a son, and one son respectively.

Fig. 599. *Case II.* A boy, III. 3, under the care of Dr Gillman Morehead in Dublin, with a very large subcutaneous haemorrhage extending from the knee all over the thigh as far as Poupart's ligament and the iliac crest. He had been twice previously in hospital, once for epistaxis and once for copious bleeding from a scratch. The mother, II. 3, an unintelligent woman, denied the existence of haemophilia in her family. Two of her brothers, II. 1, and II. 2, were healthy, as were also the patient's two brothers, III. 1, and III. 2. (See Bibl. No. 893.)

Fig. 590. *Clay's Case.* I. 1, and I. 2, presumably normal. II. 3, William, bled freely for some days after the extraction of a tooth; no other data. II. 4, Joshua, injured his head and bled for some days, and then the haemorrhage "only yielded to very energetic treatment." II. 5, when about 14 bled for eleven days after tooth extraction. III. 1, bled for seven or eight days after tooth extraction. III. 3, cut his finger and bled two or three days. On another occasion he bled three or four days after having a tooth removed. III. 2, Joshua, patient of Dr Clay (of Piccadilly, Manchester). The history of his bleedings was as follows: at 4 the extraction of a tooth caused him to bleed four days. At 11 he cut his thumb and bled five or six days. At 12 he bled two or three days. It is to be noticed, however, that none of these haemorrhages were the subject of remark, and were only elicited during the time Clay was attending him at the age of 14 for haemorrhage following tooth extraction. The tooth, a molar, had been removed by a druggist, and there was great laceration of gums and even the cheek. The alveolar process was broken. Blood was of "a decidedly arterial character." Every means were employed to arrest the haemorrhage, but it lasted ten days. The evidence of haemophilia in this family as recorded by Clay is quite inconclusive, although it is impossible to assert that the affected individuals were not bleeders. In the only case, III. 2, in which we have details it is evident that the damage done during the operation was such that very considerable bleeding might have been expected even in a normal person. (See Bibl. No. 162.)

Fig. 591. *Schreyer's Case.* Kretzschmar family of five boys living in Tirpersdorf in Vogtland (Sachsen). The parents and grandparents were healthy and free from haemophilic taint. III. 1, male, bit his tongue, and in spite of all remedies bled to death. III. 2, and III. 3, two boys, both healthy. III. 4, aged 5; livid spots appeared on his legs, especially below the knee. These spots developed into lumps as big as a pigeon's egg. When wounded it was very difficult to control the haemorrhage, which continued till he was blanched and syncopic. Was strong and healthy otherwise. At 5 had very severe bleeding from a hollow tooth. III. 5, a bleeder, with symptoms similar to those of III. 4, but not so severe. III. 4 and 5, were attended by Schreyer. (See Bibl. No. 54.)

Fig. 592. *David Burnes' Case.* A doubtful case of haemophilia. I. 1, and I. 2, no information. II. 2, healthy. II. 3, and II. 4, "had the haemorrhagic tendency." III. 1, male, aged 44, seen by Burnes in 1839, suffering from orchitis. Leeches were applied with benefit, but the oozing from their bites could not be arrested for days. Later he cut his finger in opening a window. The primary bleeding was arrested, but inflammation set in, associated with secondary haemorrhage, and he was brought back to London from Margate, where he had gone to recuperate. Four days after his return it stopped. Twice after taking blue pill he was seized with epistaxis, and bled from the gums. III. 2, Charles, aged 30, seen by Burnes in 1837. Leeches were applied to the abdomen on account of enteritis, and he bled for eight days from the bites. In 1840, however, he was circumcised without any unusual haemorrhage. III. 3, John, aged 25, seen by Dr Hooper of Buntingford (Herts.) in 1827, who however communicated the facts to Burnes from memory, as his notes at the time had been burned. The history was that III. 3 fell from his horse and severely injured his arm. In spite of the statement of the patient that he bled copiously from the slightest scratch and that he nearly died after tooth extraction, twelve leeches were applied, and the bleeding had to be arrested by lunar caustic. The injured arm suppurated, and he died. III. 4—10, normal. We consider the diagnosis of haemophilia is not justified by the evidence given in the cases III. 1—3. We have marked II. 3 and 4 as bleeders because there are no data on which to criticise them. (See Bibl. No. 116.)

Fig. 593. *Knudtzon's Case.* Two bleeders in Kolvereid (Norway). I. 1, and I. 2, not affected. II. 2, anaemic. II. 4, a bleeder, aged 11, first seen by Knudtzon in 1900 for a hydrops of the knee. Later, haemorrhagic spots appeared on the limbs, haemarthrosis of the knee and extravasations of blood under the skin. On one occasion he became profoundly anaemic as the result of bleeding from a loose tooth which Knudtzon removed. Great haemorrhage ensued, but finally it stopped spontaneously. III. 1, aged 3 years; two and a half years previously he cut his lower lip and bled severely for days. Three weeks previously he had had great haemorrhage from an excoriation. Our II. 2 should represent one healthy female and one male who died of a cause unknown. (See Bibl. No. 611.)

Fig. 594. *A. Miles' Case.* A genealogical tree of the following case appeared in Alexis Thomson and Alex.

Miles' *Manual of Surgery* illustrating haemophilia. We have communicated with the authors soliciting further details, but unfortunately the lapse of time has made investigation impossible beyond the written records of the case. I. 2, named Diggle. This family came from Lancashire. Subsequent generations lived near Edinburgh. IV. 6, the mother of the two observed cases, would give no information about the family, but one of her sisters went to a great deal of trouble in collecting the facts on which Mr Miles constructed the pedigree. III. 1, died at the age of 70. III. 9, died in child-bed. V. 4, was under the care of Mr Alexis Thomson, suffering from a trivial accidental wound, which persistently bled in spite of all remedies. The haemorrhage finally stopped after the blood of the house surgeon had been applied to the wound. V. 5, at the age of 9 or 10 years, was seen by Mr Miles at the Royal Infirmary, Edinburgh, alleged to be suffering from acute osteomyelitis. His left leg suddenly became swollen and painful, but the signs were sufficiently atypical to cause Mr Miles to enquire into the history. He then learned that the boy was easily bruised, and found faded bruises upon him. He had never had a severe haemorrhage, but the mother admitted that he bled more than usually. (See Bibl. No. 814.)

Fig. 595. *Caillé's Case.* The following account refers to four children probably rachitic or scorbutic, and suffering from advanced oral sepsis. On the mother's side there was a bad history of tuberculosis. I. 1, and 2, healthy. II. 1, Mr W., aged 41, born in New York of German descent. "Rheumatic polyarthritis" at the age of 33 and epistaxis. II. 2, his wife, aged 30, born in Germany. Married at 20. Menstruation lasted seven to eight days. Haemorrhoids and epistaxis troublesome, especially when she was pregnant. Mouth foul, gums swollen and bleeding. Her sibs, II. 3—6, all died of phthisis. III. 1, George, aged 9, "raised on condensed milk." His gums bled during eruption of his teeth. At the age of 3 epistaxis lasted three weeks. At 4 haematuria occurred with frequency of micturition (two hourly). A year later an abrasion of the lower lip bled for three weeks. At 9 again there was a slight attack of haematuria. "At the present time every fall or blow is followed by ecchymosis of joints....The right knee is markedly swollen with blood clots in the popliteal space." Epistaxis occurred; breath foul; gums bleeding and swollen; "he also has phymosis [sic]." III. 2, Harry, aged 5. Breast-fed for two years. Haemorrhage occurred from the gums during dentition and from injuries to the tongue or mucous membranes. At 3 he fell down a cellar and sustained an inch-long scalp laceration requiring 5½ months' treatment in hospital. "Blood...soiled the bed-clothes every night." Later he was admitted for haemorrhage from the gums and malnutrition. When cured circumcision was performed, and the haemorrhage could not be thoroughly controlled for two weeks. In the same year he was readmitted with haemorrhage from the gums and stomatitis. "Moribund"; "able to go home in three days." III. 3, Viola, aged 2. Suckled for fourteen months. Stomatitis, swollen and bleeding gums. Epistaxis occurred very readily. IV. 4, six months at breast. "Large haematoma at birth." Admitted for purpura haemorrhagica and submaxillary abscess; emptied by aspiration: healed readily. We do not find that the above data conform to the concept of haemophilia. (See Bibl. No. 609.)

Fig. 596. *af Forselles' Case.* An account of a bleeder, II. 2, in Mäntsälä, South Finland. I. 1 and 2, healthy. II. 2, could give no information of previous generations, except that a grandfather had an ulcer of the leg. According to his mother, II. 2, aged 29, had, at the age of 1, a purpuric eruption over the whole body. Then follows an extensive account of injuries producing exhausting bleeding, joints and epistaxis. Two plates. II. 3—6, were healthy, as were III. 1 and 2. (See Bibl. No. 644.)

Fig. 597. *Daland and Robinson's Case.* The following somewhat unconvincing account was published in 1894—5. The subject, III. 6, observed was a boy of 13, whose position in the family is rather obscure but who was probably the youngest. In infancy he had an attack of "cholera infantum" associated with petechiae, and at the age of 5 an arthritis, relieved by salicylic acid. Since the age of 5 he had had fifty bleedings from mucous membranes, chiefly nasal, and once from the tongue after trauma. An haemorrhage following a cut is specified. The first child in the family, III. 1, was premature and died. III. 2, a boy, started to bleed from the nose at 16 months, while at 3½ years he had developed hydrocephalus. Epistaxis continued, and he died of it. The third child, a boy, died at 2 years of scarlet fever. Not affected by haemophilia. The fourth child was a poorly developed boy. After suffering from haemorrhage during the teething period, he died at 18 months of haemorrhage from the mouth. The fifth child, III. 5, died at the age of 3 months without exhibiting signs of haemophilia. There is no evidence of haemophilia in the family. Indeed there is so much that is pathological in the members of this family, that a diagnosis of haemophilia on the data given can hardly be justified. (See Bibl. No. 619.)

Fig. 598. *Bruinsma's Case.* History of a German, II. 1, aged 45. In his family it is stated that haemophilia was frequent. His sister also lost two children (sex ?) through the disease. I. 1, and I. 2, healthy. II. 2, aged 45, Bruinsma's patient. First showed symptoms of haemophilia at 1½ years of age, having wounded his tongue, and bled for eight days. He always bled for days or even weeks from small wounds. At 15 he developed "rheumatism" in his feet and elbows. Haematuria on one occasion which lasted three weeks. From his 21st—26th year he was healthy. After that, however, two attacks of rheumatism, and later, bleeding, which only stopped when he was exhausted. On one occasion he was cauterised, but this failed to arrest the haemorrhage. III. 1, one of the boys of II. 2, showed suggillations on his legs. III. 3, and III. 4, two children, who bled to death while young. (See Bibl. No. 282.)

Fig. 600. *Meinel's Case I.* I. 1, not a bleeder. I. 2, said to have been a bleeder, but the evidence rests only on the statement that she suffered copious haemorrhage from trivial injuries. She menstruated regularly and died in old age of some febrile complaint. I. 3, 4, no information. II. 1, nearly bled to death several times and suffered repeatedly from suggillations. Had a large haematoma on his head from coming in contact with a door. II. 2, sisters (number not stated), healthy. II. 3, a weaver (G. Frank von F.), aged 35, bled profusely from socket after removal of a tooth three years ago. Consulted Meinel for a large haematoma as big as a goose's egg over eyebrow, the result of a push. Had a large haematoma on right thigh. III. 1, family of II. 3, healthy: sex and number not stated.

Fig. 601. *Meinel's Case II.* I. 1, 2, healthy. II. 1, sibs of II. 2, healthy; number and sex not given. II. 2, a starchmaker, Rainer of Eckersmühlen; tall man, with fine white skin, aged 42. Had always bled from trivial injuries and had suffered from suggillations, etc. Meinel was called to see him on May 8th, 1848, and found him suffering from uncontrollable epistaxis. Belocq's tamponage had to be resorted to, and the bleeding was temporarily arrested, but recurred on removal of the cannula. Ultimate recovery. Had a venous murmur. III. 1, healthy. III. 2, repeatedly seen by Meinel with epistaxis leading to complete collapse. Had venous murmur. The evidence of haemophilia in this case is very meagre.

Meinel's Case III. Frau Gebhardt, of Wallesan, aged 48. At the age of 27 the extraction of a tooth led to three days' bleeding, which ultimately necessitated the actual cautery. She had also had epistaxis. Her father died young. She married a healthy man, and had two boys and two girls, all healthy. Meinel himself considered this a doubtful case of inherited tendency to bleeding, with which opinion we are in accord. (See Bibl. No. 195.)

Fig. 602. *Graham's Case.* A case, II. 1, in Australia. I. 1, strong and healthy. I. 2, died 16 years ago, but there was no history of haemophilia in her relations. II. 1, two years older than II. 2, had two great haemorrhages, once after the removal of a tooth, and once from injury to finger. His joints were also affected. II. 2, aged 20, in Prince Alfred Hospital, Sydney, suffering from swelling of left knee as the result of a knock against a stone. Aspiration revealed synovial fluid, but from the puncture there was great haemorrhage. II. 3, bled to death from wound of foot due to the bite of a rat. II. 4, and II. 6, two healthy females. II. 7, five children, dead. III. 1, six healthy children. The evidence of haemophilia in this family is not convincing. (See Bibl. No. 512.)

PLATE L. Fig. 603. *Gettings' Case.* We are greatly indebted to Dr H. S. Gettings, of Chase Lodge, Chasetown, near Walsall, for permission to publish the history of this family which is intimately known to him and has been observed over a long period. A peculiar interest attaches to this case in so far that typical haemophilia has been observed in one sibship only (VI. 49—57), although the medical history of a very large number of the individuals referred to is accurately known. Up to the present no "de novo haemophilia" has been recorded with anything like the minuteness of Dr Gettings' case. I. 1, and I. 2, no data. II. 1, 3, 4, 5, no information with respect to their medical history. III. 1, R. G., of Bilston, born 1777 (?), died 1810. III. 2, C. W., of Bilston, wife of III. 1, born 1775, died 1849. III. 3, R. S., born 1775, died 1809. III. 4, M. S., born 1777, died 1803. III. 8, G. S., born 1791, died 1849. III. 10, J. S., born 1765, died 1835. III. 12, J. B., of Bilston, born 1782, died 1833. III. 13, Miss C., of Sedgeley. III. 14, J. K. III. 15, Miss S., born about 1788, died of heart disease in 1836 (?). IV. 1, Elizabeth G., born 1806, died 1868. IV. 3, William G., born in 1798. IV. 5, John G., born 1800, died 1879, not a bleeder. IV. 6, Mary S., born 1802, died 1883, not affected. IV. 7, John S., born 1800, died 1852, not affected. IV. 10, 12, 13, unaffected. IV. 20, maternal grandmother of bleeders, born 1811, died 1859, of heart disease. She was, otherwise, a heathy woman. She married, in 1832, IV. 19, Thomas B., born 1800, died 1872, and by him had eight children, viz. five daughters and three sons. One of these daughters, V. 79, was the mother of bleeders. IV. 21, Emma K., born 1822, died demented in 1892. IV. 22, James K., born 1823, died of heart disease in 1870, not a bleeder. IV. 24, Anne K., born 1826, died of effects of a ventral hernia in 1893. V. 1—V. 36, known to have been unaffected. V. 36, father of bleeders, had never manifested any tendency to bleed. V. 37—67, all unaffected. V. 68, Mary B., born 1833, died in 1907 of pneumonia. She was always a very healthy woman. V. 70, Louisa B., born 1836, died of fatty degeneration of the heart in 1904. She never manifested an excessive tendency to bleed. V. 72, born 1836, died of heart disease following rheumatism in 1854. V. 73, Eliza B., born 1842, alive, healthy. Much of the family history was supplied by her, particularly of persons not known to Dr Gettings. V. 75, Thomas B., alive, healthy, had never shown a haemophilic tendency. V. 77, Henry B., born 1847, was drowned between the ages of 5 and 10. Up to that time he showed no symptoms of haemophilia. V. 78, a male infant, name and date uncertain, but known to have shown no signs of haemophilia. V. 79, Helen B., mother of bleeders, born 1851, died of pneumonia in 1893. She never manifested the slightest tendency to haemorrhage, either at menstrual periods or after confinements. She was married in 1875 to V. 36, and by him had 11 children. V. 80—86, healthy. V. 87, three female children who died in infancy. V. 88, Walter W., born 1858, died of rheumatism, 1864. V. 89, William W., born 1860, alive, healthy, had never bled. V. 91, four sons, all unaffected. V. 92, alive, healthy. V. 93, Annie W., born 1854, died 1897, healthy, married

V. 94, and died of puerperal fever. VI. 1—48, known to have shown no tendency to haemorrhage. VI. 49, Harold S. G., born 1877, a typical bleeder. He displayed the symptoms before he was 2 years old, showing bruises from the most trivial injury. Epistaxis next developed and at first this was persistent and severe. After the age of 6 the tendency to bleeding from the nose began to disappear and after the age of 12 he was no longer affected. The shedding of the first teeth gave rise to many haemorrhages, but was accomplished without danger to life. When epistaxis disappeared, the joints, previously healthy, began to give trouble. At first, effusions into the knees cleared up without much trouble. At 9 years of age painful effusions into the left elbow gave repeated trouble. In 1887, at 10 years of age, he twisted his left ankle in dropping two or three feet from a wall, and an effusion ensued. In 1888 the right ankle became affected. For some years both ankles were repeatedly the seat of effusions especially after twists. In 1889, after several falls during riding, a large swelling developed in the left knee. At first no great trouble was experienced, but after a walk the joint became acutely swollen and painful, and had to be immobilised for five months. The result of this was limitation of movement. In 1890, the first of repeated attacks of haematuria made its appearance without apparent cause. In 1894 he was an invalid for seven months as a result of a twist of the right knee, and this joint has been repeatedly the subject of effusions since. In 1899 he insisted on having a molar tooth extracted and bled severely for fourteen days. In 1900 an abscess in the gum bled for a week and could only be controlled by digital pressure. Slight haemorrhages from the gums are frequent and unless controlled by adrenalin a trivial shaving cut would bleed for a day. The occurrence of joint effusions and haemorrhages was markedly influenced by season, being much less liable to occur in summer than in winter. VI. 50, Cuthbert G., bleeder, born 1878, had an early history like VI. 49, epistaxis and bruising marking his childhood. His joints became affected later, particularly the elbows. Of late years, however, the tendency to joint effusions has been less. He has, however, suffered more than the others from serious non-articular manifestations, two of the most severe being when he was medical resident in a large hospital. On the first occasion a haemorrhage took place into the substance of the tongue and spread extensively and stopped only when close to the glottis. On the second occasion, in 1906, a molar was extracted, and he nearly lost his life after eight days' haemorrhage, at which time his blood corpuscles were reduced to 2,000,000 per mm.^{3}, and the haemoglobin to 25 %. All kinds of remedies were tried but the only one that was successful was continuous digital pressure kept up by the medical residents and sisters in the hospital for three days. The haemorrhage was not so much from the socket as from the outer edge of the alveolus. During an attempt at plugging the socket his tongue was burnt by a small electric lamp in two places which bled for seven or eight days. VI. 50, has also suffered from haematuria on several occasions. In addition he has had certain illnesses difficult to diagnose exactly but almost certainly the result of his constitutional malady. One of these was in 1894, when he was 16 years of age, at which time he was attacked with stiffness and intense pain in one hip. He was bedridden for weeks when the symptoms gradually disappeared. The symptoms suggested haemorrhage into the ileo-psoas muscle with consequent pressure on the nerves, a suggestion in harmony with the fact that when the pain and stiffness subsided, the limb was found to be partially paralysed. In 1897 an exactly similar attack occurred on the other side with the same result. In 1908 and 1909 he had three attacks of violent pain in the abdomen, accompanied by vomiting and symptoms of intestinal obstruction. The attacks lasted three to five days, the first signs of recovery in each case being the passage of a motion containing free blood. It was considered to be a haemorrhagic effusion into some part of the wall of the bowel. VI. 51, Ralph G., born 1879, was the third bleeder of the family. He did not evince any symptoms until nearly the time of his death, which occurred at the age of 3, from uncontrollable nasal haemorrhage. VI. 52, Muriel G., born 1881, alive and healthy. She menstruates too frequently and the flow may last 12 days. VI. 53, George G., born 1883, perfectly healthy, has never exhibited an abnormal tendency to bleed. VI. 54, Madeline G., born 1884, healthy, menstruates every three weeks but is otherwise normal. VI. 55, Norman G., born 1886, was the seventh child and the fourth bleeder in the family. At 13 months he fell and tore the fraenum of the upper lip and bled to death. VI. 56, three females, born respectively in 1887, 1889, 1890. The first two are alive and healthy, the third died when 3 weeks old. VI. 57, Cedric G., born 1891, a bleeder. He has had many external haemorrhages, and at the age of 19 still suffers from epistaxis and bleeding from the gums. His joint troubles developed early, the elbows, knees, wrists and fingers being the seat of effusions. This state of affairs continues, and recently, while in bed with influenza, he had at the same time effusions in both elbows, both knees, both ankles, and one wrist. The haemophilic symptoms in this family have been very carefully studied by Dr Gettings, who remarks that the joint attacks cannot be distinguished from ordinary synovitis, and they never arise without cause. Attacks are, however, induced much more easily at certain times than at others. In a severe attack the joint becomes swollen, slightly hot and exquisitely painful, the acute stage lasting three or four days, at the end of which time subsidence takes place. On certain occasions there is a tendency for the affected joint to remain swollen for weeks or even months. With regard to the external haemorrhages, the initial bleeding ceases entirely, and it looks as if nothing abnormal were to occur. After some hours, however, it starts again, and then can only be controlled with difficulty. The flow is generally an oozing which wells up continuously from

the seat of injury. VI. 58—92, not affected with haemophilia. Dr Gettings directed particular enquiries into the histories of VI. 93—126 without finding the slightest evidence of haemophilia in any individual. They are all stated by him to have been unaffected. VI. 93, born 1857, died of diphtheria in 1864; he never bled. VI. 94, died at the age of 9 months. VI. 95, died of diphtheria. VI. 96, died of some infantile complaint. VI. 97, three sons, the first, alive and healthy, aged 44, the second died at the age of 40, the third is alive and well. VI. 98, alive and well, aged 36. VI. 100, healthy, alive. VI. 102, died at the age of 2, but never exhibited a tendency to bleed. VI. 103, died at the age of 33 of heart disease. VI. 104, died of heart disease. VI. 105, alive, healthy. VI. 106, died at the age of 1 year but not from bleeding VI. 107—126, all healthy. So far as is known no member of the seventh generation is affected with the disease. (Unpublished.)

Fig. 604. *John Thomson's Case.* We are indebted to Dr John Thomson, of Edinburgh, for the following case, the history of which has been worked out by Dr T. Y. Finlay, house physician in the Royal Hospital for Sick Children, Edinburgh. Several of the affected individuals have been under Dr Thomson in Edinburgh. I. 1, and I. 2, no information. II. 1, and II. 2, said to have bleeders; no actual data. II. 4, Phoebe D., died of "brain softening" at the menopause. She married twice and produced haemophilic stock by each husband. III. 1, Robert M., was a lecturer in chemistry, and died at the age of 72, not a bleeder. III. 3, Ann M., not affected. III. 5, died at menopause of "brain softening." III. 7, died at the age of 53. III. 8, John W., died at the age of 73. III. 10, Edward W., a bleeder, joints affected, died at the age of 30. III. 12, Phoebe W., not a bleeder, married and died of an unknown cause in childbed. IV. 1, alive. IV. 3, a bleeder, died about the age of 21 of morphia habit contracted in soothing the pain of his haemophilic joints. IV. 4, a bleeder and morphinist like his brother, died about 21 years of age. IV. 5—9, healthy. IV. 10, James W., died at the age of 5 of "measles and bleeding." IV. 11—19, not affected. IV. 20, a bleeder, died of haemoptysis at the age of 23. IV. 21, healthy. V. 1, healthy. V. 3, not a bleeder, died at the age of 9 months of atelectasis (?). V. 4, James S., alive, aged 25, a bleeder, no details. V. 5, Archibald, died at the age of 18 of fits and jaundice; not haemophilic. V. 6, died of bleeding in childhood. V. 7—13, not affected. V. 14, William W., aged 13, a bleeder, no details. V. 19, John McD., aged 25, a bleeder, affected with joint troubles. V. 20, alive, aged 23, healthy. V. 21, aged 18, alive and healthy. V. 22, aged 15, alive and healthy. V. 23, aged 11, a bleeder, joints affected. V. 24, aged 6, a bleeder with haemarthroses; mentally deficient. (Unpublished.)

Fig. 605. *Fritz Neumann's Case.* History of a male, III. 12, aged 21, who bled severely after the removal of the left lower first molar which had an abnormal fang complicating the extraction. Short account of other members of the family. I. 1, aged 42, "bled to death. It is not known whether he showed symptoms before this period." I. 2, healthy. II. 1, "died of cerebral haemorrhage while still young." II. 2, died of haematuria at the age of 40: no previous history of liability to haemorrhage. II. 3, aged 48, healthy. II. 5, "nearly bled to death after tooth extraction years ago." II. 6, suffered since her 16th year from menorrhagia and epistaxis: repeated attacks of joint swellings which took weeks to disappear. She was 46 years of age and married to II. 7, a healthy man. III. 1, and III. 2, healthy. III. 3, menstruated freely, and had frequent painful swellings of joints, especially the knees and ankles. The knees were "frequently quite blue." III. 4, "bleeds copiously from the nose"; married to III. 5, a healthy woman. III. 6, and III. 7, died young. III. 8, female, stated by Neumann to have been a bleeder. She had copious menstrual flow and epistaxis, and haemorrhage from most trivial injuries was arrested with difficulty. III. 9, died young. III. 10, III. 11, healthy. III. 12, Neumann's patient, aged 21, had never previouly shown any symptom of haemophilia till 1900, when he consulted Neumann with reference to the superior and inferior first molar teeth, both of which were in an advanced state of decay. The extraction of the lower molar turned out to be a "fairly difficult" operation, for on attempting to dislodge the tooth towards the mouth, the *second* molar began to move with it, so that in order not to displace the latter, and as there was apparently some abnormality in the fangs of the first molar, an attempt was made to force the first molar back against the second molar. In spite, however, of this manœuvre, success was only achieved when the lower wisdom extractor was used. The cause of the difficulty was found to be that the posterior root of the first molar projected backwards under the second (figured in Neumann's paper). The result of the "fairly difficult operation" was the immediate flow of blood which was characterised by its "helle Farbe" (artery?). The socket was plugged, and in an hour the patient was able to go home, but the bleeding started again three hours later, and compression of the carotid was carried out for half an hour. Still later, the bleeding was again severe, the patient becoming unconscious. With the thermo-cautery, however, the bleeding was arrested. He was able to visit the dental surgeon next day, and during the visit the bleeding started again and he collapsed and had to be taken home. Ultimate recovery. The history as here related does not in our opinion justify the diagnosis of haemophilia. The severity of the dental operation and the copious flow of bright blood are more consonant with the diagnosis of rupture of an artery. The alleged haemophilia in the other members of the family is not convincing. At first sight the account of the joint swellings of III. 3 would seem to indicate haemorrhage into the joints. It may be pointed out, however, that in cases

in which haemophilic joints have been rashly opened by operators it has been due to the simulation to tuberculosis in which *tumor albus* is the characteristic not *tumor coeruleus*. We therefore see no evidence of haemophilia in this case. (See Bibl. No. 729.)

Fig. 606. *Hadley and Holland's Case.* Dr W. A. L. Holland published the short history of a bleeder V. 3, who had been a patient in the Queen's and General Hospitals, Birmingham, in 1901. In a private communication from Dr Holland we learn that the family history which is appended to his paper was worked out by Dr E. C. Hadley, of Birmingham, from whom we have obtained the original notes of the case. As the two accounts differ somewhat we have followed that in the original notes by Hadley. I. 1, and I. 2, no data. They had eight children, II. 1—8, all the sons, six in number, having, it is alleged, bled to death; no data. II. 7, a daughter, whose history could not be traced. II. 8, died at the age of 46. She was not affected with the disease. Her husband, II. 9, died five years ago at the age of 85, having never shown any tendency to haemorrhages. II. 8, and II. 9, had 16 children, viz. 13 boys and three girls. III. 1, aged 14, bled to death from a "black gathering under the glands of the ear." III. 2, said to have bled to death from a gathering which formed on his finger. III. 3, bled to death from a gathering on one of his toes. III. 4, died from traumatic bleeding from the lip. III. 5—8, died of infantile diseases before the age of 4. They were not, so far as was known, bleeders. III. 9, and III. 10, died of haemorrhage. No details available. III. 11, one of the sources of information of the family history; she is not a bleeder. III. 12, healthy. III. 13, female, dead, not haemophilic. III. 14, William P., aged 40, a bleeder, known to Dr Hadley when he was a patient in the hospital suffering from uncontrollable epistaxis. III. 15, aged 38, "has never had haemorrhages." III. 16, healthy. IV. 1, 3, 4, dead, cause unknown. IV. 2, male. Now aged 28, "is a bleeder but does not bleed very profusely." IV. 5, female, aged 22, unmarried. IV. 6, girl, aged 20, unmarried. IV. 7, female, mother of Hadley's patient. "She does not bleed but says she has never hurt herself. She says she loses a great deal of blood at her confinements and once had what she describes as 'flodding.'" IV. 8, alive, healthy. IV. 9, boy, bled to death at the age of 3. IV. 10, boy, bled to death at the age of 5. IV. 11, bled to death. IV. 12, James S., died of bleeding 14 months ago in Ward 18, General Hospital, Birmingham. He was known to Dr Hadley. IV. 13—16, three boys and one girl, all free from the disease. V. 1, a bleeder, had twice been in the Queen's Hospital, Birmingham, once, bleeding from the mouth, the second time from a very large haematoma of the thigh, the result of a kick. V. 2, a boy, had not shown the symptoms of the disease. V. 3, Willie R., aged 5, admitted into General Hospital, Birmingham, in 1901, with burns of left shoulder, arm and chest, due to ignition of clothes. He had an effusion of blood into right elbow joint and burns of the second and third degree. There was no bleeding from the burned surface until the sloughs began to separate. Picric acid was used as a dressing and although the bleeding was profuse it was not alarming. On the day of admission his right nostril began to bleed, and as it continued to ooze steadily plugging had to be resorted to. After the removal of the plug the haemorrhage started again and plugging was again carried out. Twelve months previously he had been in the Queen's Hospital with uncontrollable bleeding from the upper lip, and again five months later with haemorrhage from the mouth. Four days before the burn above described he had fallen on his way to school and sustained the above-mentioned effusion into his elbow. His mother said that he always bled profusely from the most trivial wounds such as scratches. V. 4, a boy. Had not yet shown evidence of haemophilia. V. 5, a girl, died of inflammation of the lungs when $7\frac{1}{2}$ months old. V. 6, 7, two miscarriages. (See Bibl. No. 743[a].)

Fig. 607. *H. B. A. Pearson's Case.* Under the title "Notes of a case of haematuria due to the haemorrhagic diathesis" H. B. A. Pearson has shortly described the history of a family in which the condition passed from the paternal grandmother to all her children, viz. three daughters and one son, the latter in turn transmitting it to his son and daughters. We are, however, unable to subscribe to the opinion that any of the cases described are haemophilia. The patient, seen by H. B. A. Pearson himself, and suffering from haematuria, suggests both to the author and to us local disease of the kidney. I. 2, "died suddenly at the age of 28 years from haemorrhage." II. 2, 3, 4, "all died suddenly under the age of 30 years from haemorrhage," no details. II. 5, "died at the age of 60 years from haemorrhage into the brain or meninges after an illness of 24 hours' duration." III. 1, 2, 3, "died young from haemorrhage." No details, sex not stated. III. 4, died at the age of 15 years "from heart disease and haemorrhage." III. 5, "aged 24 years, died suddenly with vomiting of blood." III. 6, "aged 5 months, bled to death." III. 7, H. B. A. Pearson's patient, an unmarried female, 40 years of age, suffering from haematuria. She had not any rash of a petechial or purpuric character, neither had she suffered from epistaxis, haematomata nor swelling of the joints due to haemorrhage. She was first attacked by haematuria at about 30 years of age, and on several subsequent occasions. "The cause of the haemorrhage is evidently associated with some abnormal condition affecting the kidney or its hilum." III. 8, 10, 11, 12, 13, five sisters of III. 7. Of them it is stated that they were living, "all in good health except that one of them in her confinements has *ante-* and *post-partum* haemorrhage." One of the sisters of III. 7 (which sister is not stated) "lost a boy (IV. 1), aged 1 year and 7 months, a girl (IV. 2), aged 2 years and 7 months, and a girl (IV. 3), aged 5 years, from haemorrhage," no details. (See Bibl. No. 777.)

Revised Pedigree.

Fig. 493 bis. *Treves' Case.* On page 313 of this section is found the history of a girl aged six, the subject of great haemorrhage after tooth extraction. She was in the London Hospital in 1885, under the care of Treves, who published the history of her case, together with that of her family, in which haemophilia was alleged to have occurred for several generations. In Treves' publication it is expressly mentioned that the "actual Christian names have been preserved while the surnames are fictitious." We consulted the London Hospital Registrar's records and found the surnames, and some months ago communicated with doctors practising in Saffron Walden in order to obtain further data with reference to this family, members of which were said to have long resided in this district. The results of our enquiries were negative. We were informed that no haemophilic family was known to doctors who had practised in Saffron Walden during the last quarter of a century. In November 1910, Dr Russell Andrews informed us that arrangements had been made to admit into the Maternity Wards of the London Hospital a woman who was alleged to be a bleeder and to come of a bleeder stock. We at once got into communication with the woman, who proved to be the sister of Treves' patient. The mother, IV. 11 (IV. 10, in Pedigree 493), was sent for and brought another daughter, V. 5, and from them the history of the family subsequent to Treves' publication (1885) was obtained. She was also visited at her home, and other members of her family were seen. From the data obtained we were thus enabled to add a large number of individuals, in generation V., born since 1885, and a generation VI. The case is of considerable interest in that female bleeders are said to have occurred as well as males. The results of cross questioning IV. 11, showed that the actual evidence of haemophilia in this family is not very sound, and a search of the London Hospital records of members of this family who have been admitted at various times serves but to confirm this statement. The main source of Treves' information was III. 7, Harriet B., now dead. IV. 11, was her only surviving child. III. 7, alleged that I. 3, II. 3, and II. 4, were bleeders, but in what way was not stated. IV. 11, however, said that it was a tradition in the family that they were affected in this way, and we have marked them as bleeders. Concerning I. 1, and I. 2, there was no information. I. 3, was George B., who married I. 4, Mary ——. II. 3, and II. 4, were stated to have been bleeders. In Treves' paper, III. 1, and III. 3, were figured as bleeders. These were known to IV. 11, who confirmed this opinion, although she added that she did not know of any instance in which bleeding had occurred and she did not think they bled more than III. 5, who was not figured as a bleeder. III. 1, was Daniel B. III. 3, was Stephen B., and III. 5, was Stewart B. They are now dead. III. 7, Harriet B., lived to a very old age and had never manifested any symptoms of haemophilia. III. 8, George G., not a bleeder, and a member of a healthy stock. III. 9, Susan B., still living, over eighty years of age (1910), and has never bled excessively. She is stated by IV. 11 to be now somewhat feeble-minded. III. 10, Charles P., died of old age. He was not haemophilic. III. 11, Sophia B.; III. 12, Charles C.; III. 13, Polly B.; III. 14, James W.; III. 15, Lydia B., were all healthy, although all are now dead. IV. 1, was Frederick B., who was regarded as a bleeder, on the evidence that he was run over by a railway train which crushed both his legs and he bled to death. IV. 2—6, were alleged by IV. 11, to have been unaffected. IV. 7, William B., was stated to be a bleeder, but in what way could not be elicited. He is dead. IV. 9, a healthy female. IV. 10, a girl, lived only a week. IV. 11, Eliza P., mother of Treves' patient and the person who has supplied us with the new information. She is 55 years of age, of fragile appearance, but intelligent if somewhat garrulous. She is much impressed with the excessive degree of tendency to bleed in her family, and her descriptions are coloured with this idea. Where we have had the opportunity of verifying her statements in the London Hospital records we have found, as might perhaps be expected, that her descriptions are not altogether accurate. She herself has always been healthy, and although she has repeatedly cut herself she has never evinced any excessive tendency to haemorrhage. She has never had epistaxis. Her courses began when she was fifteen, and were perfectly natural, both as regards time and quantity. She bore 12 children, including two miscarriages, without any trouble, and passed the climacteric period easily and without any complications, six years ago. IV. 12, Alice G., died young, of a cause unknown. IV. 13, George G., also died young. He never showed a tendency to bleed. IV. 14, George P., first cousin and husband of IV. 11. He is alive, aged 55 (1910), and a turn-cock by occupation. He was figured as a bleeder by Treves, and this is also re-asserted by his wife. On probing this matter it would appear that he has bled severely on three occasions, twice from cuts on the hand, and once he passed blood in the urine. We were unable to get any information with regard to the almost fatal haemorrhage after tooth extraction referred to by Treves. One of the cuts on his hand was inflicted while he was a young man. On the second occasion he was 50 and came to the London Hospital to have his hand dressed in the out-patient department. He is now a healthy man and has never had any of his joints affected. The evidence of haemophilia in his case seems to us most doubtful. IV. 15, Joseph P., is also alleged to be a bleeder, although no data could be obtained which could justify such a diagnosis. He is married. IV. 17, William, also said to have been a bleeder. He died, aged 32. Neither IV. 11,

nor IV. 14, could give any evidence of haemophilia in his case, and the cause of his death was unknown. IV. 18—IV. 23, are stated to be living and healthy. IV. 18, is Eliza P., married to IV. 19, William S., living in London. IV. 20, Sarah P., married IV. 21, V. V., living in Brixton. IV. 22, Lizzie P., married to IV. 23, by name A., living with III. 9, at Saffron Walden. IV. 25, a girl, died young, of an unknown cause. IV. 26, a boy, said to have been a bleeder, but in what way was not known. IV. 11, said that III. 13, "had trouble from bleeding in her eldest boy." IV. 27, and IV. 28, said to be healthy; alive. IV. 29, a female, dead; cause unknown. IV. 30, alive and well. V. 1, Florence P., was the patient described by Treves. She never showed any tendency to bleed till she went to school, about the age of six. It was at this time that her tooth was extracted by a local doctor and she bled copiously. Later she used to suffer from epistaxis and was easily bruised. She began to menstruate at the age of 14, and was regular. Up to the age of 20 she continued to suffer from epistaxis, but not in an excessive degree. About this time she struck herself on the upper lip with a hammer while opening a box. The wound bled freely, but the haemorrhage was easily arrested at Greenwich Hospital. She married at the age of 21. Thirteen months later she was confined, and died three hours later of exhaustion. A fortnight previously she had had a fall, and bled from the vagina, the bleeding lasting up to the onset of labour. During the actual confinement haemorrhage was not severe. Her child, a boy, VI. 1, never showed any signs of the haemorrhagic diathesis up to the time of his death from diarrhoea at the age of 14 weeks. V. 3, Alice P., died at the age of 12 months, from fits. She was not a "bruiser." V. 4, Albert P., aged 28, regarded by his mother as a "terrible bleeder." As a baby he bit his tongue at the back and the bleeding was so severe that his mother brought him to the London Hospital, where he was treated as an in-patient. At eight, he cut his eyebrow on the edge of a table and sustained a cut one inch long. Two local doctors inserted five stitches, but the haemorrhage was so severe that his mother brought him to the London Hospital. We are unable to find any record of this visit in the Hospital registers. There was no bleeding during his first dentition. About 16, he fell over a pole, injured his knee, and was admitted into Shadwell Hospital. Again, a year later, he injured his knee and was admitted into the London Hospital under Mr Warren Tay (24/8/1896, Register No. 2660). The joint, according to the notes, was much swollen and discoloured, and a plaster splint was applied. He was discharged on 14/10/1896, *i.e.* about seven weeks (his mother says *eleven* weeks). He was again admitted on Nov. 12, 1896 (Reg. No. 3547), in order that a gum and chalk splint should be applied in place of the plaster one. He was discharged in six days. His mother alleges that he was again thirteen weeks in the London Hospital before he was 20. We have, however, been unable to find any record of this stay, which was said to be caused by a blow on the eye with a hammer. He has bled greatly from carious teeth, which have been allowed to rot away on account of the alleged danger from extraction. He is not lame, and can walk for miles. He is a strong dock labourer, an occupation which does not seem to us compatible with the idea that he is a genuine bleeder. V. 5, Minnie P., married to V. 6, by name H. So far she has had no children. V. 5, was seen by us in Nov. 1910. She is a somewhat poorly-developed anaemic young woman, 26 years of age, being born in 1884. Both she and her mother assert that she has been a great sufferer from epistaxis and bruises. Her joints have never been affected. At 17 she cut her head with a bottle which fell and burst. A wound was produced three-quarters of an inch long and of considerable depth. The scar is still visible. She was treated in Greenwich Hospital for some weeks, and as great swelling ensued (infection?) she came to the out-patient department of the London Hospital to have it dressed. She began to menstruate at the age of 18, but it was only after she married, when 25 years old, that she lost unusual quantities of blood during her periods. At 20 she cut her hand with a broken bottle, and bled off and on for five weeks. The scar is still visible. In 1909 she cut her left forefinger with a knife and was brought to the out-patient department of the London Hospital, where she was told that she "had cut an artery." A scar three-quarters of an inch long is visible at the site of the wound. Her teeth are much decayed, and her mouth is septic. V. 7, a miscarriage in the fourth month. V. 8, Edith P., aged 23, married to J., V. 9. She is the person concerning whom our inquiries were instituted. She has never bled excessively from accidental injuries or when menstruating. Her teeth are much decayed, but none have been removed for fear of haemorrhage. In her nineteenth year, being then pregnant, she was admitted into the London Hospital (Feb. 1, 1907), for inevitable abortion and severe haemorrhage. For three months she had had amenorrhoea, and two days prior to admission she noticed bleeding from the vagina. While engaged at her work—a sweet-maker—she felt a "pop in her inside" and flooding began, the blood dropping on the floor. She was at once conveyed to the hospital, an examination showing the vagina to be full of clots. Under an anaesthetic the os uteri was dilated and the ovum removed. The after-bleeding was easily stopped by a hot douche, and she left the hospital cured 11 days later. She is again pregnant, and is on the list for admission into the hospital on December 5th. V. 10, Lily P., alive, aged 20; seen Nov. 1910. Her mother alleges that she bit her tongue when four years old, and was brought to the London Hospital out-patient department on account of the haemorrhage. In our second interview, IV. 11, told us that V. 10, received a kick on the knee when she was 14 months old. Swelling occurred, and she was an out-patient at the London Hospital. She now suffers from scoliosis, but is otherwise healthy. Her menstruation is normal. V. 11, Beatrice,

aged 18, perfectly normal. V. 12, Ada, alive, aged 17, seen Nov. 1910. At 8, she bled from a cut on the lip, the result of a fall on the stairs. She began to menstruate at 15, and this has always been copious. She is considered by her mother to be a bleeder. V. 13, a miscarriage in the fourth month. V. 14, William, aged 12, seen Nov. 1910. His mother says he was well until the age of five, when he fell and sustained a cut on the back of his head. The wound was small but deep and bled copiously. "He has never bled since, but is a bad bruiser." At the date of our second interview, when this boy was seen, he had a swelling on his forehead, the result of being pushed by a boy against an iron desk a week before. The mother, who was not present when the accident occurred, alleged nevertheless that "it bled terribly," whereas the patient himself said it bled for "about five minutes," the bleeding being arrested by cold water. No bruises were found upon him. If while playing football he is kicked on the shin nothing unusual happens, but knocks are apt to produce swellings which may burst and discharge dark red fluid. This boy is probably affected in a very slight degree with a tendency to the production of haematomata, but there is no evidence that he is a genuine bleeder. V. 15, George, aged nine, alive and healthy. V. 17, and V. 18, four females and three males, stated by IV. 11, to be healthy. V. 19, seven healthy children, sex not known. V. 20, and V. 21, both unaffected. Our recent inquiries into the history of this family would lead us to doubt the diagnosis of haemophilia. It may be that some of these individuals, especially the females, show an unusual tendency to epistaxis and menorrhagia, but there is no certain case of haemophilia, at any rate in the last four generations.

ADDENDA TO BIBLIOGRAPHY OF HAEMOPHILIA.

912. Elam, Charles: *A Physician's Problems.* London, 1869, p. 28. [Trivial reference to a man and his son, both of whom had haemoptysis at the age of 21, but lived to the age of 70. The family of the son "have each one as they approached that period of life been affected either in the same way or one equivalent."]

913. Weber, Albrecht: Fall von successiver Erblindung beider Augen durch extraoculare und intraoculare Blutungen bei Haemophilie. *Archiv für Ophthalmologie.* Leipzig, 1897, Bd. xliv. S. 214. [Male, aged 21. After striking his face against a door he bled from the mouth and nose, and into the right eye. He was seen 11 years later, suffering from a haemorrhage into his left eye, this resulting in total phthisis bulbi from exulceration of the cornea. He was stated to have bled very profusely after wounds received while fighting duels. There was no evidence of haemophilia, however, in his family.]

914. Kellie, Kenneth: Haemophilia, with adhesions in the knee joint. *Reports of the Society for the Study of Disease in Children.* London, 1908, Vol. viii. p. 262. [Short account of a male, aged 13. He was well until 3, when haemorrhage from a cut finger was found to be difficult to arrest. At 5 his right knee became swollen, and later his left elbow. Since then he has had many recurrences of joint trouble, and on one occasion bled severely after tooth extraction. His parents were healthy, and there was no history of bleeding among the collaterals of the family. He was one of nine children, five of whom died in infancy. Of the other three the eldest son, aged 30, "has always suffered from bleeding"; a daughter (married) and a son are healthy.]

915. Holsti, H.: Hämofili med ledgångsaffectioner. *Finska Läkaresällskapets Handlingar.* Helsingfors, 1909, Bd. li. Heft 5, S. 840. [Male bleeder, aged 19, a farmer's son from Soini (Finland). He had bruises and bled easily from slight wounds and after tooth extraction. On one occasion he had a haematoma on the chest wall and extending from the fourth rib over the clavicle to the lower part of the neck. Haematuria on several occasions. His principal trouble had, however, been with his joints, which would suddenly swell up and become painful. A diagnosis of tuberculosis was made, but a diagnostic dose of tuberculin was negative. Massage produced subcutaneous haemorrhage. His parents were alive and healthy, and had shown no tendency to bleed. Four brothers were healthy, one died from scarlet fever. Three sisters were well. A brother of the mother bled easily and suffered from swollen joints. He died at the age of 20, of a cause unknown.]

916. Hubbard, Thomas: Hemophilia with remarks on the hemorrhagic diathesis. *Transactions of the Thirty-first Annual Meeting of the American Laryngological Association.* New York, 1909, p. 238. [History of a boy, aged 4, who bled profusely after the removal of tonsils and adenoids. His parents were healthy, the patients being the youngest of three children. One sister had exophthalmic goitre. The father had severe epistaxis when about 4 or 5 years old. The maternal grandfather was called a "bleeder," the evidence in his case being severe haemorrhage from a tooth socket and an attack of haemoptysis from which he died. The patient himself had "several suggestive but not severe haemorrhages from an injury to the fraenum of the upper lip when an infant." Otherwise, he had not bled.]

917. Labbé, Marcel : Un cas d'hémophilie sporadique. *La clinique.* Paris, 1909, Tome iv. p. 837, [History of a male wood carver, aged 33, in the Hôpital St Antoine, Paris. In infancy repeated attacks of epistaxis and prolonged haemorrhage after cuts. At 10 he fell and developed a swelling of the knee. Leeches were applied and considerable haemorrhage ensued. In the following year the socket of a tooth which had been extracted required plugging. Later he had repeated bleedings from a tooth stump. On admission into the hospital he had ecchymoses over the knuckles, left elbow and lower limbs. Ten days before his left knee swelled. He was also suffering from gonorrhoea. There was no history of haemophilia in his family, His father was alive; the mother died of pneumonia at the age of 33, and a brother had died of tuberculous meningitis at the age of 37.]

918. Matsuoka, M. : Ueber die Haemophilia spontanea. *Deutsche Zeitschrift für Chirurgie.* Leipzig, 1909, Bd. cii. S. 364. [Two cases in the orthopaedic clinic in Kioto: (1) male, aged 16; no history of haemophilia in the family. Parents and four sibs healthy, although according to the mother her second son had a tendency to bleed. Patient bled from the gums when young, and he was also the subject of suggillations. After his 8th year, elbows, hands, knees, and ankles were repeatedly affected. Aspiration of his right elbow and both knees revealed the presence of discoloured blood; (2) male, aged 25; affected with a tendency to bleed from childhood; swelling of joints. No family history of haemophilia.]

919. Nolf, P. et Herry, A. : De l'hémophilie, pathogénie et traitement. *Revue de médecine.* Paris, 1909, Tome xxix. p. 841 and 1910, Tome xxx. pp. 19, 106. [Exhaustive account of experiments on the blood of three alleged bleeders. Influence of serum and solution of Witte's peptone on the coagulation time. The three cases were as follows : (1) male, aged 13, who at the age of 3 developed enteric fever. Afterwards he showed a tendency to rebellious haemorrhages, including epistaxis. He also suffered from bruises and haemarthroses of two finger joints; (2) male, aged 12, brother of preceding. Had suffered from epistaxis, bruises and haemarthroses of hip. The parents of these two boys were not haemophilic nor could any history of the disease be found in their ascendants. The mother had several healthy brothers, and her sister had numerous boys, none of whom were affected; (3) male, aged 19, who bled copiously after small cuts, and had haemarthrosis of knee and ankle. A brother died after repeated haemorrhage at the age of 27. Nolf and Herry treated nine cases of haemorrhage, two of which were "probably haemophilic"; (4) male, aged 20, who had bled after tooth extraction; and (5), his sister, aged 8, who bled from the nose almost daily.]

920. Shaefer : Hématome provoqué par une injection anaesthésique locale chez un hémophile *La revue de stomatologie.* Paris, 1909, Tome xvi. p. 462. [Male, aged 30; haematoma of the gum, following a submucous injection of novocain and adrenalin. Injection of normal horse serum; recovery. The patient had always been liable to bruising, and bled repeatedly from the mouth. Melaena at the age of 12; haematuria three years before; frequent swellings of knees and ankles, leaving a certain amount of lameness. One brother died, at the age of 7, after an incision into a swelling which was probably a haematoma. Another brother died as the result of an accident. The mother had *post partum* bleeding, and her mother had a liability to cutaneous haemorrhages.]

921. de Teyssier : La gélatine administrée par la bouche dans le mélaena du nouveau-né. *Lyon médical.* Lyon, 1909, Tome cxiii. p. 1073. [Child bleeding from the bowel on the seventh day. Administration of gelatine by the mouth.]

922. Weil, P. Émile et Boyé, G. : Hémophilies expérimentales et comparées. *Bulletins et mémoires de la Société médicale des hôpitaux de Paris.* Paris, 1909, 3 S. Tome xxviii. p. 727, also *La tribune médicale.* Paris, 1909, Tome xli. p. 789. [Reference to the existence of haemorrhagic conditions resembling haemophilia in the horse, dog and ox. Experiments to show that after injection of leech extract or peptone it is possible to reproduce some of the symptoms of haemophilia. The paper in general supports the theory that haemophilia is essentially due to some abnormality in the blood itself.]

923. Weil, P. Émile, et Boyé, G. : Action des extraits d'organes sur le sang des hémophiles. *Comptes rendus des séances et mémoires de la Société de Biologie.* Paris, 1909, Tome lxvii. p. 454. [Short paper with reference to the treatment of cases of haemophilia with extracts of the various organs of sheep, pigs, oxen, and horses.]

924. Addis, Thomas : Hereditary haemophilia: deficiency in the coagulability of the blood the only immediate cause of the condition. *The Quarterly Journal of Medicine.* Oxford, 1910, Vol. iv. p. 14. [Account of experiments supporting the view that deficiency in the blood coagulation is the principal factor in the pathogenesis of haemophilia. The author's experiments were carried

out on twelve patients, four belonging to the families described by Groves (Pedigree Nos. 500, 502), and four members of the Mampel family (Pedigree No. 389). Three other cases are referred to with meagre data of symptoms. Case I. was a male who when young suffered from all varieties of haemorrhage, for which he was admitted into the hospital 27 times. For a number of years, however, he had been entirely free of symptoms except great haemorrhage after the removal of a tooth. "He had two first cousins on his mother's side of the family who were haemophilics."]

925. Arnsperger, H.: Die Behandlung der hämorrhagischen Diathesen, *Deutsche med. Wochenschrift*, 1910, Bd. xxxvi. S. 1113 [as in title, short account of treatment of haemophilia].

926. Boyé, Georges: Contribution à l'étude de la pathogénie de l'hémophilie et de quelques états hémorragipares (hémophilie expérimentale et comparée). *Thèse de Paris*, 1910, pp. 164. [Long account of the coagulation of the blood, with special reference to the conditions in haemophilia. Experiments showing retardation of coagulation by extract of leech and solution of peptone. The *rôle* played by the organs and tissues in inducing coagulation is also considered. Four cases are referred to: (1) an extremely obese male, aged 38, suffering from haematemesis and cerebral symptoms. His father had epistaxis; (2) female, aged 57, with haematemesis and melaena and a past history of bleedings. Her father and one of her grandsons had epistaxis and bled easily; (3) female, aged 24, with history of epistaxis, ecchymosis, haemorrhages and pains in the joints. Various neurotic symptoms. Appendicitis, operation, recovery. Her father and her half-brother had epistaxis and bled easily; (4) female, aged 26, bleeding from a tooth socket. History of haemorrhages after abortions and teeth extraction. One of her three sisters menstruated freely and had a haemorrhage during an attack of enteric fever. A brother has a goitre and "is haemophilic."]

927. Bulloch, W.: Female haemophiliacs and *de novo* cases of haemophilia. *The Lancet*. London, 1910, Vol. i. p. 1300. [Reply to Osler on the alleged existence of female haemophiliacs see Bibl. No. 938.]

928. Chavasse: Au sujet de l'hémophilie. *Bulletins et mémoires de la Société de Chirurgie de Paris*, 1910, n. s. Tome xxxvi. p. 518. [Case of a male, aged 47, observed in Algiers some years previously. Phlegmon of leg, incision 5 cm. long. Considerable haemorrhage. Patient had an alcoholic history.]

929. Cruet, Pierre: Hémophilie articulaire. *La médecine des accidents du travail*. Paris, 1910, Tome viii. p. 75. [Good general account of the various joint manifestations observed in haemophilia; no original cases cited.]

930. * Ficacci, L.: Sulla artropatia emofiliaca. *Bulletino della reale Accademia medica di Roma*. Roma, 1910, Tomo xxxvi. p. 12: 1 pl. [not seen].

931. Fontoynont: L'hémophilie acquise d'origine paludéenne au point de vue chirurgical. *Bulletins et mémoires de la Société de Chirurgie de Paris*, 1910, n. s. Tome xxxvi. p. 431. [The author considers that in countries like Madagascar, malaria leads to a fragility of the red blood corpuscles, which may determine severe haemorrhages in the course of surgical operations. Several cases of such haemorrhages are briefly referred to in children, the subjects of calculi.]

932. Guillot: À propos de trois cas d'hémophilie. (Rapport par M. Walther.) *Bulletins et mémoires de la Société de Chirurgie de Paris*, 1910, n. s. Tome xxxvi. p. 409. [(1) Medical student who had previously been well, received a dissection wound which healed slowly and was followed by tuberculosis of a metacarpal-phalangeal joint. Puncture; recovery. Some months later a peri-anal abscess burst and left a blind external fistula. Later, gangrenous phlegmon of the right ischio-rectal fossa. Incision; sanguinolent oozing for weeks. Subsequently, epistaxis and haemorrhage from gums and bowels were superadded and the patient died. (2) Girl, aged 18, with club foot. Extensive operation for the relief of the deformity. Considerable haemorrhage lasting for days. She had always menstruated freely. No history of haemophilia in her family. (3) Male, aged 18, with R. inguinal hernia and varicocele on both sides. Radical cure for rupture. Extirpation of the dilated veins. Haemorrhage and haematoma. Recovery. The patient alleged that for a long time he had bled considerably from minute wounds. M. Walther, who reported Guillot's cases, also included one of his own, a male who bled severely after incision into an occipital haematoma which had been induced by a very violent blow on the head. Three times he had bled profoundly after tooth extractions. He averred that his mother had menorrhagia "durant quinze et vingt jours (?)" [sic] and his maternal grandfather had repeated haemorrhages, being on one occasion three months in a hospital with a cut on his thumb.]

933. Kottmann, K. und Lidsky, A.: Beitrag zur Hämophilie mit spezieller Berücksichtigung der Gerinnungsverhältnisse des Blutes an Hand von Gerinnungskurven. *Münchener med. Wochenschrift.* München, 1910, Bd. lvii. S. 13. [Graphic representation of the course of the coagulation of haemophilic blood, as determined by the use of a coaguloviscosimeter. The great retardation of the coagulation time can be shortened by the addition of thrombokinase obtained from liver, or by serum. Calcium chloride does not appreciably accelerate the coagulation time of haemophilic blood. The serum used to accelerate the coagulation time in these experiments was obtained from the unaffected sister of a bleeder, she herself being the mother of bleeders. The mother and three sisters of the bleeder had normal coagulation periods.]

934. Krauss, H.: Zur Therapie der Hämophilie. *Münchener medizinische Wochenschrift.* München, 1910, Bd. lvii. S. 2421. [Short reference to two brothers the subjects of a tendency to bleed; treatment by serum.]

935. Larned, Charles W.: Hemophilia, with the report of a case of typhoid fever in a hemophilic subject. *The American Journal of the Medical Sciences.* Philad. and New York, 1910, Vol. cxxxix. p. 363. [Male, aged 40, who from childhood until the age of 15 had frequent attacks of epistaxis. Wounds bled freely. When over 30 he nearly bled to death after an operation for ingrowing toe nail. At the age of 37 he had an attack diagnosed as "Henoch's purpura." At 40 he passed through a typhoid infection without complications. He has never had haematuria or haemarthroses. Patient's father "gives a haemophilic history from boyhood until the age of 23 years, after which he seems to have outgrown the tendency. One paternal uncle is similarly affected. Two paternal uncles and one paternal aunt are not bleeders. The paternal grandmother was a haemophiliac and died during confinement, aged 39 years. The father's maternal aunt was not a bleeder, but several of her children were. The patient has one brother who is not a bleeder and one sister who was a bleeder and died at the age of 8 years, but not of haemorrhage. Two brothers have a history of repeated and excessive bleeding from the lips during boyhood. They are now 34 and 38 years respectively and seem to have outgrown the tendency. Several of the patient's first cousins are bleeders, although their histories are somewhat indefinite."]

936. Legueu, F.: Au sujet de l'hémophilie musculaire. *Bulletins et mémoires de la Société de Chirurgie de Paris,* 1910, n. s. Tome xxxvi. p. 434. [Male, aged 27, admitted into the hôp. Laennec with great pain in the lower part of his abdomen associated with flexion of the thigh. He had been seized acutely while at work and his thigh was almost immediately flexed on his abdomen. Examination revealed a swelling in the region of the psoas muscle. After three weeks' rest in bed he was able to go home, but a few days later he was again seized with terrific pain in the same region as before, and was re-admitted into the hospital. An enormous swelling in the R. iliac fossa was found, and under the idea that it was an abscess a puncture was made into it. Clots of black blood in a decomposing state came away. Next day the swelling being as large as ever, an incision 20 cm. long was made and exposed a huge haematoma in the muscles. Clots were evacuated and bleeding was severe. Patient nearly died. A sequestrum of clot and muscular tissue came away and gradual recovery set in. Six months later a similar attack occurred in the L. lumbar region but was treated by rest in bed. Recovery.]

937. Michon: *Bulletins et mémoires de la Société de Chirurgie de Paris,* 1910, n. s. Tome xxxvi. p. 442. [In the discussion on Guillot's paper (Bibl. No. 932), Michon reported the case of a male aged 29 who had slightly wounded his hand with the point of a scissors. A haematoma developed and it subsequently became infected. Injection of serum, and incision which was followed by considerable oozing of blood. Recovery. At the age of 13, he had had haemarthrosis of the knee. An incision had been made and the wound bled off and on for four months. He also had several attacks of haematuria. No family history of haemophilia.]

938. Osler, William: Female haemophiliacs and *de novo* cases of haemophilia. *The Lancet.* London, 1910, Vol. i. p. 1226. [Two cases referred to, (1) a girl aged 19, suffering from epistaxis and purpura. She had always bled freely after tooth extraction and her shoulder joints had been swollen. Her last illness began with epistaxis, bleeding from the gums and under the skin, and, in spite of remedies, she died. She was one of three children, of whom one, a male, aged 12, had always bled excessively when cut. He also had epistaxis. The father had also suffered from epistaxis, as also one of his brothers, a son of the latter being similarly affected. There were no bleeders on the maternal side; (2) a male bleeder, who had been affected from infancy. He had had epistaxis and on three occasions nearly died after accidents. On three other occasions he had severe abdominal pain, associated with vomiting and melaena. Once he bled for 12 days after the removal of a tooth. Joint swellings had been frequent. A brother was similarly affected.

It was not possible to obtain a history of haemophilia in the ascent. The mother of the two boys died of tuberculosis; none of her brothers were bleeders; maternal grandmother and grandfather were not known, although the patient's father had never heard of any bleeders either in his wife's family or his own.]

939. Plumier, L. L.: Un nouveau traitement de l'hémophilie. *Le Scalpel et Liége médical.* Liége, 1910, Année lxii. p. 727. [General account of Nolf's researches on coagulation of blood and his recommendation of the use of peptone solutions in the treatment of haemophilia and other haemorrhagic states.]

940. Ricard: *Bulletins et mémoires de la Société de Chirurgie de Paris*, 1910, n. s. Tome xxxvi. p. 441. [In the discussion on Guillot's paper (Bibl. No. 932) Ricard communicated the following: Female, aged 22 or 23; operation for anal fistula; grave haemorrhage. A year later, the removal of her appendix was followed by the development of a discoloured swelling which on being opened gave exit to clots of blood, and free bleeding. Injection of serum; recovery.]

941. Sahli, H.: Weitere Beiträge zur Lehre von der Hämophilie. *Deutsches Archiv für klinische Medizin.* Leipzig, 1910, Bd. xcix. S. 519. [Important experimental studies on the blood of three male bleeders belonging to one of the families previously described by Sahli (Pedigree No. 378). The corrections in generations IV. and V., to which we have drawn attention (p. 264), are inserted in this paper, and the name of IV. 10 is given as Frau Zaugg-Ruch, her two haemophilic boys, V. 10, and V. 11, being named Hans and Fritz respectively. In Sahli's new paper the three bleeders particularly described are Hans Ruch[1], IV. 3; Fritz Ruch, IV. 45, and Ernst Ruch, IV. 46. Sahli says that IV. 45, and IV. 46, were brothers of IV. 3, but from the fact that they are stated to be the sons of Barbara Ruch-Loosli, III. 12, it is evident that they were cousins. With reference to IV. 3, the information in addition to what we have given (p. 264) was that he had nearly bled to death after tooth extraction and had had an acute exacerbation of the swellings of the knee. He had also a peculiar trouble in his R. iliac fossa, associated with vomiting and constipation. Fritz Ruch, IV. 45, was aged 12, and lived in Hauswyl. He had a long history of severe haemorrhages following trivial injuries, and two years before, his knee became partially anchylosed as result of a kick from a horse. He had never shown epistaxis or haematuria. His brother Ernst, IV. 46, had likewise a typical haemophilic history, including ecchymoses, haematomata, epistaxis, haematuria and haemarthroses. Some months before, his right arm had been injured by a horse and great swelling, with paralysis of the ulnar nerve, ensued. In Sahli's clinic a swelling of the left knee developed without any apparent trauma, and he also had repeated epistaxis, and a haemorrhagic infiltration in the tip of his tongue.]

942. Shinshi, B.: On the knowledge of haemophilia and a report on a case with internal haemorrhages. *The Sei-i-Kwai Medical Journal.* Tokyo, 1910, Vol. xxix. p. 501. [History of a Japanese conscript, aged 22. He was always delicate, and suffered from palpitations and dyspnoea. At the age of 15 years he voided about 500 grms. of blood by the bowel, and this was repeated four or five times a year. He also had pains in the joints, epistaxis and haemorrhages from the gums. At ten years, a great haemorrhage followed an extraction of a tooth. When seen by Shinshi he was anaemic and had haemorrhagic spots on the gums. The usual symptoms of dyspnoea and palpitation were present, and he voided about 800 grms. of blood by the bowel, but recovered. His parents and seven brothers and sisters were all alive except a sister, who died of an unknown disease when one year old.]

943. Squire, E. W.: Haemophilia in a female. *British Medical Journal.* London, 1910, Vol. i. p. 1168. [Child who at birth showed a brown patch over the left eye. Twelve months later, an attack of bronchitis with melaena. Some months afterwards haemorrhage, lasting three or four days, from a scratch on the finger. A year later bleeding from the mouth on two occasions; death from bleeding, the child being then about 3½ years old. No history of swollen joints, scurvy or purpura. A brother of the patient showed no tendency to haemorrhage, and no family history of such a tendency could be obtained on going back two generations beyond the parents.]

944. Stainer, Edward: The hereditary transmission of defects in man. Oxford, 1910, pp. 95. [General account as in title. On page 53 brief account of three cases of alleged haemophilia in a family. (1) a male, "had epistaxis often"; (2) his daughter, "had profuse menstruation commonly. Haemorrhage after having a tooth pulled out"; (3) one of his granddaughters, aged 22, "had epistaxis now and again." Menstruation profuse. Bruised easily and had bled long after two

[1] In Fig. 378, p. 264 (Sahli I.), IV. 3, called Karl Ruch, should be Hans Ruch.

cuts. Double ovariotomy for two cysts which were found filled with soft sticky blood clot and altered blood. No special haemorrhage at operation, but later it was severe. Purpuric spots. Recovery.]

945. v. Stubenrauch. Kniegelenk eines Bluters. *Deutsche med. Wochenschrift,* 1910, Bd. xxxvi. S. 2074. [Short account of the lesions of a male bleeder, aged 26 years. No history of haemophilia in his family. He was seized with pain in the thigh and following this a fluctuating swelling in the region of the psoas muscle made its appearance. Incision, haemorrhage, sepsis, death. The autopsy revealed in addition haemarthrosis of the left knee with rusty discoloration of the synovial membrane, and superficial destruction of the cartilage covering the condyles of the femur.]

946. Tecqmenne, Ch. : Arthropathies d'origine hémophilique. *Le Scalpel et Liége médical.* 1910, Année lxii. p. 425. [Male, aged 8, suddenly seized with pain in the knee, which was swollen. The mother alleged that he had frequently had epistaxis, and profuse haemorrhages from wounds. He had also bled from the gums. Swelling of the hip and knees, together with ecchymoses, had occurred. "Il existe d'ailleurs des antécédents familiaux," but they are not given.]

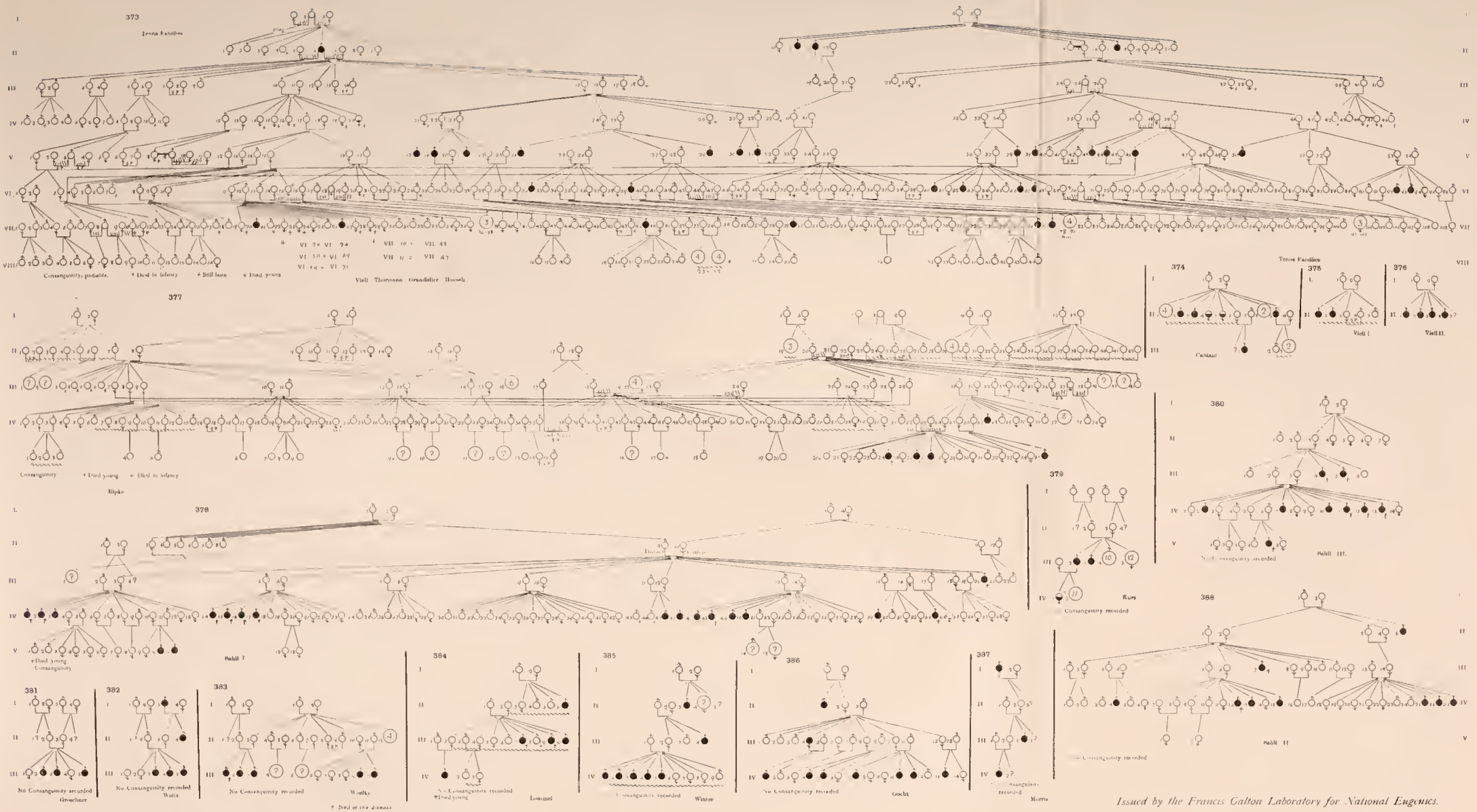
947. Towne, G. S. : Report of a case of hemophilia. *Albany Medical Annals.* Albany, 1910, Vol. xxxi. p. 321. [Three cases referred to : (1) male, aged 24, operated on, for a left-sided indirect inguinal hernia. Great haemorrhage during and subsequent to the operation. Inquiry into his history revealed the fact that every cut, scratch or abrasion had always bled profusely, and this trait had also been present in his maternal grandmother. None of his other ancestors were known to be affected ; (2) fatal haemorrhage during the course of enteric fever ; no details. "In this case the family history was very striking in its hereditary transmission from one generation to another"; (3) bleeding in a female during pregnancy ; no details.]

948. Umbreit : Glossitis haemorrhagica bei Hämophilie. *Deutsche Zeitschrift für Chirurgie.* Leipzig, 1910, Bd. cv. S. 608. [Case of a male, aged 30. No history of haemophilia in the family. On several occasions he nearly bled to death after tooth extraction, and once he sustained a grave haemorrhage from a small wound in the forearm. Admitted into the hospital Friedrichshain (Berlin) with a large extravasation of blood into the substance of the tongue. Incision, recovery.]

949. Welch, J. E. : Normal human blood serum as a curative agent in haemophilia neonatorum. *American Journal of the Medical Sciences.* Philad. and New York, 1910, Vol. cxxxix. p. 800. [As in title, treatment of cases.]

NAME INDEX TO ADDITIONS TO BIBLIOGRAPHY.

Note to p. 348. Pedigree 493 bis. V. 8, has recently (14/xii/'10) been confined of a healthy male child ; the labour was quite uncomplicated and in particular there was no abnormal bleeding. This confirms the view taken above of the very doubtful nature of the evidence on which haemophilia is occasionally asserted of women.



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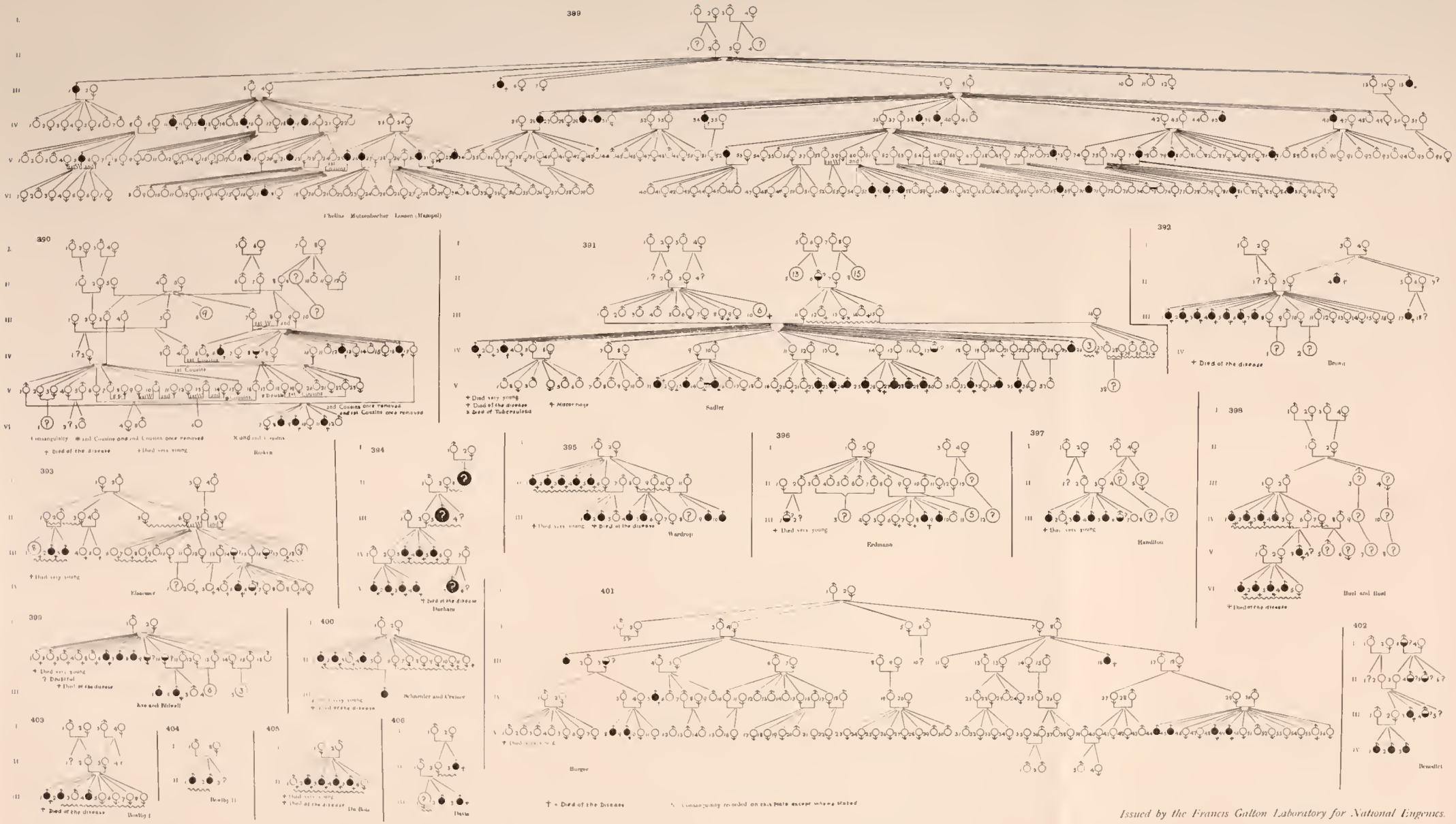
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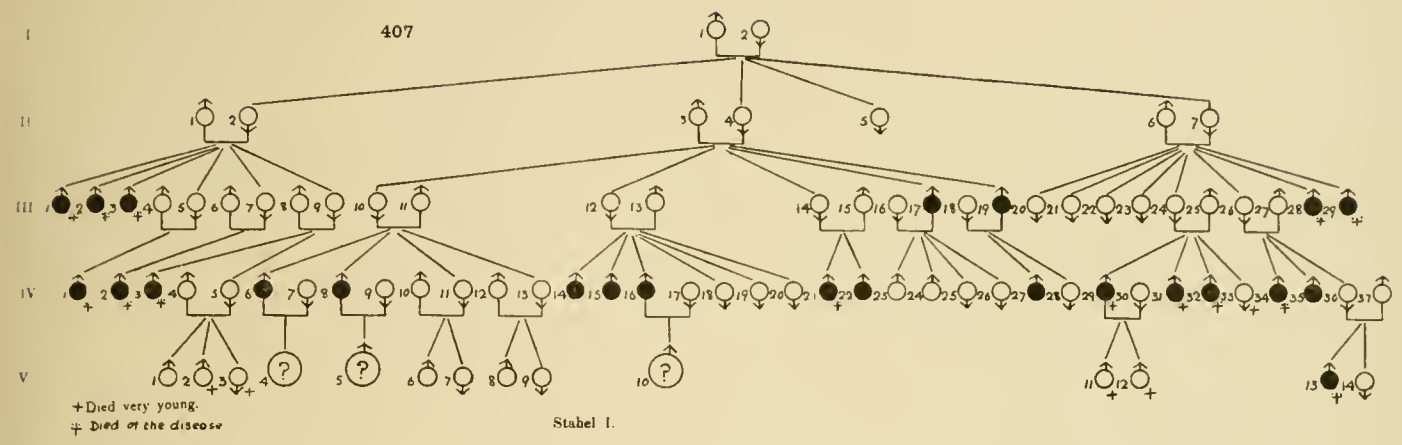

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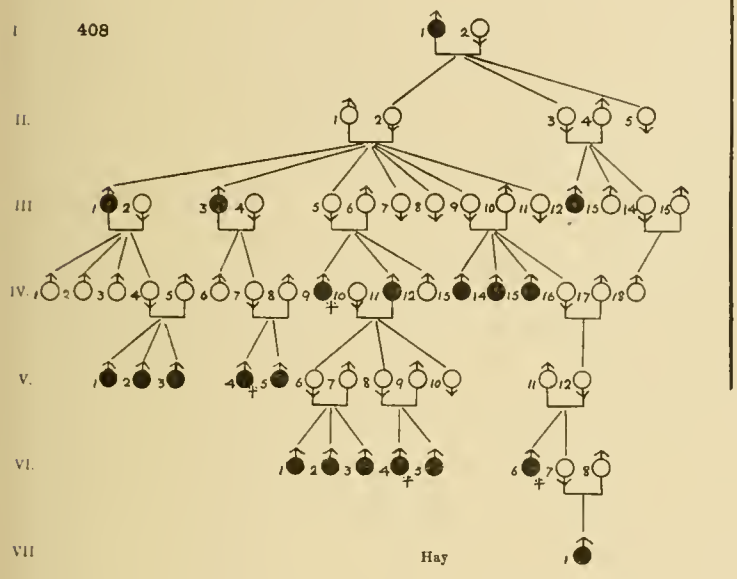

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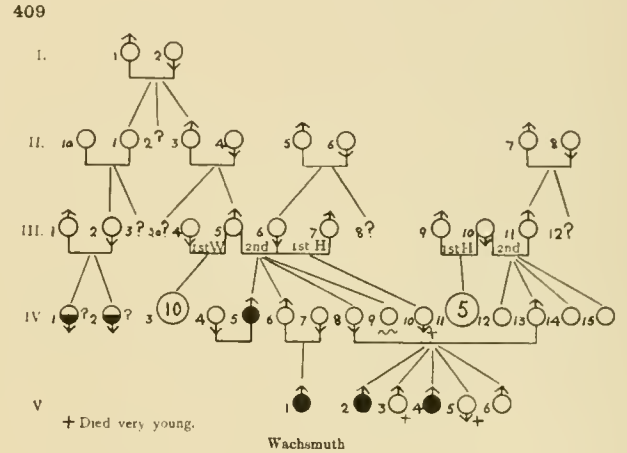

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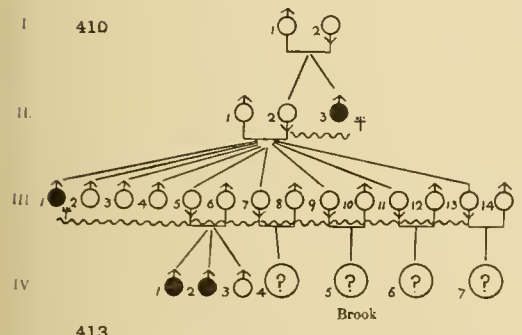

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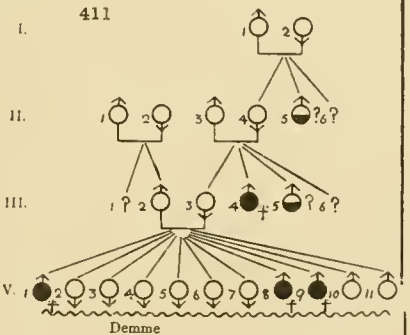

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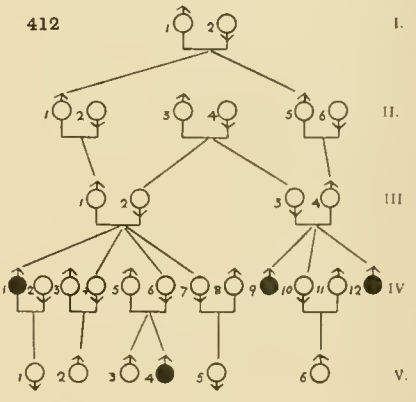

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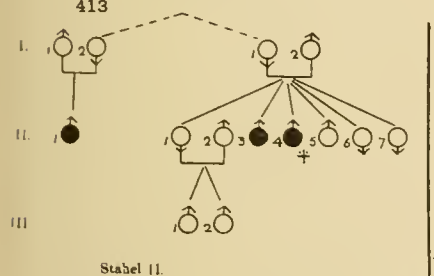

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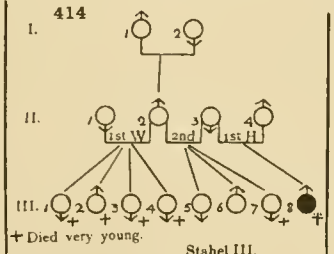

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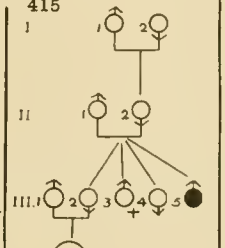

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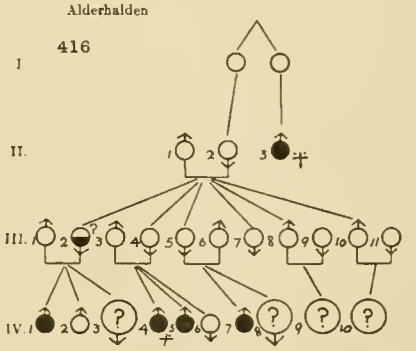
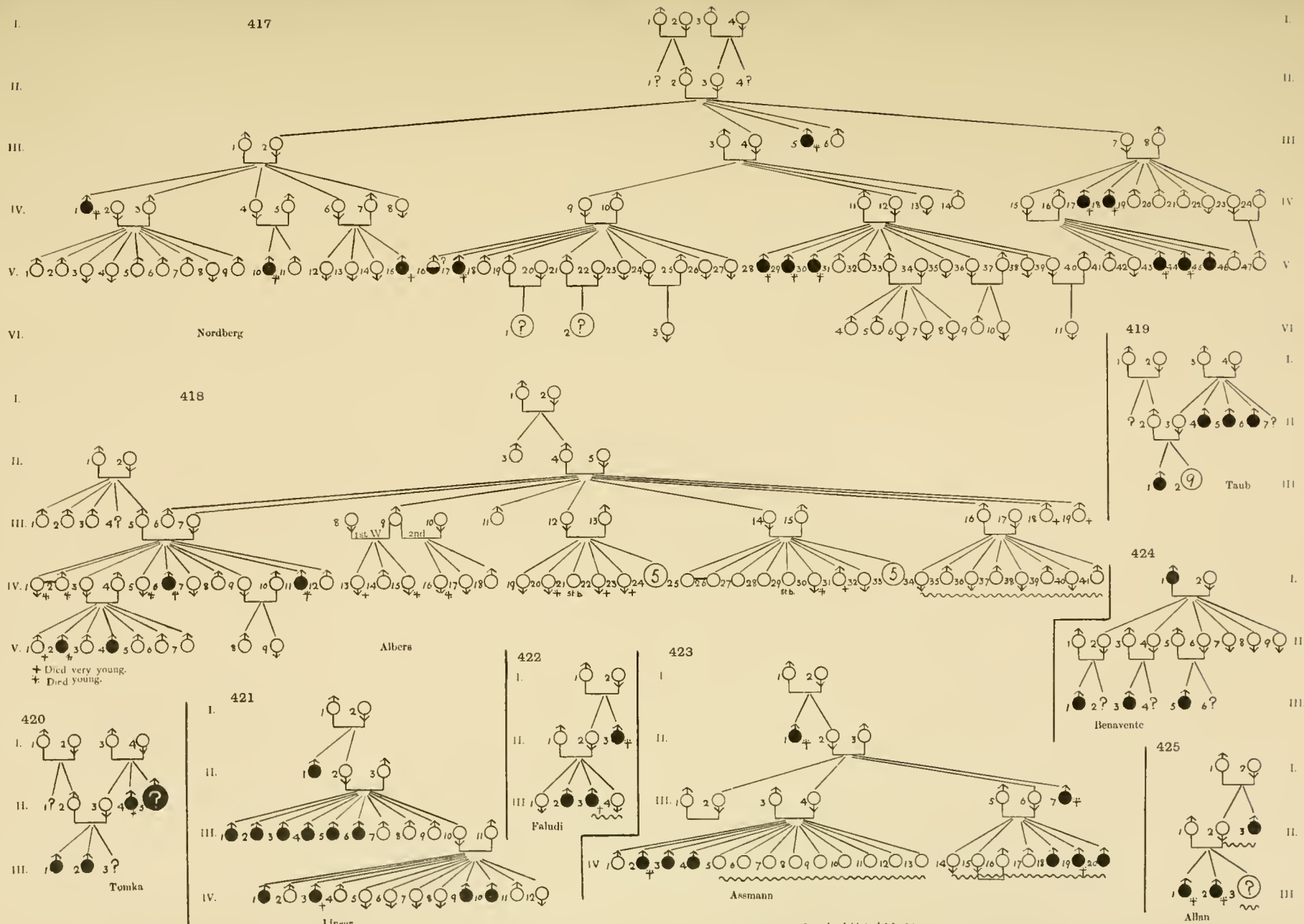


H. Fischer



+ Died very young.
+ Died young.

No Consanguinity recorded in the above Pedigrees

† = Died of the disease

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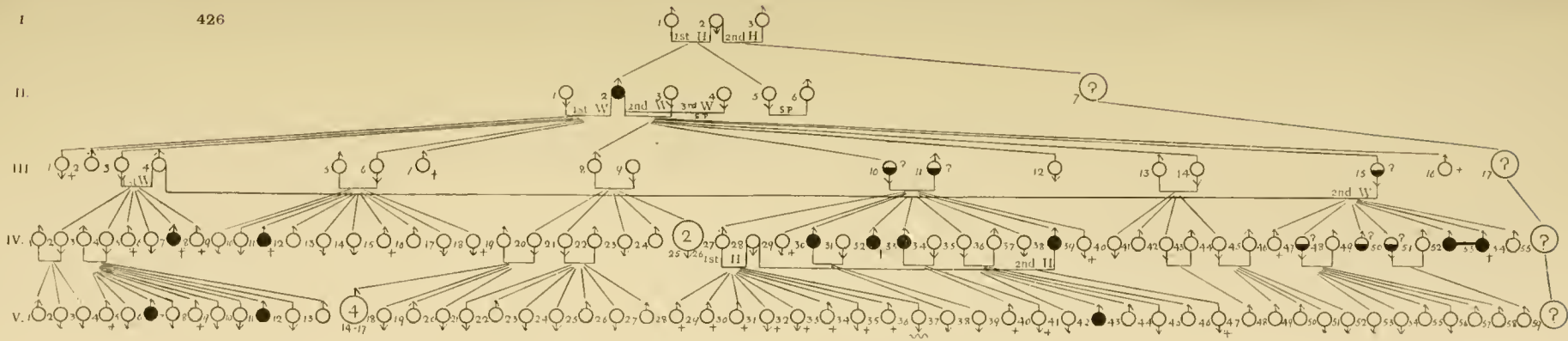
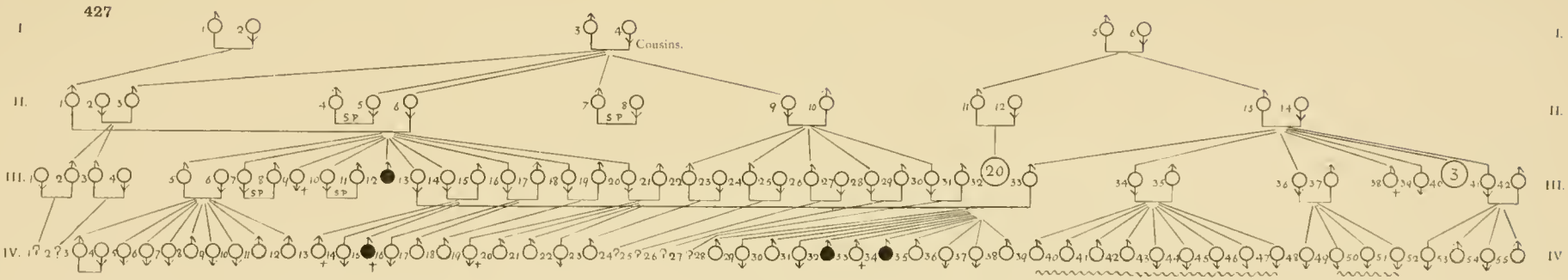
426



† Died very young † Died in infancy * Still-born.
No Consanguinity recorded.

Max Fischer

427

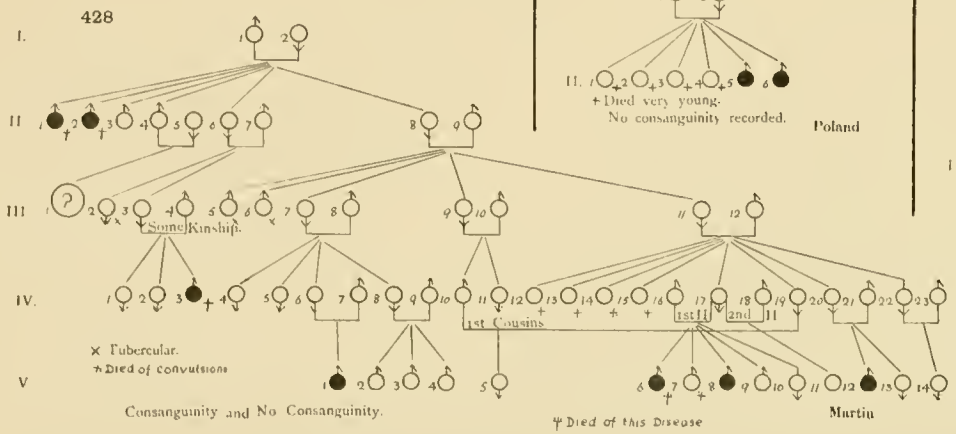


† Died very young. † Died in infancy † Still-born.

Cousins.

Fildes

428



× Tubercular.
* Died of convulsions

Consanguinity and No Consanguinity.

† Died of this Disease

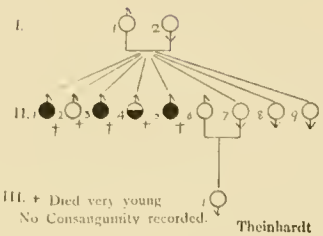
Murtin

429

† Died very young.
No consanguinity recorded.

Poland

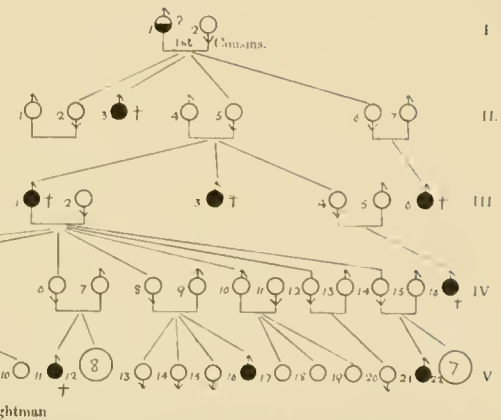
430



† Died very young
No Consanguinity recorded.

Theinhardt

431



1st Cousins.

Consanguinity

Wightman

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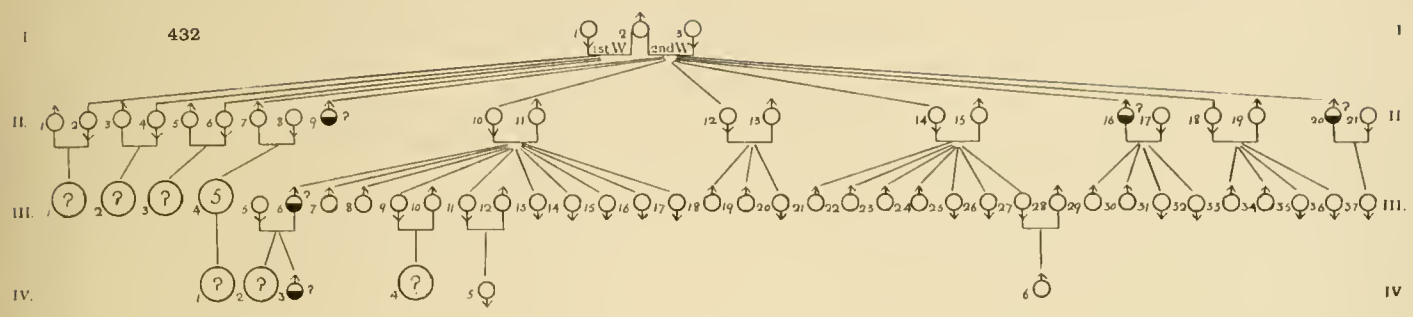
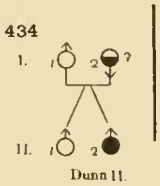
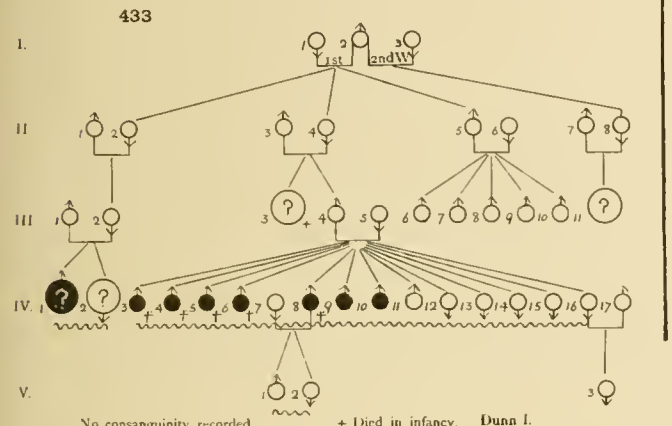
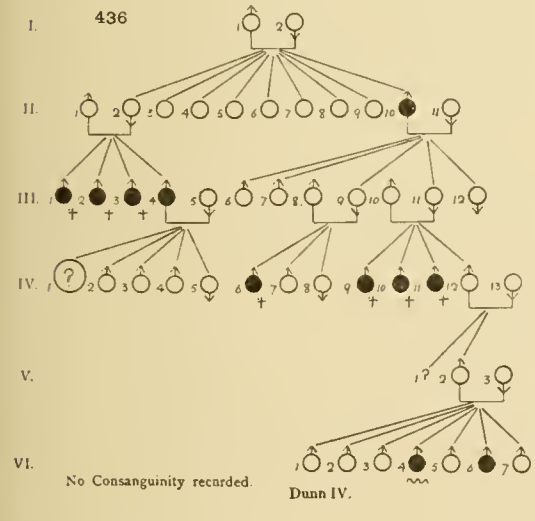
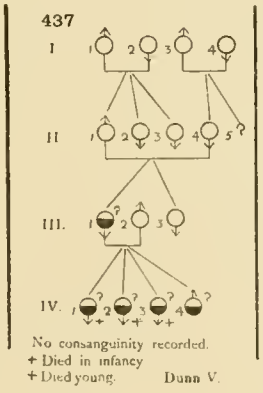
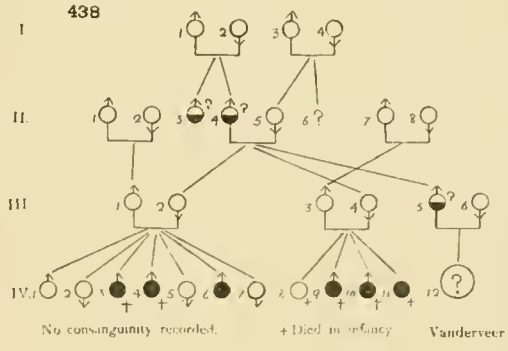


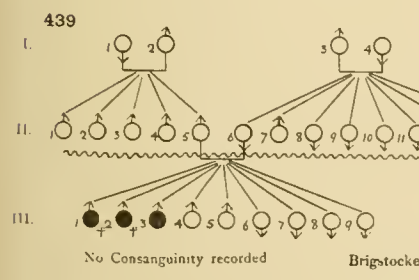


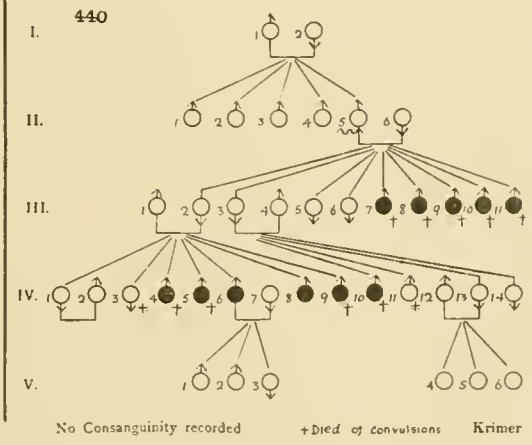


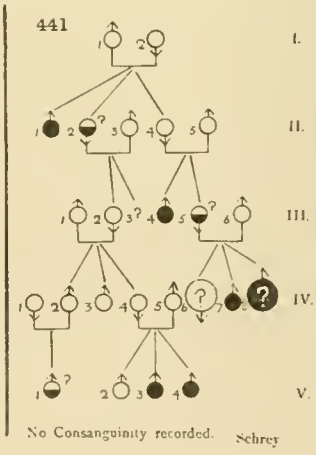






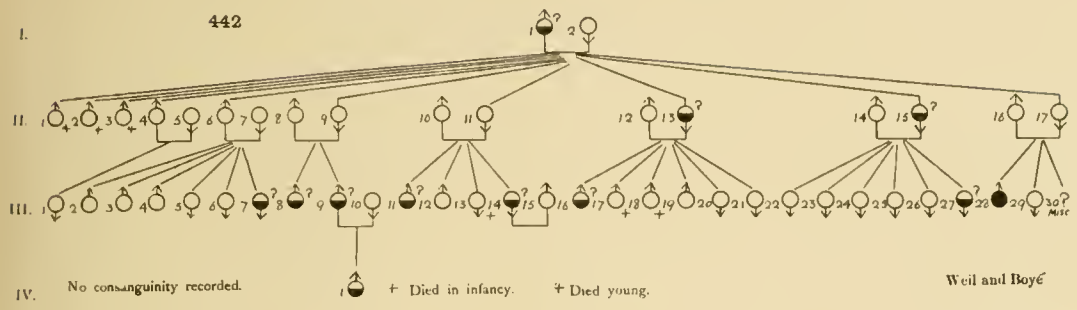






† Died of the disease

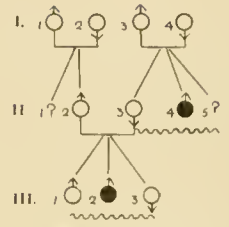
442



No consanguinity recorded. † Died in infancy. ✝ Died young.

Weil and Boyé

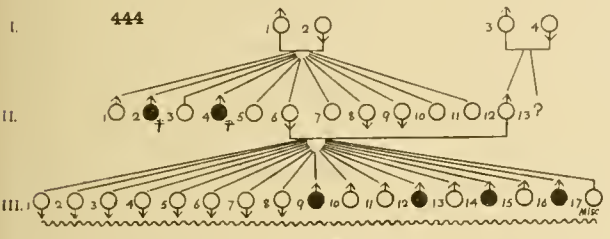
443



No consanguinity recorded.

Weil I

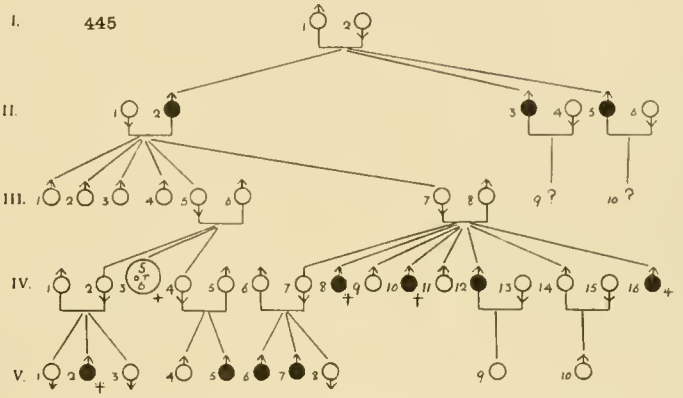
444



No consanguinity recorded.

Weil II.

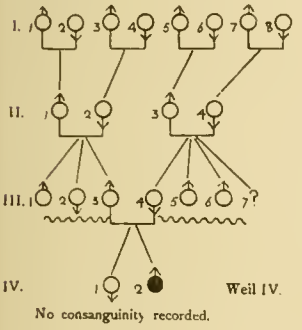
445



No consanguinity recorded. † Died in infancy ✝ Died young.

Weil III.

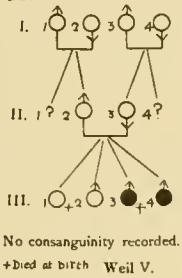
446



No consanguinity recorded.

Weil IV.

447

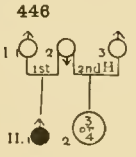


No consanguinity recorded.

†Died at birth

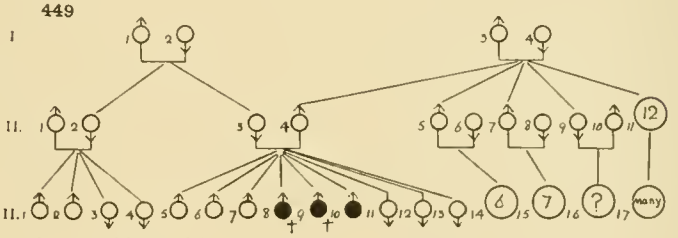
Weil V.

446



MacCormacI.

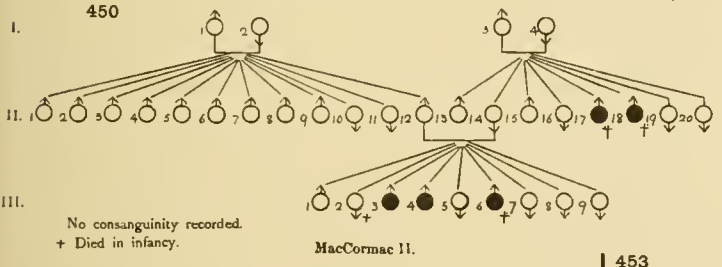
449



No consanguinity recorded.

Miloe

450

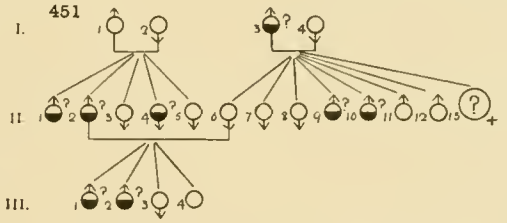


No consanguinity recorded.

† Died in infancy.

MacCormac II.

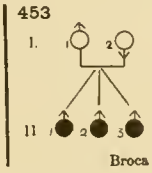
451



No consanguinity recorded. † Died in infancy.

MacCormac III.

453



Broca

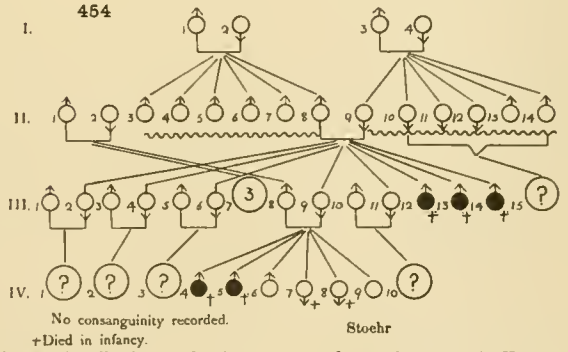
452

No consanguinity recorded.

✝ Died of the disease

von Limbeck

454



No consanguinity recorded.

†Died in infancy.

Stoehr

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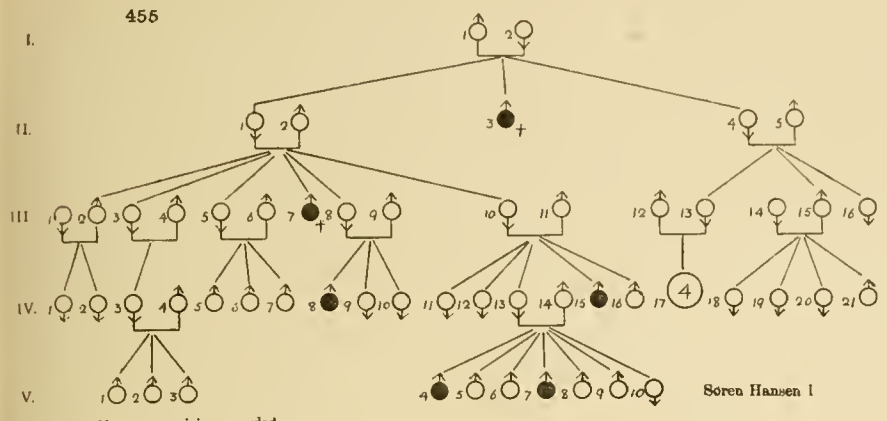
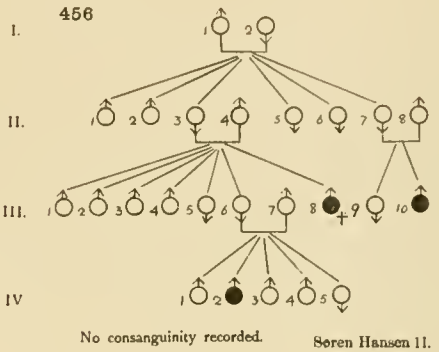
455



No consanguinity recorded.

Soren Hansen I

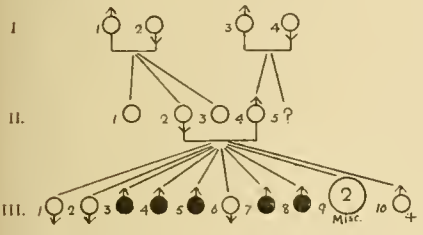
456



No consanguinity recorded.

Soren Hansen II.

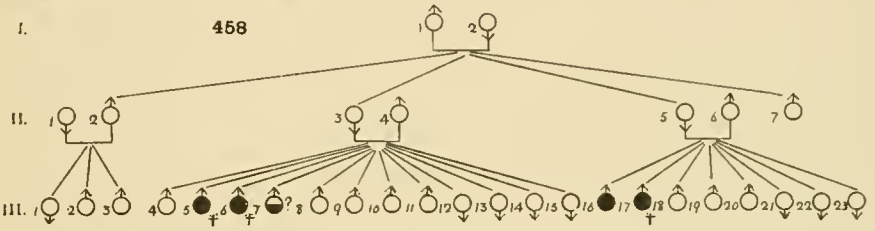
457



No consanguinity recorded. † Still-born.

Soren Hansen III.

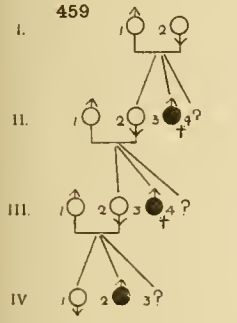
458



No consanguinity recorded.

Soren Hansen IV.

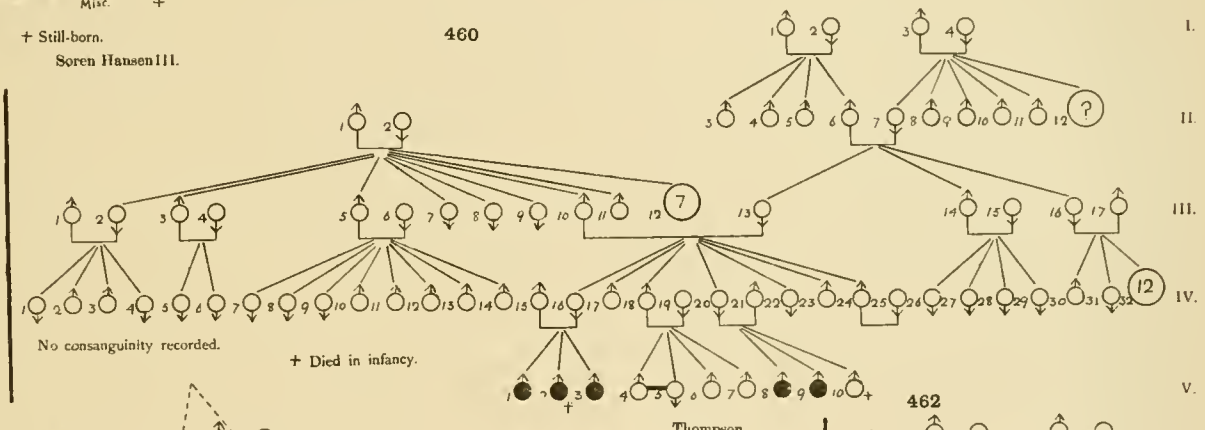
459



No consanguinity recorded.

Ledoux

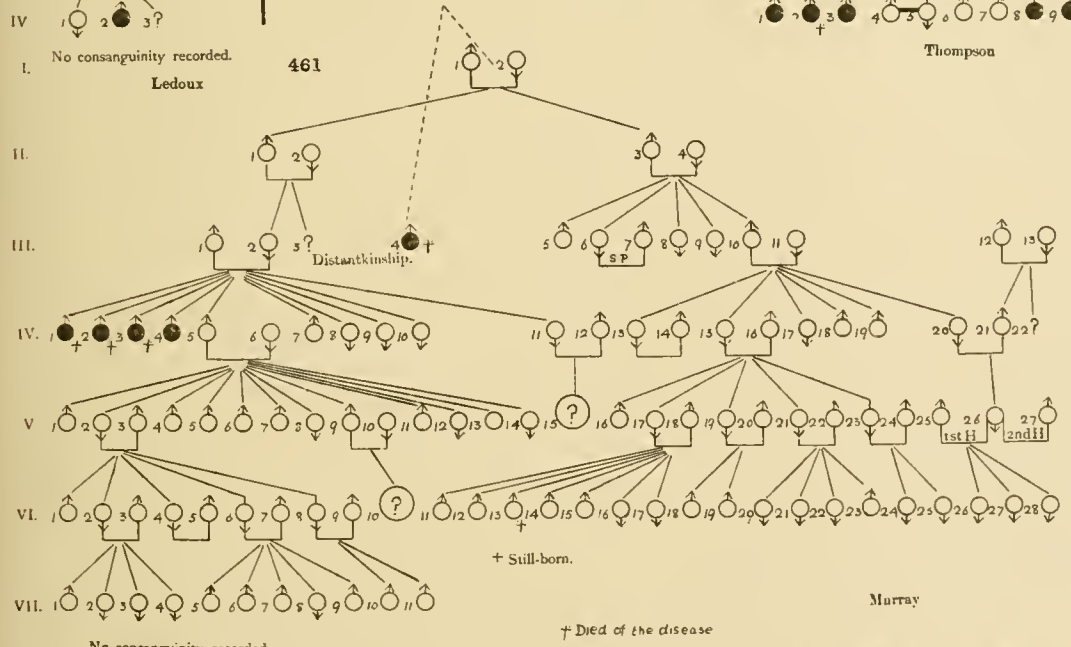
460



No consanguinity recorded. + Died in infancy.

Thompson

461



Distantkinship.

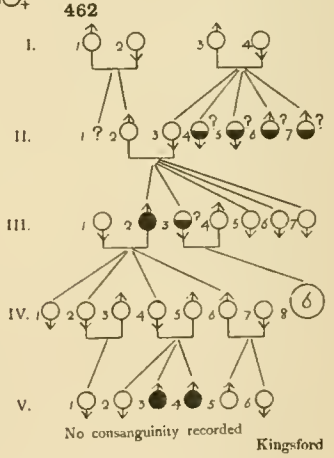
+ Still-born.

† Died of the disease

Murray

No consanguinity recorded.

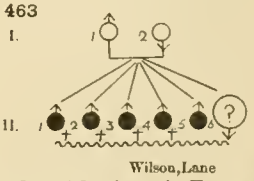
462



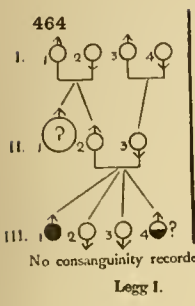
No consanguinity recorded

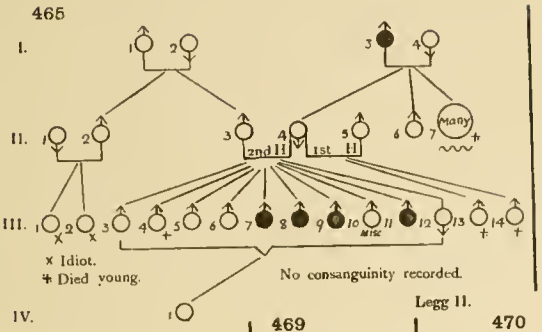
Kingsford

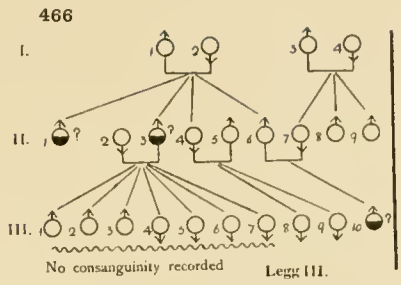
463

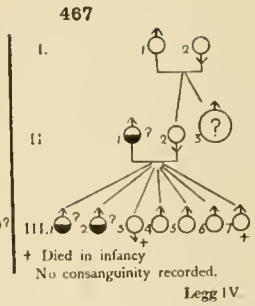


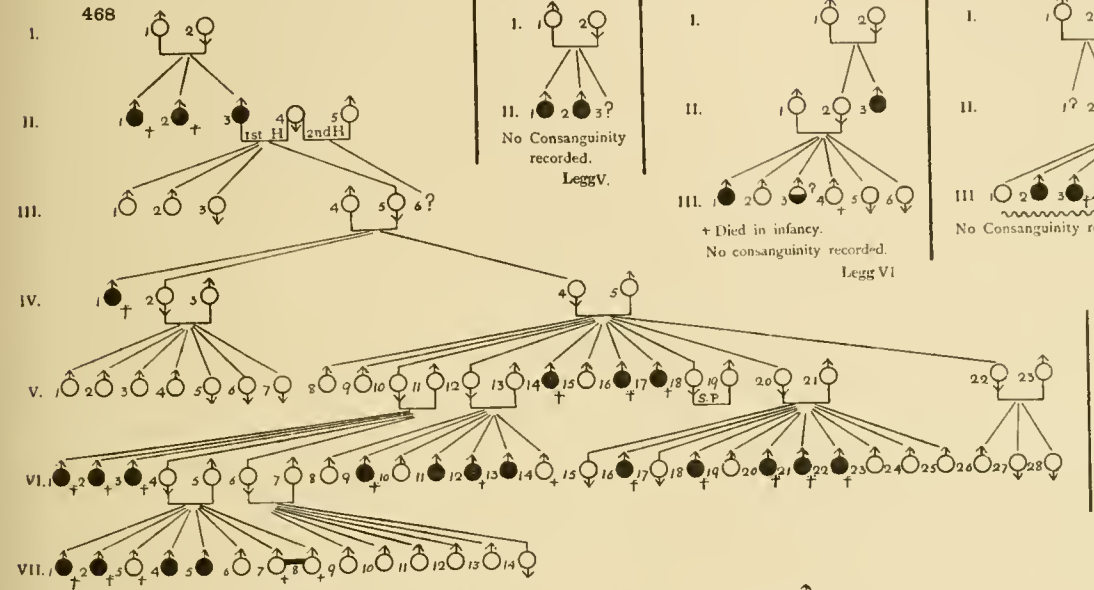
Wilson, Lane


464
No consanguinity recorded.
Legg I.


465
1st H
2nd H
many
× Idiot.
† Died young.
No consanguinity recorded.
Legg II.

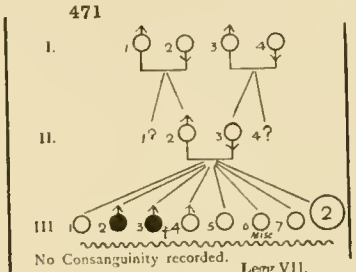

466
No consanguinity recorded
Legg III.

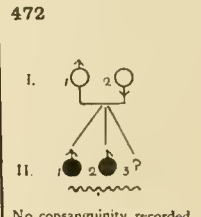

467
† Died in infancy
No consanguinity recorded.
Legg IV

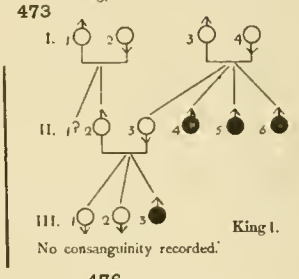

468
1st H
2nd H
† Died in infancy. No Consanguinity recorded. Legg IX.

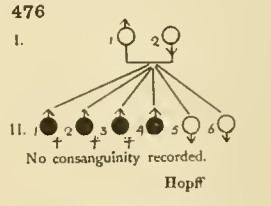
469
No Consanguinity recorded.
Legg V.

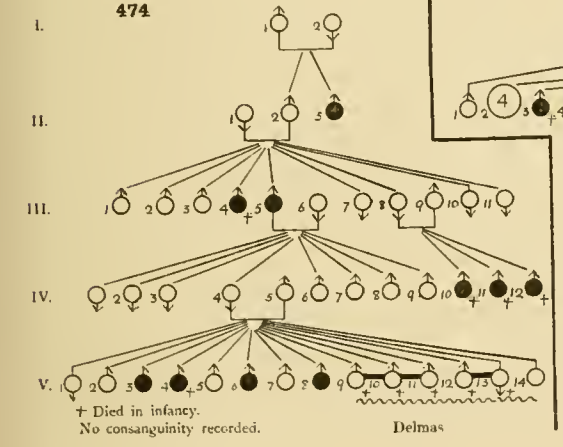
470
† Died in infancy.
No consanguinity recorded.
Legg VI


471
No Consanguinity recorded.
Legg VII.

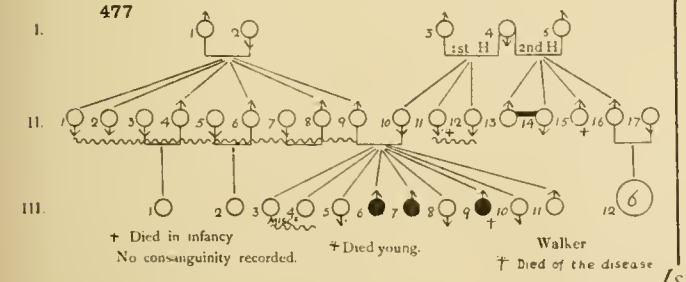

472
No consanguinity recorded.
Legg VIII.

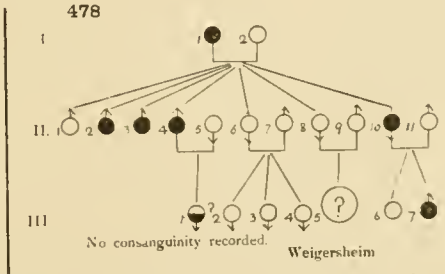

473
King I.
No consanguinity recorded.

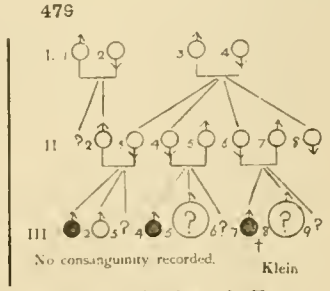

476
No consanguinity recorded.
Hopff


474
† Died in infancy.
No consanguinity recorded.
Delmas

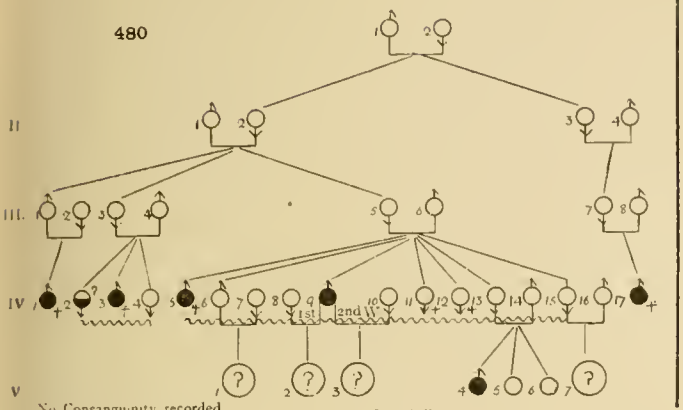
475
1st W
2nd W
† Died in infancy.
No consanguinity recorded.
King II.


477
1st H
2nd H
† Died in infancy
No consanguinity recorded.
† Died young.
Walker
† Died of the disease


478
No consanguinity recorded.
Weigersheim

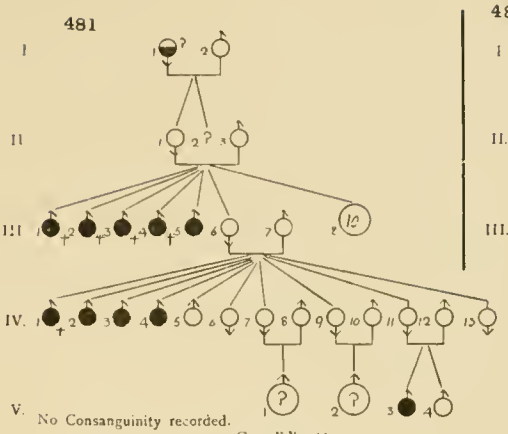

479
No consanguinity recorded.
Klein

480



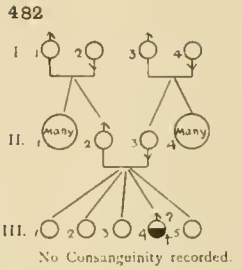
No Consanguinity recorded.
† Died young.
Grandidier I.

481



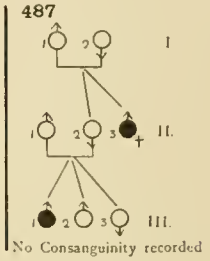
No Consanguinity recorded.
Grandidier II.

482



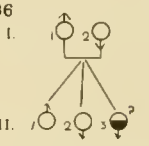
No Consanguinity recorded.
Grandidier III

487



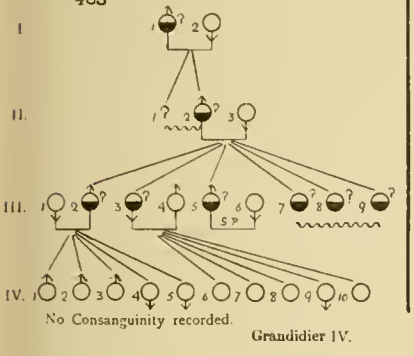
No Consanguinity recorded
Grandidier VIII.

486



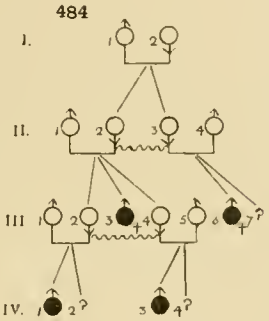
Grandidier VII.

483



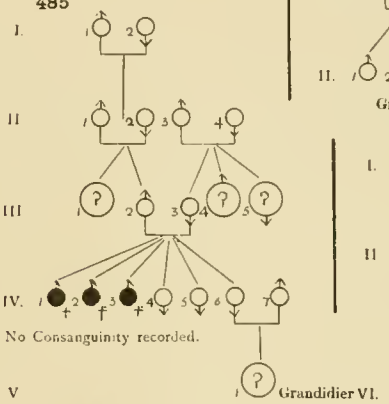
No Consanguinity recorded.
Grandidier IV.

484



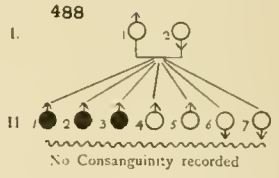
No Consanguinity recorded
Grandidier V.

485



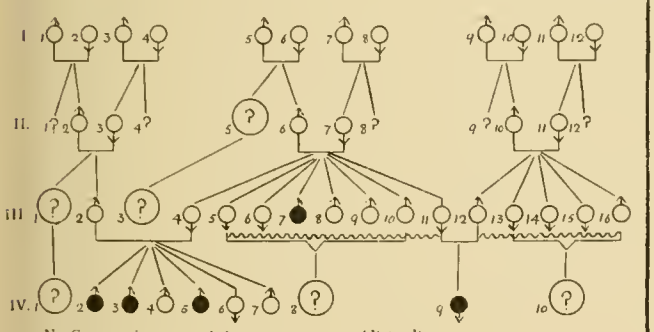
No Consanguinity recorded.
Grandidier VI.

488



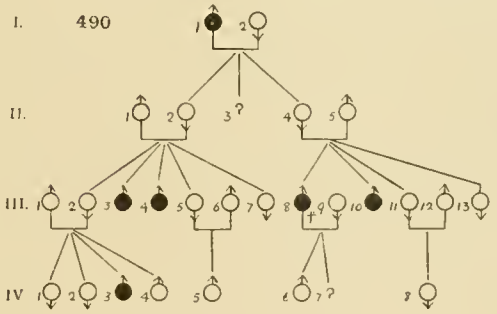
No Consanguinity recorded
Grandidier IX

489



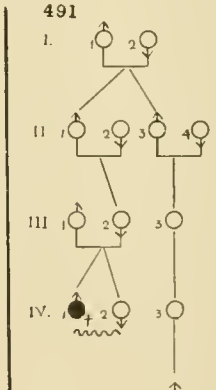
No Consanguinity recorded.
Altstaedt

490



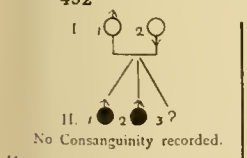
No Consanguinity recorded.
Grant

491



No Consanguinity recorded.
Lemp

492

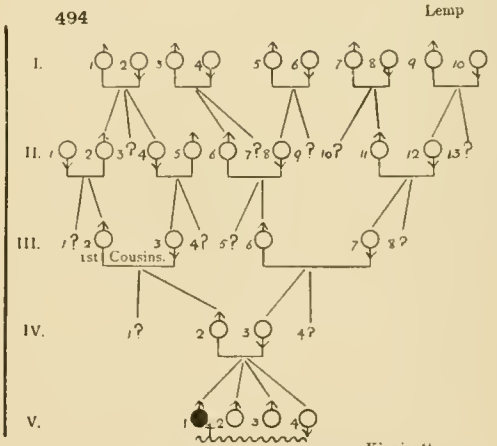


No Consanguinity recorded.
Baum

493

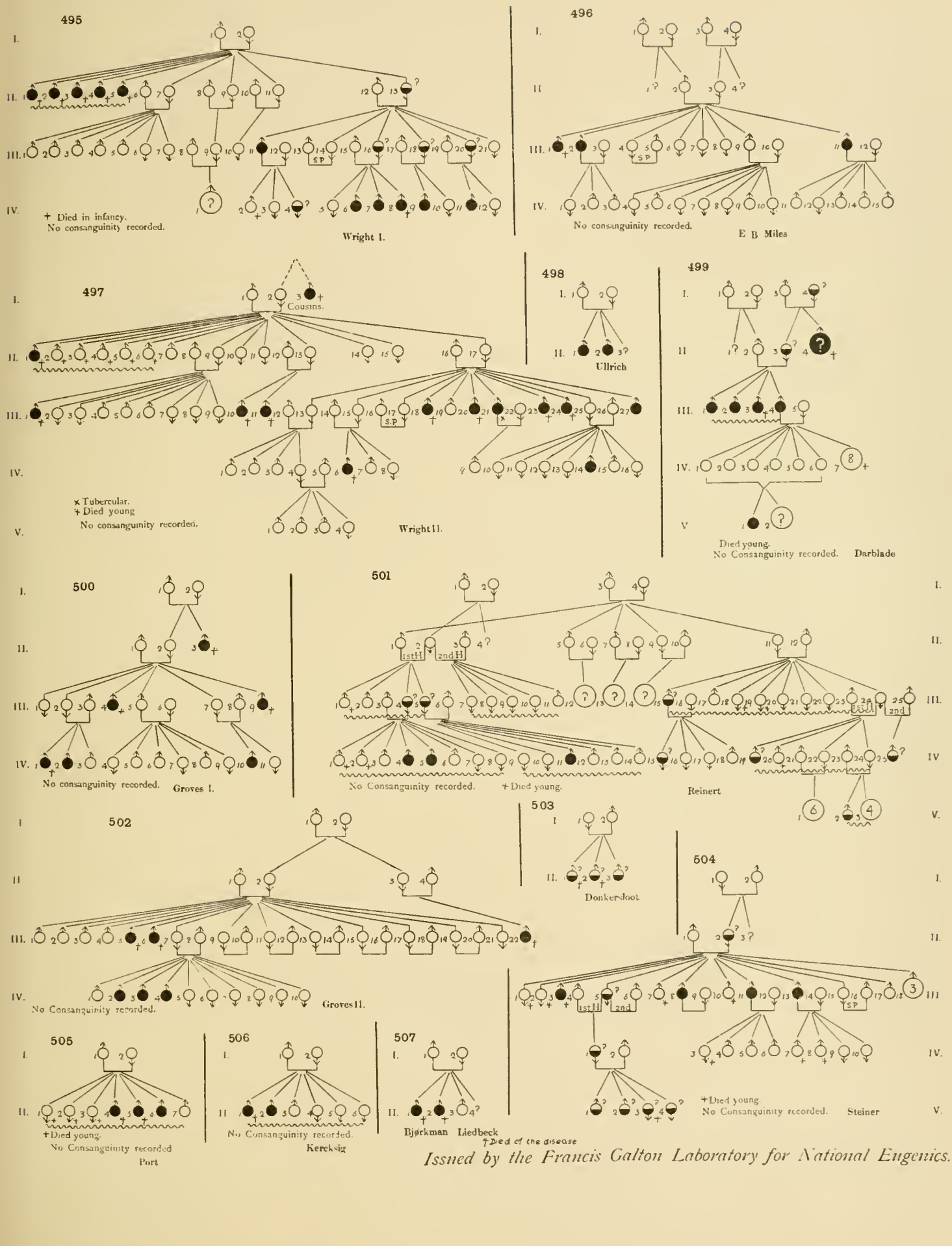
Consanguinity and No Consanguinity
† Died of the disease
Treves

494



Consanguinity.
Kinnicutt

Issued by the Francis Galton Laboratory for National Eugenics.


495
† Died in infancy.
No consanguinity recorded.
Wright I.
496
No consanguinity recorded.
E B Miles
497
Cousins.
× Tubercular.
† Died young
No consanguinity recorded.
Wright II.
498
Ullrich
499
Died young.
No Consanguinity recorded.
Darblade
500
No consanguinity recorded.
Groves I.
501
1st H
2nd H
No Consanguinity recorded.
† Died young.
Reinert
502
No Consanguinity recorded.
Groves II.
503
Donkersloot
504
† Died young.
No Consanguinity recorded.
Steiner
505
† Died young.
No Consanguinity recorded
Port
506
No Consanguinity recorded.
Kerchsig
507
Bjørkman
Liedbeck
† Died of the disease

508

† Died young.
No Consanguinity recorded

Grusche

509

† Died in infancy
No consanguinity recorded.

Schliemann

510

No consanguinity recorded.

Rave

511

No Consanguinity recorded.

Hertzka

512

† Died young. † Still-born. † Miss
No consanguinity recorded.

Simon I

513

Simon II

514

No consanguinity recorded.

Wijmans (i)

515

No consanguinity recorded.

Wijmans (ii)

516

Otto

517

No consanguinity recorded

Bramwell

518

No consanguinity recorded.

Page

† Died of the disease.

519

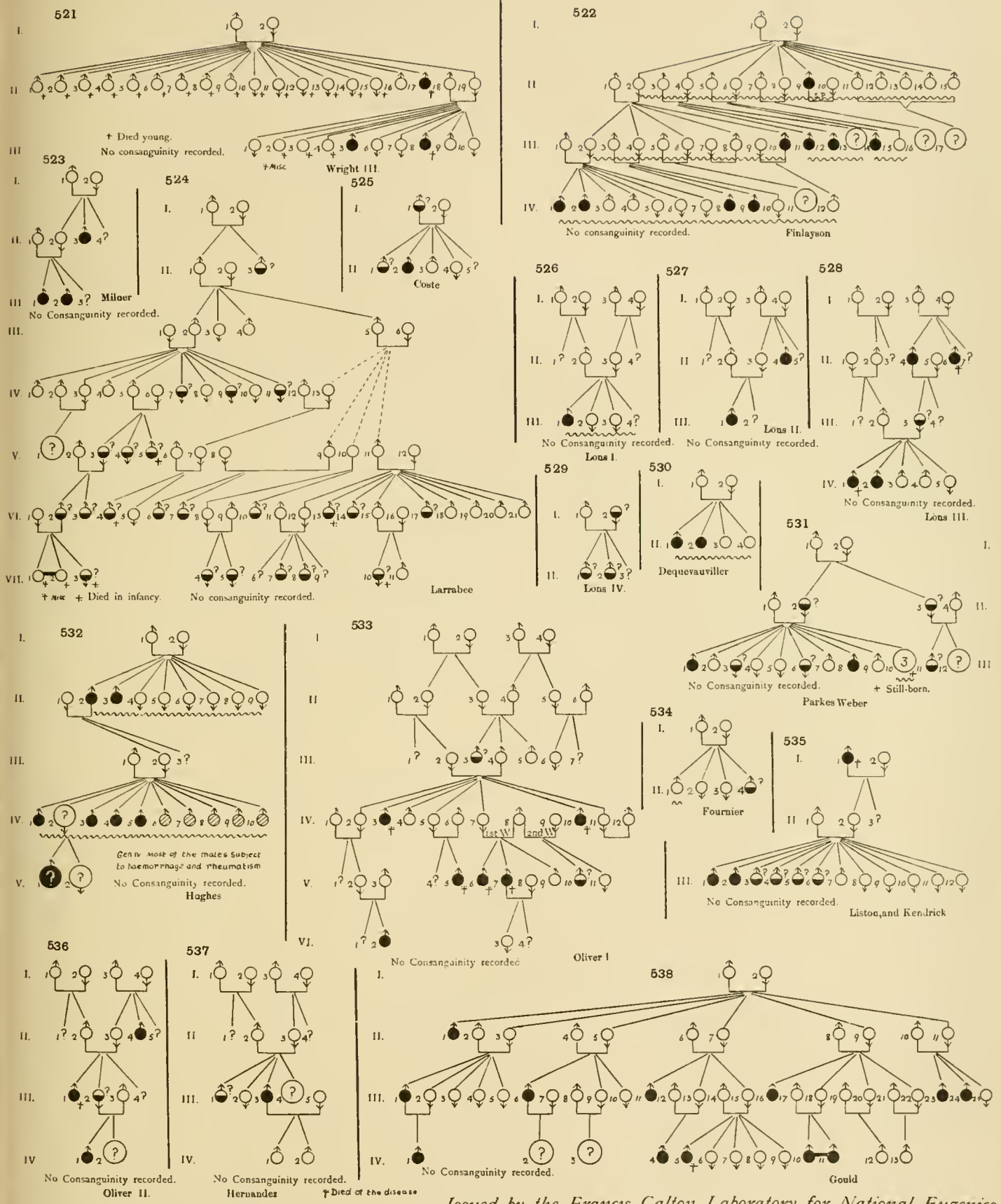
No consanguinity recorded.

Campani

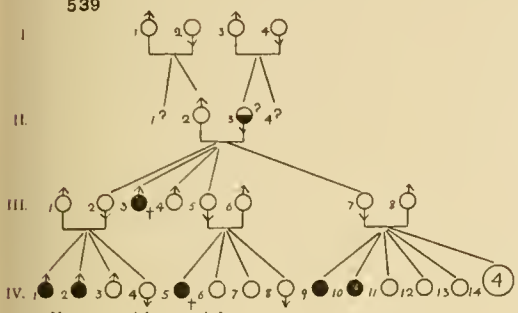
520

No Consanguinity recorded

Young


521
† Died young.
No consanguinity recorded.
Wright III.
522
No consanguinity recorded.
Finlayson
523
Milner
No Consanguinity recorded.
524
525
Coste
526
No Consanguinity recorded.
Lons I.
527
No Consanguinity recorded.
Lons II.
528
No Consanguinity recorded.
Lons III.
529
Lons IV.
530
Dequevauviller
531
No Consanguinity recorded.
† Still-born.
Parkes Weber
532
Gen IV most of the males subject to haemorrhage and rheumatism
No Consanguinity recorded.
Hughes
533
No Consanguinity recorded
Oliver I
534
Fournier
535
No Consanguinity recorded.
Liston and Kendrick
536
No Consanguinity recorded.
Oliver II.
537
No Consanguinity recorded.
Hernandez
† Died of the disease
538
No Consanguinity recorded.
Gould
† Abor. † Died in infancy.
No consanguinity recorded.
Larrabee

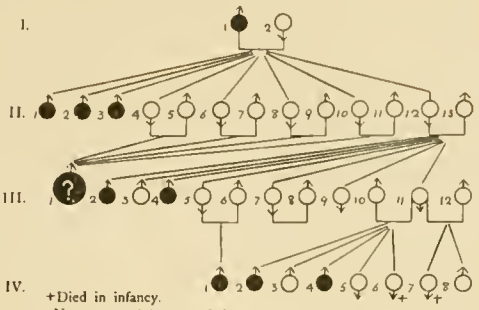
539



No consanguinity recorded.

Newcombe

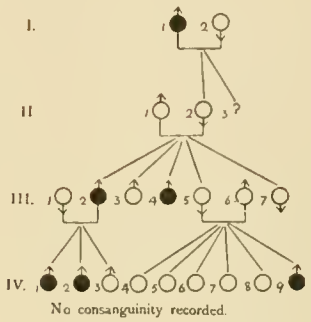
540



+Died in infancy.
No consanguinity recorded.

Heath

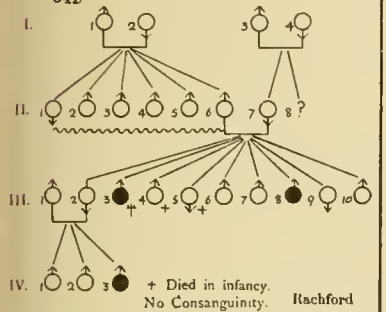
541



No consanguinity recorded.

Masters

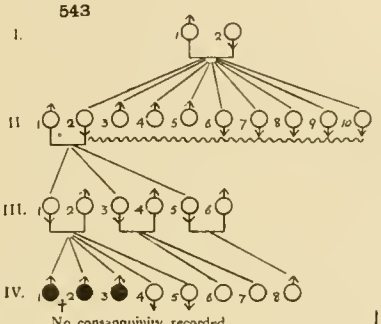
542



+ Died in infancy.
No Consanguinity.

Rachford

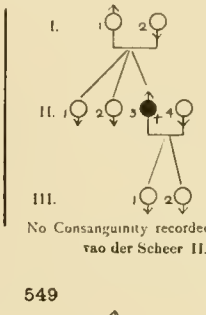
543



No consanguinity recorded.

van der Scheer I.

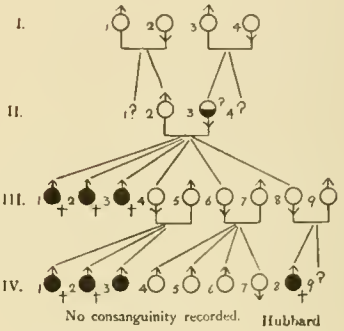
544



No Consanguinity recorded.

van der Scheer II.

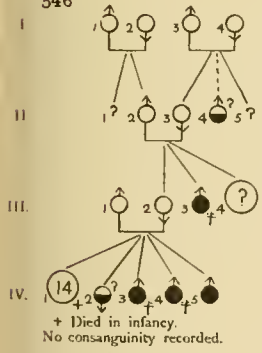
545



No consanguinity recorded.

Hubbard

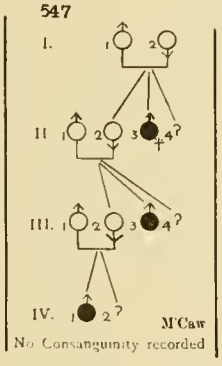
546



+ Died in infancy.
No consanguinity recorded.

Lafargue — Laborie

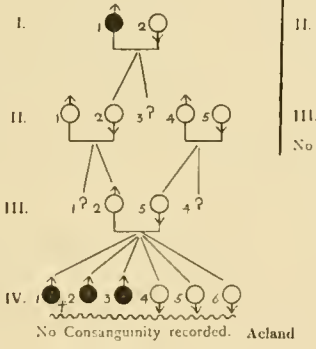
547



M'Caw

No Consanguinity recorded

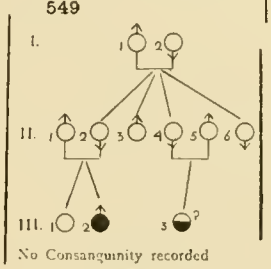
548



No Consanguinity recorded.

Acland

549



No Consanguinity recorded

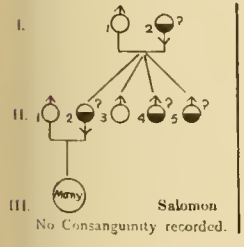
Forster(i)

550

No Consanguinity recorded.

Forster(ii)

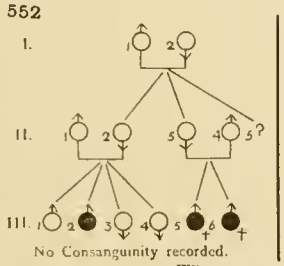
551



No Consanguinity recorded.

Salomon

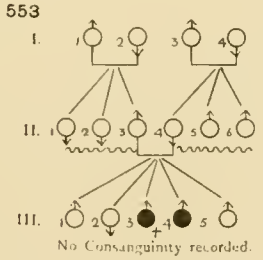
552



No Consanguinity recorded.

Wilmot

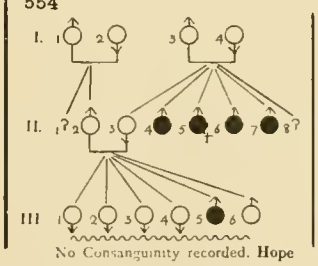
553



No Consanguinity recorded.

W. Jenner

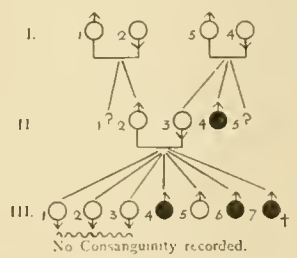
554



No Consanguinity recorded.

Hope

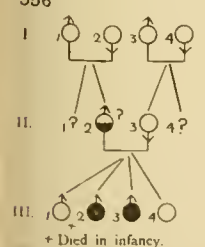
555



No Consanguinity recorded.

Maixner

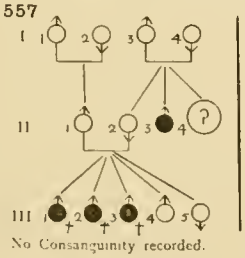
556



+ Died in infancy.
No Consanguinity recorded

Price

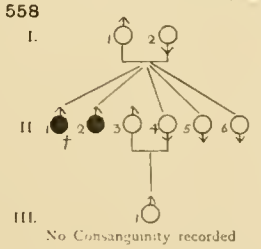
557



No Consanguinity recorded.

Payne

558

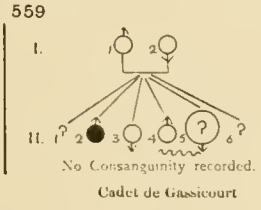


No Consanguinity recorded

† Died of the disease

Besserer

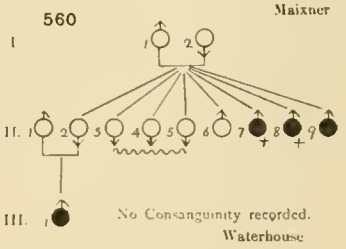
559



No Consanguinity recorded.

Cadet de Gassicourt

560



No Consanguinity recorded.

Waterhouse

561

No Consanguinity, in affected branches

+Died young.

Nettleship.

562

No Consanguinity recorded. Zoege von Manteuffel

563

No consanguinity recorded. Muller

564

V. No consanguinity recorded. Nesterovski

565

+ Died in infancy.

No consanguinity recorded. Kerr

566

No consanguinity recorded.

Fussell

567

No Consanguinity recorded. Nash

566

× Tubercular

No Consanguinity recorded. Power

569

+ Died young.

Consanguinity and No Consanguinity. Mosonyi

570

No Consanguinity recorded. Hutchison

571

+ Died in infancy

× Tubercular

No Consanguinity recorded. Barlow

572

No Consanguinity recorded.

Trimme Beth

573

No consanguinity recorded.

Heymann

574

+ Died young.

No consanguinity recorded Greig Smith I.

575

No consanguinity recorded. Greig Smith II.

576

† Died of disease

No Consanguinity recorded Hollinay

577

No Consanguinity recorded

Speidel

576

× Tubercular. Finger

No Consanguinity recorded

579

No Consanguinity recorded

Osborne

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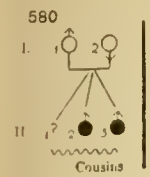
580



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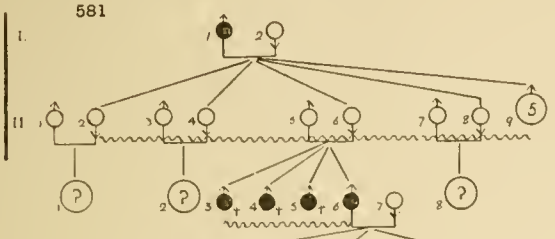
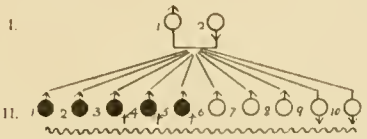


† Died very young. No Consanguinity recorded. Muir I.

582



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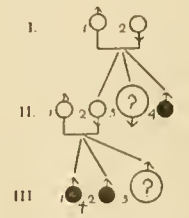
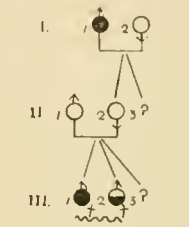


No Consanguinity recorded
Consbruch

584



No Consanguinity recorded.
Froelich

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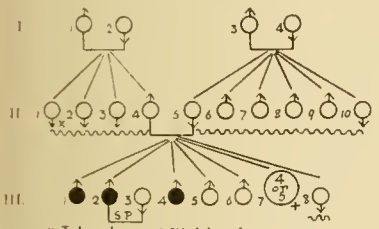
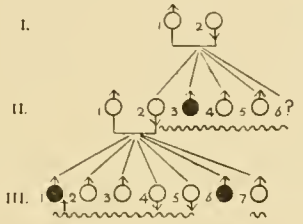


× Tubercular. † Died in infancy
No Consanguinity recorded. Jacob

586



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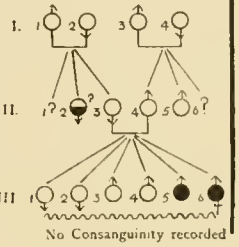

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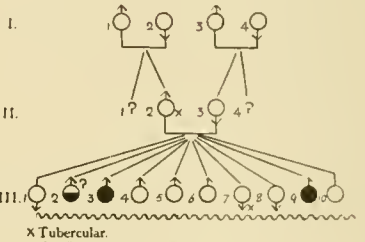
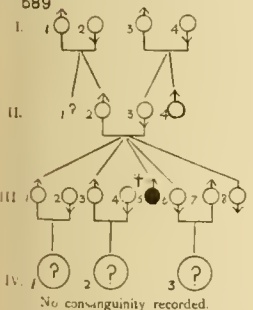


× Tubercular.
No Consanguinity recorded. Moses

589



No consanguinity recorded.
M'Causland I.

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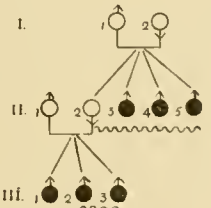

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Clay

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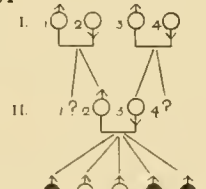

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Schreyer

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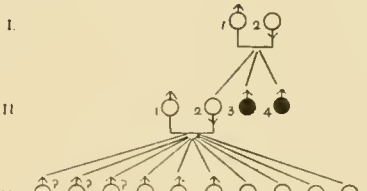
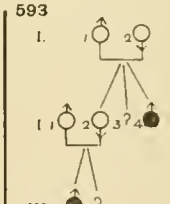


No consanguinity recorded Burnes

593



No consanguinity recorded.
Knudtzon

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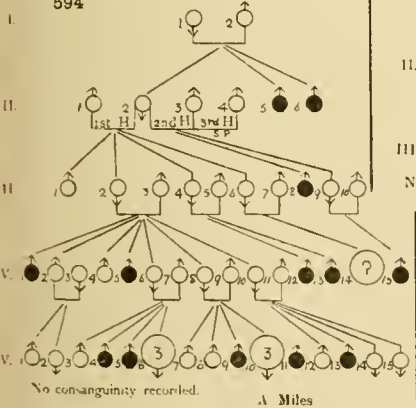
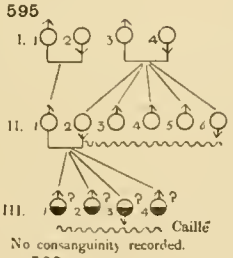


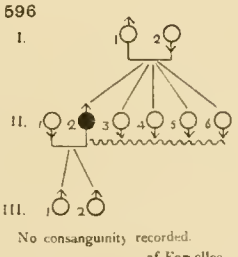
No consanguinity recorded. A Miles

595



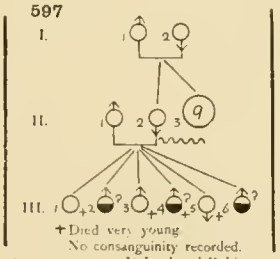
Caillé
No consanguinity recorded.

596



No consanguinity recorded. af Forselles

597



† Died very young
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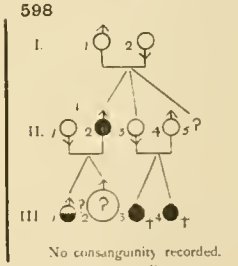
Daland and Robinson

598



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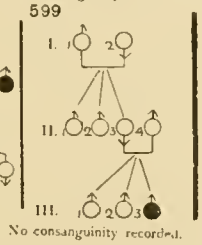
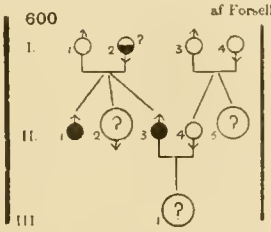
Bruinsma

599



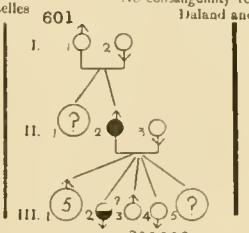
No consanguinity recorded.
M'Causland II.

600



No Consanguinity recorded.
Meinel I

601



No consanguinity recorded Meinel II

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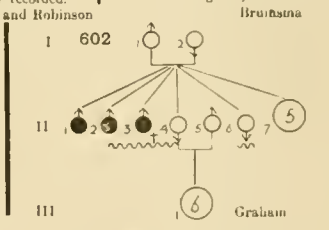
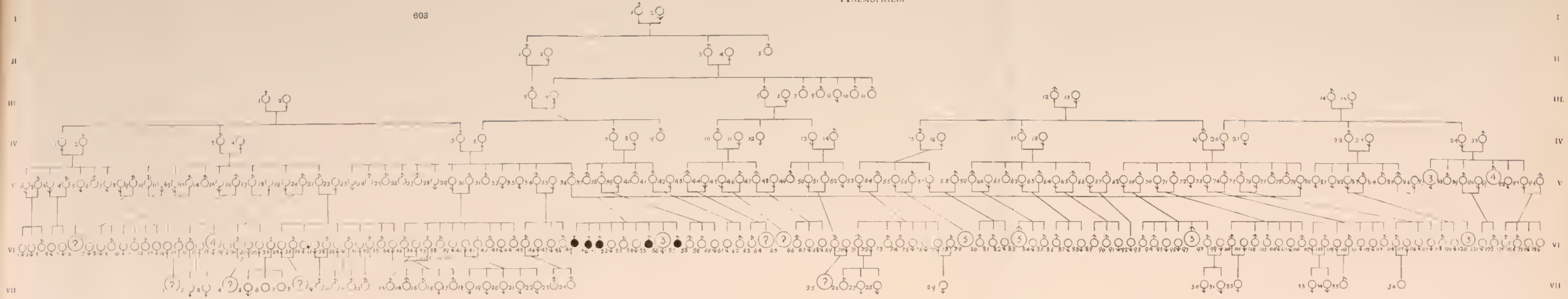


No Consanguinity recorded

† Died of the disease

603



No Consanguinity

Gettings.

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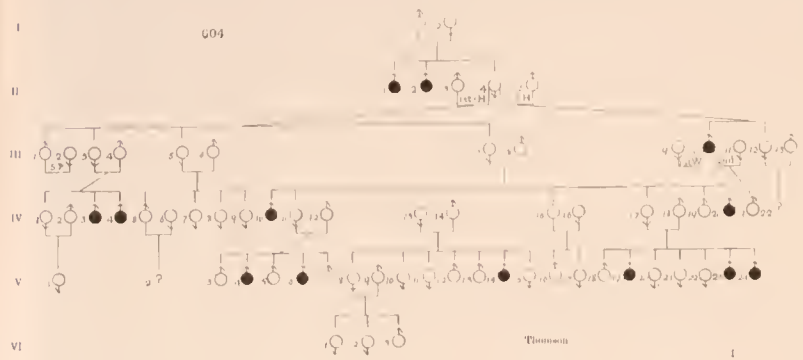

No Consanguinity.

Thomsen

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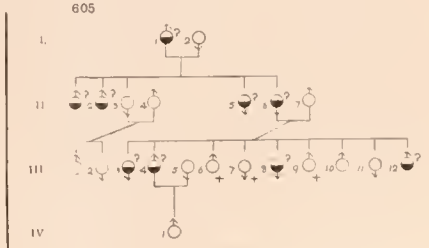
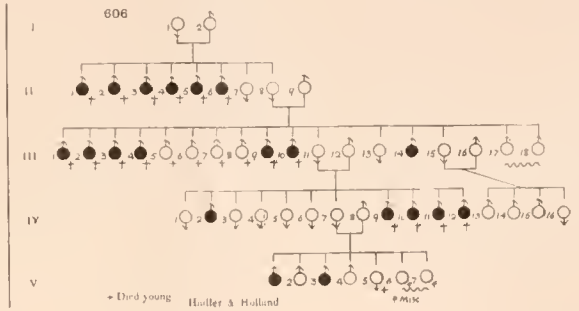


+ Died young

Neumann

606

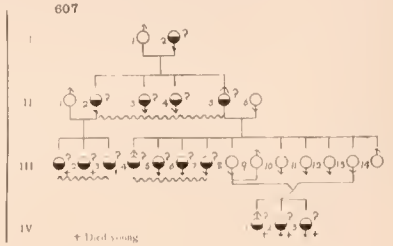


+ Died young

Hoffer & Holland

No Consanguinity recorded

607

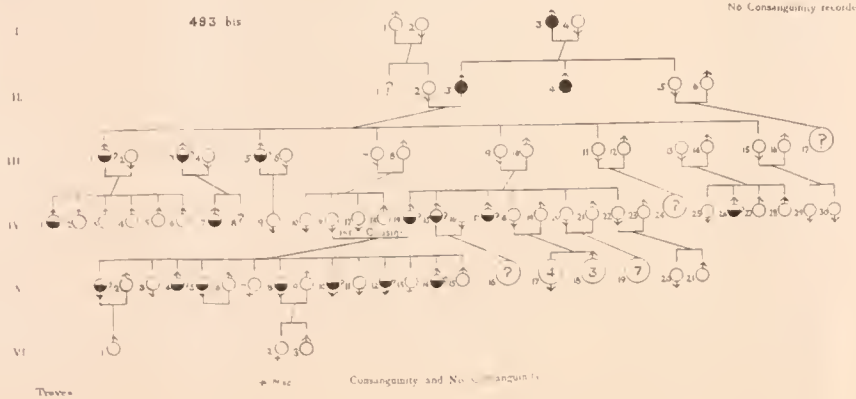


+ Died young

H. H. Pearson.

No Consanguinity recorded

483 bis



Consanguinity and No

Treves

UNIVERSITY OF LONDON

FRANCIS GALTON LABORATORY FOR NATIONAL EUGENICS

EUGENICS LABORATORY MEMOIRS. XV.

TREASURY OF HUMAN INHERITANCE

PARTS VII AND VIII

SECTION XV A. DWARFISM

BY

H. RISCHBIETH, M.A., M.D. CANTAB., F.R.C.S. ENG.

AND

AMY BARRINGTON, EUGENICS LABORATORY

WITH 8 PLATES OF PEDIGREES AND 35 PLATES OF ILLUSTRATIONS

PLATES LI—LVIII PLATES O—Z, AA—WW

PEDIGREES, FIGS. 608—841

CAMBRIDGE UNIVERSITY PRESS

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1912

THE TREASURY OF HUMAN INHERITANCE

ISSUED BY

THE FRANCIS GALTON LABORATORY FOR NATIONAL EUGENICS

The Francis Galton Laboratory is issuing in parts at short intervals a collection of published and unpublished family pedigrees, illustrating the inheritance in man of mental and physical characters, of disease and of abnormality.

Students of heredity find great difficulty in obtaining easy access to material bearing on human inheritance. The published material is voluminous, scattered over a wide and often very inaccessible journalistic area. The already collected although unpublished material is probably as copious, but no central organ for its rapid publication in a standardized form exists at present. The Eugenics Laboratory alone possesses several hundred pedigrees of family characteristics and diseases which it is desirable to make readily accessible. Many medical men possess similar material, and there is a growing desire among genealogists to pay more attention to family characters and supplement the merely nominal pedigrees current in the past.

For a publication of this kind to be successful at the present time, it should be entirely free from controversial matter. The *Treasury of Human Inheritance* will therefore contain no reference to theoretical opinions. It will give in a standardized form the pedigree of each stock. This will be accompanied by a few pages of text describing the individual members of the stock, giving references to authorities, and, if the material has been published, to the *locus* of original publication. When necessary the characteristic will be illustrated by photography or radiography. In this way, it is hoped in the course of a few years to place a large mass of material in the hands of the student of human heredity. It will not cut him off from, but directly guide him to original and fuller sources of information. Further, the *Treasury* will provide students of eugenics and of sociology, medical men, and others, with an organ where their investigations will find ready publication, and where as time goes on a higher and more complete standard of family history than has hitherto been usual can be maintained. It is proposed to issue the *Treasury of Human Inheritance* in quarto parts at about quarterly intervals. Each part will contain about 6 to 10 plates of pedigrees and of such other illustrations as may be needful.

The following parts have already been issued:—

Parts I and II (double part) contain pedigrees of Diabetes Insipidus, Split-Foot, Polydactylism, Brachydactylism, Tuberculosis, Deaf-Mutism, and Legal Ability. Price 14*s. net.*

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The subscription to each set of four parts is 24*s.*, and all communications with regard to pedigree contributions should be sent to: The Editor, *Treasury of Human Inheritance*, Eugenics Laboratory, University College, London, W.C. Subscriptions should be made payable to The Hon. Secretary, Galton Eugenics Laboratory, at the above address.

Single parts may be purchased from Messrs Dulau & Co., Ltd., 37, Soho Square, London, W., either directly or through any bookseller.

EDITORIAL NOTE

Owing to the illness of Dr Rischbieth which has prevented him from replying for some months to business letters sent to him, the part of this section which is due to him is not issued in a form which has received his final approval. The Editor is responsible for many corrections and additions, beyond the footnotes in brackets. Most of the historical and bibliographical references have been supplied by the Staff of the Laboratory, which is wholly responsible for the Bibliography, the Iconography, the Indices, a large section of the photographic illustrations and all the family histories on six out of the eight pedigree plates. The Editor regrets this divided responsibility, but a large proportion of the text of Dr Rischbieth's contribution was in type before we were deprived of his assistance and it did not seem possible without cancelling the portion already in type to obtain other and adequate medical aid. The Laboratory Staff has done its best to complete this important section, but the Editor realises that this account of Dwarfism lacks something of unity and completeness owing to the circumstances of its production. He has most heartily to thank his colleague Professor George D. Thane for much helpful advice and replies to many questions.

K. P.

With these Parts, Vol. I. of the *Treasury of Human Inheritance* is completed. Prefatory matter and a copious Index to the volume will be issued, Price 2*s.* 6*d.* (Dulau and Co., Ltd., 37 Soho Square) to those desirous of binding these parts as a single volume. Buckram cases for binding with an impress of Sir George Frampton's bust of Francis Galton can be obtained by sending a postal order for 2*s.* 9*d.* to the Hon. Secretary of the Eugenics Laboratory, University College, London.

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(This will be replaced by a complete list of contents when the first volume is finished.)

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Section I. *Achondroplasia and Probable Achondroplasia.*

ILLUSTRATIVE PLATES.

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Section XVa. DWARFISM.

By H. Rischbieth, M.A., M.D., B.C. Cantab., F.R.C.S. Eng.

I. INTRODUCTION.

The term "dwarf" (German "Zwerg," French "nain") is a general one applied to individuals markedly below the average height of the species. There are dwarf races (ethnic dwarfs) and dwarf individuals in normal races; amongst the latter dwarf growth appears usually to be the effect of disease.

We are here concerned with the heredity of dwarf growth the result of disease (that is, with those varieties of the group which show this influence in causation clearly at the present day), but in order that the relationships of members of this group to others be preserved, a short reference to ethnic dwarfs will perhaps be permitted though the subject belongs to anthropology, not pathology. Dwarf races seem to have been mentioned in the literature and legends of most languages. The Greek and Latin writers refer frequently to dwarf races[1]. In modern European languages legends of dwarfs or pygmies, fairies, pixies, cobbolds and Nibelungs are

[1 The word pygmy (πυγμή) signifies the length from elbow to knuckles, although it is not clear that it has not sometimes been used in the sense of cubit, rather than of 13·5″. Homer (*fl.* 900 B.C. ?) refers in the opening sentences of the *Iliad*, Book III. to the legends of the war between cranes and pygmies. Herodotus (*fl.* 443 B.C.), Book III. Chap. 37, says that the "image of Hephaestos at Memphis was in form resembling a pygmy." Rawlinson (Vol. II. p. 434) commenting on this passage gives good illustrations of the pygmy figures of Ptah-Sokar found in Egypt, one at least being of achondroplasic type (see also our Plate S (14)). While the Greeks in the best period represented Hephaestos as a vigorous man, they still placed dwarf-like statues of this god near the hearth, and he was said to be lame and weak from birth. This legend must be considered in line with others that attribute metal and smith's work to the dwarfs. Ctesias (*fl.* 401 B.C.) places a race of pygmies in India, and asserts that their oxen and asses are of the size of rams (*Operum reliquiae*, ed. Baehr, 1824, p. 293; Baehr commenting on Ctesias, while admitting that Ctesias only heard Indian stories at second-hand, refers to travellers' tales of men and horses of small stature in India and considers that a dwarf race cannot be dismissed as wholly fabulous). Aristotle (*fl.* 347 B.C.) refers on two occasions to dwarfs. In the *Probl. Sect.* X. 14 he considers the origin of the νᾶνος. This word signifies really one whose limbs are too small for his body, and should refer to the achondroplasic and not the true dwarf; Aristotle suggests as causes, locality, nourishment and heredity. In the *Hist. Animalium*, VIII. 12, Aristotle refers to the cranes changing their dwelling from the Scythian plains to the low ground by the rivers of Upper Egypt, and speaks of their attacking the pygmies. He asserts that this is no mere tale, but there is really a small race, both of men and horses, and that the pygmies live in holes. Strabo (*fl.* 24 B.C.) refers in his *Geographica* at several points to the legends of the pygmies and mentions Homer and Hesiod (a search through Hesiod has not provided any evidence) as speaking of them (Lib. I. Cap. II. 28, 30 and 35, Lib. VII. Cap. III. 6). Lib. I. Cap. XXXV. places them in Aethiopia. Lib. II. Cap. I. 9 refers to their Homeric fight with the cranes, and Strabo tells us that writers about India mention pygmies. In Lib. XVII. Cap. II. 1 he states that the smallness of the Aethiopian cattle probably suggested pygmies, "whom nobody worthy of credence has seen." Pomponius Mela (*fl.* 50 A.D.) places (*De Situ Orbis*, Lib. III. Cap. VIII. l. 71) the pygmies towards the source of the Nile ("Fuere iuterius Pygmaei, minutum genus, et quod pro satis frugibus contra grues dimicando deficit"). Pliny (*fl.* 59 A.D.) in his *Natural History*, Bk. VII. Chap. II. places the *Pygmaei Spithamaei*—the span-high pygmies—far up in India above the sources of the Ganges, on the skirts of the mountains, and he makes them 27″ in stature. He quotes Homer's account, and says that they make their homes of clay or mud, birds' feathers and egg-shells, but notices Aristotle's statement that they live in holes. In Bk. VI. Chap. XXX. Pliny also refers to the pygmies at the source of the Nile. Aulus Gellius (*fl.* 140 A.D. Lib. IX. Cap. IV.) is of opinion that Pliny's statements about the pygmies are idle. Aelianus (*fl.* 150 A.D.), *De Nat. Animal.*, mentions dwarf animals in India and Africa (XVI. 37). Elsewhere (XV. 29) he says that the pygmies had a kingdom of their own and were governed by a queen Gerana, who, when divine honours were paid to her, became inflated and despised the goddesses themselves, saying that their figure would not compare with hers. Juno changed Gerana into a crane (γέρανος), which bird is always at enmity with the pygmies because they brought infamy upon her owing to their excessive honours. Philostratus (*fl.* 250 A.D.) relates in his *Iconum Lib. Sec.* Cap. XXII. how Hercules fell asleep and how while sleeping he was attacked by armies of pygmies. He collected them all in his lion's skin and took them to Eurystheus. Ovid (*fl.* 13 B.C.) refers in his *Metamorphoses*, Lib. VI. 90—3, to the Juno and Gerana incident. Besides these references stretching over a thousand years to dwarf races—in districts where races of small stature have existed till modern times—there are one or two references to individual dwarfs. Thus Suetonius (*fl.* 110 A.D.) gives such in *Caes. Octavius Augustus*, Cap. 43, l. 320, Cap. 83, ll. 422—5. In the former passage he mentions a Roman knight Lucius "postea nihil sane, praeterquam adolescentem Lucium, honeste natum, exhibuit; tantum ut ostenderet, quod erat bipedali minor, librarum septemdecim, ac vocis immensae." The commentators are doubtful whether the name of this knight was Lucius. A dwarf of like stature appears to be referred to by Horace, Lib. II. Sat. III. l. 309. In Lib. I. Sat. III. 45, the same writer mentions Mark Antony's dwarf, Sisyphus, said to be under 2 ft. high, but very witty, and taking his name from his dexterity and cunning. Our Plate S (14) shows that the Romans were familiar with achondroplasic dwarfs. Edward Tyson published in 1699 a work entitled: *Orang Outang sive Homo Sylvestris, or the Anatomy of a Pygmy.* It is followed by a very learned essay on the pygmies of the ancients, whom Tyson believed to have been apes. This essay was republished in 1894 in the *Bibliothèque de Carabas*, Vol. IX. See Bibl. Nos. 15 and 294. For the folklore side of dwarfism, Cap. XVII. of J. Grimm's *Deutsche Mythologie* (Ausg. 1875, S. 369 *et seq.*) is still unexcelled. Editor.]

familiar. Dwarf races have been reported or fabled to exist in the Canary Islands (van Helmont, 17th century), on the Orinoco (Humboldt), in Abyssinia, Brazil, Japan and India (Wood, Bibl. No. 138, p. 245). Buffon[1] refers to a report of a dwarf race in Madagascar; this may have been merely a legend. But Schweinfurth, the African traveller, confirmed the statements of the ancient Greeks and later voyagers and travellers[2] that a race of dwarfs existed at the source of the Nile by finding representatives of such a race at the Court of King Munza at Monbuttu in 1870, and to them he applied the name "Akka[3]." In Sir H. M. Stanley's last expedition another tribe of dwarf negroes was found near Ruwenzori[4]. It is now known that dwarf races (negrillos) exist in many parts of Central Africa, and in South Africa as the Bosjesman, or Bushman, they have long been known. The negrillos have since received much attention from anthropologists, amongst them Virchow. Such a race or its traces also occurs in Southern Asia—in the Andaman Islands, Malay Peninsula, Philippine Islands, Borneo, New Guinea—the Negrittos[5]. These are regarded as of allied race to the negrillos of Africa.

The average height of the "Akkas" as given by de Quatrefages de Breau is:—men, 1 m. 50 to 52 cm. (4 feet 11 ins. approximately); and women, 1 m. 40 to 43 cm. (4 feet 7 ins. approximately). Haliburton[6] has reported the presence of a dwarf race in the Pyrenees, in the Atlas Mountains and in North America. With dwarf races such as the Negrillos such comparatively small races as the Laplanders, Eskimos and Japanese seem analogous. In Europe the excavations of ancient burial-grounds by Nuesch, Kollmann, Manouvrier, and others, appear to show the existence of a dwarf race contemporary with the Romans. The last of the dwarf races is believed to have disappeared from Europe about the 10th century.

The bearing of these facts concerning the wide distribution (either formerly or at the present day) of dwarf races upon the subject of pathological dwarf growth seems important, more especially with reference to varieties of the latter that show heredity in causation. There is, as is well known, amongst the proletariat of European cities and elsewhere, a great group of very undersized individuals. These are said to be especially common in Bavaria and in Italy. Are these survivals of an old dwarf race, or are they the product of disease or other factors of their environment? The pure ethnologist may possibly have one opinion and the pure pathologist another. Again, it seems possible that some of the instances that have been reported of racial dwarfism are really instances of disease: *e.g.* one of Haliburton's cases, to judge from the illustration and account, appears to be an instance of ateleiosis occurring in several members of a family. In mountainous regions such as the Pyrenees or Atlas

[1] *Supplément à l'Histoire naturelle*, T. IV. pp. 505—12, Paris, 1777. A. Grandidier (*Histoire physique, naturelle et politique de Madagascar*, T. I. pp. 652—54, Paris, 1908) discusses the legend at length, but considers no evidence for a pygmy race forthcoming.

[2] Dapper's account (*Naukeurige Beschrijvinge...*, Amsterdam, 1676, pp. 166, 216—8) of the Bakke-Bakke (?=Akka), or Mimos, a dwarf race in and beyond the Grand Mococo's kingdom seems definite enough both in locality and habits. Stanley, like Dapper, remarks on the practical invisibility of these dwarf-enemies. Nonnosius' account, both as to locality and habit, is less definite (Photius, *Myriobiblon*, Cod. 2, pm. 7). Thevenot refers to dwarf black slaves from Nubia presented by the King of Abyssinia to the Grand Seignior (*Voyage au Levant*, Lib. II. c. 68). See Bibl. Nos. 12 and 13.

[3] See Bibl. No. 148.

[4] See Bibl. Nos. 259 and 260.

[5] See de Quatrefages, *The Pygmies*, London, 1895; also R. B. Bean, *American Anthropologist*, Vol. XII. p. 220, and D. P. Barrows, *Ibid.* Vol. XII. p. 358, 1910.

[6] See Bibl. Nos. 308, 309 and 328.

Mountains, cretinism seems liable to be confused with other dwarf conditions. There are certainly cretins in the Pyrenees, but it is stated that there are other dwarfs as well. Some confusion in this respect appears to have been handed on from very early times. Homer and Ctesias certainly had ethnic dwarfs in mind, and Milton also when in *Paradise Lost*, Book I. lines 575 and 576, he refers to "that small infantry, warred on by cranes"; Milton had undoubtedly Homer's and probably Philostratus' words about a race of pygmies in his thoughts. The Homeric incident[1] has been represented by an ancient Greek artist, and this illustration is reproduced by Garnier in *Les Nains et Géants*, p. 3; the dwarfs are undoubtedly achondroplasic. Though this merely implies that the artist took as his model some dwarf with which he was familiar—which would most probably be achondroplasic—it appears to illustrate the possibility of confusion arising between racial and pathological dwarfism. He might have chosen instead, as a model, an ateleiotic or other dwarf without the marked deformity of achondroplasia, and in that case it would have been difficult to be sure that pathological and not racial dwarfism was represented in this picture. Dwarf races, as they are presumably liable to the same diseases as other races, may possibly have their own instances of pathological dwarf growth.

It has thus only been within comparatively recent times, as a result of exploration of regions hitherto untraversed, and of the increased knowledge of anthropology and ethnology, to a large extent resulting therefrom, that the facts with regard to dwarf races became known and the truth of some old legends was established. This with the increased knowledge of causes, nature and effects of disease has now rendered possible a general classification of dwarf growth, which is at least broadly accurate.

Historical Dwarfs. In order to illustrate the characteristics of dwarfs as individuals, a short account of historical dwarfs will perhaps be permitted, but for a full account the reader is referred to the works of I. Geoffroy-Saint-Hilaire[2], Wood[3], Gould and Pyle[4] and Garnier[5], from which the following accounts are largely taken. Amongst "historical" dwarfs several varieties may have been represented. It may perhaps be suspected that some of the dwarfs of the ancients were individuals of dwarf race captured in war, piracy or the slave trade; but others, such as the gladiator dwarfs of Domitian, portraits of which exist, were achondroplasic. In other instances there is no means of judging, exactly, to which group they belonged, though several varieties can in many cases with certainty be excluded (such *e.g.* as cretinism).

In the Bible[6] it is stated that no dwarf may officiate at the Altar (Lev. xxi. 16—20). In the Museum of Bulak there is an Egyptian statuette, discovered during excavations, of a dwarf named "Knoumhotpu[7]," chief steward of the linen, VIth Dynasty. The Egyptian gods Bes and Ptah-Sokar show a figure which is that of

[1] Some Homeric critics (see Paley, ed. *Iliad*, III. 3) consider the incident as an interpolation; it was familiar to Aristotle, but the legend may have come from Africa between Homer and Aristotle's time.

[2] See Bibl. No. 75, p. 140. [3] See Bibl. No. 138. [4] See Bibl. No. 332. [5] See Bibl. No. 205.

[6] "And the Lord spake unto Moses, saying, Speak unto Aaron, saying, Whosoever he be of thy seed in their generations that hath any blemish, let him not approach to offer the bread of his God. For whatsoever man he be that hath a blemish, he shall not approach: a blind man, or a lame, or he that hath a flat nose, or any thing superfluous, or a man that is brokenfooted, or brokenhanded, or crookbackt, *or a dwarf*, or that hath a blemish in his eye, or be scurvy, or scabbed," etc.

[7] Maspero, *Guide to the Cairo Museum*, Eng. edn., 1903, p. 473; reproduced, Porak, Bibl. No. 253.

dwarf growth. (See Plate S (14).) They are undoubtedly achondroplasic. Horus, the child-god, has by some been associated with infantile myxoedema. Denga dwarfs, who could dance "the dances of the gods," were brought from the Soudan, "the land of the gods," into Egypt. On certain vases such dances are depicted, and a royal rescript of about B.C. 3443 refers to a Deng who was a "dancer of god[1]."

During the period of the prosperity of Rome a dwarf was part of the establishment of every noble family. Julia, the daughter of Augustus Caesar, owned two dwarfs, Canopus and Andromeda, each 2 feet 4 inches in height. Mark Antony possessed a dwarf of stature, according to historians, of less than 2 feet, who, it is said, was given the appellation "Sisyphus" (see our p. 355, ftn.). There are authentic accounts of the dwarfs of the Emperors Tiberius, Domitian, Alexander Severus and Heliogabalus amongst others. The last-named Emperor caused marriages to be celebrated between the male and female dwarfs of his court. Some of the dwarfs of old were individuals renowned for wit and wisdom; for instance, Philetas[2] of Cos (the tutor of Ptolemy Philadelphus and a distinguished poet and grammarian), Alypius of Alexandria, a subtle dialectician (nicknamed "the pygmy," and said to be 17·5 inches in height!), Licinius Calvus, a celebrated rhetorician (of 3 feet) and Aesop, the author of the famous *Fables* (of about the same height) have been reputed dwarfs. The statue of the young dwarf Lucius, graven by order of Augustus Caesar, has been preserved until the present day[3]. The gladiator dwarfs of the Emperor Domitian are historical. A statuette represents Caracalla in caricature as an achondroplasic dwarf (see Plate S (14)).

Many other accounts of dwarfs have been given by ancient classical authors. Most of them were reported to be of stature from 3 feet 6 inches to 3 feet, or even less. Many such narratives are, however, possibly fabulous. It is said that in the period of deformity that occurred during the decline of the Roman Republic and the first three centuries of the Empire, a trade in dwarfs sprang up, and that the nefarious practice was followed of producing dwarf growth in well-formed infants by means of bandages and instruments designed to retard development. Prescriptions were employed with the same fearful object; such were inefficient feeding by design, with resulting rickets, dwarfing of growth and deformation of limbs. The inunction of the grease of bats, moles, dormice and other animals of the kind that were held to be "under a curse" throughout the "Dark Ages," was credited with the power of retarding growth and was, it is alleged, employed in this gruesome endeavour[4]. In the 16th century the fashion of dwarfs at Court after the manner of the ancients was revived. Catherine de Medici caused marriages to be celebrated between male and female dwarfs with the object of producing a dwarf race. Such marriages were, however, uniformly barren. Isabella d'Este (1474—1539) built special rooms for her dwarfs, and they may still be seen in the Corte Reale at Mantua (see Lugio, Buffoni, *Nuova Antologia*, 1891).

[1] Petrie, *History of Egypt*, 1903, p. 100. Cf. also Haliburton's excellent picture of dwarf "devil-dancers," Bibl. No. 318.
[2] Aelianus (*Varia Historia*, Lib. IX. c. 14) tells a tale that Philetas weighted his shoes with lead to prevent being blown off his feet in a high wind. Athenaeus (XII. 13) gives more credit to the tale than Aelianus.
[3] Geoffroy-Saint-Hilaire, Bibl. No. 75, p. 357. [4] See Bibl. No. 11.

Charles IX of France, in 1572, owned nine dwarfs: of these four were presented by King Sigismund Augustus of Poland and three by Maximilian III of Germany. This fashion continued until the 18th century. The dwarf had at that time, it would appear, succeeded to the office vacated by the Court jester and was allowed much freedom of speech. Christian II, of Denmark, was imprisoned in the castle of Sonderburg, and his faithful dwarf with him. According to Garnier, probably the last dwarf at the French Court was Balthazar, or Louis Pinson, who died in 1662[1]. In 1713, Princess Natalia, sister of Peter the Great of Russia, held a celebration in honour of the marriage of Valakoff, a favourite dwarf, with the dwarf of Princess Prascovia Theodorovna[2]. Such marriages were subsequently forbidden because of the difficulty and dangers of childbirth when pregnancy occurred. In England, France, Spain and other countries portraits of dwarfs were frequently painted by celebrated artists[3]. "D. Antonio l'Inglese" and "D. Sebastiano de Morra" (an achondroplasic dwarf of Philip IV) by Velasquez, are perhaps the most famous of these, but there are other portraits of dwarfs of the Spanish Court of his time by this master. One of these, "Las Meninas," shows the Infanta Margarita accompanied by the dwarfs Nicolasino Pertuseno and Maria Barbola. The latter and the dwarf called "el Primo" which is also in the Museum of the Prado are achondroplasic. Many dwarfs besides those enumerated have been famous as individuals. This would be not altogether remarkable, if we could assume relative brain weight to measure intelligence; thus in Schaaffhausen's dwarf (see Ateleiosis) the brain weight was one-nineteenth of the total weight as compared with one-thirtieth in the average adult individual of ordinary stature and growth; a somewhat similar relation has been found to occur in some other dwarfs examined post-mortem. Amongst famous men[4] who have been reputed to be dwarfs are Attila: our sole knowledge is due to Jornandes who describes him as "forma brevis, lato pectore, capite grandiore" (*De orig. Getarum*, see Bibl. No. 10); Characus, said to be of exceedingly small stature but one of the wisest counsellors of Saladin (Wood, Bibl. No. 138, p. 268, no reference given, but see Bibl. No. 27); Gregory of Tours (*fl.* c. 570), was described as a "homuncio" by St Odo (Waller, *Imp. Dict. Univ. Biog.*); Pepin, le Bref (*fl.* c. 750); Charles Durazzo, King of Naples (*fl.* c. 1380), was termed "the small" (*Nouv. Biog. Gén.* 1872); Prince Eugen (*fl.* c. 1700), nicknamed in France "Le petit Abbé"; the Duke d'Altamira, Marquis d'Astorga, president of the Spanish Junta (*fl.* c. 1808), reported by Lord Holland as the smallest man he had ever seen, smaller than many show dwarfs (*Foreign Reminiscences*, Lond. 1850, p. 146: see Bibl. No. 98); Wladislaus "Cubitalis" (1260—1333), King of Poland, called in Polish "Lokietek," the word *lokiec* = ell or cubit, of some renown for intelligence, courage and military qualities (*Nouv. Biog. Gén.* 1872); Godeau (1605—1672), he is said to have been refused in marriage on account of his "petitesse et sa laideur," but Richelieu created him Bishop of Grasse on account of his ability and success in affairs (*Nouv. Biog. Gén.* 1872 and Bibl.

[1] See Bibl. No. 205, p. 114.

[2] For an account of two marriages of Russian Court dwarfs at which 72 and 93 dwarfs respectively were present: see Bibl. No. 45, S. 524.

[3] An *Iconography* of Dwarfism, giving a list of famous pictures, prints, etc., containing dwarfs will be found below.

[4] The source of the current lists appears to be initially: *Curiosités biographiques. Bibliothèque de Poche.* Paris, 1846; but finally, probably, one of the dissertations of M. F. Quade's pupils—*e.g.* J. H. Wübbaer: *De viris, statura parvis, eruditione magnis.* Gryphiswaldiae, 1706—or the works cited therein. Editor.]

No. 45, p. 516). The German painter Jacob Lehnen (3 feet, 10 inches), the actors Moreau, Fleury and Garry, and William Hay[1], at one period in the 18th century M.P. for Sussex, were dwarfs. Pope, 4 feet 6 inches, and deformed, has been described by some writers as almost a dwarf. Varro has written of two Roman gentlemen, to whom also Pliny refers, of stature equal to two Roman cubits (about 3 feet) who from their decorations must have belonged to an equestrian order (Gould and Pyle). One of the most celebrated dwarfs of more modern times was Jeffrey Hudson, who figures prominently in *Peveril of the Peak* of Sir Walter Scott. Born in 1619 at Oakham in Rutlandshire of quite normal parents he was presented by the Duchess of Buckingham, at the age of 9 years, to Henrietta Maria, the wife of King Charles I. His height was then scarcely 18 inches. At the age of 30 years he had attained a stature of 3 feet 9 inches. This dwarf was endowed with considerable ability and was sent to France to obtain a midwife for the first accouchement of the Queen. He served as a captain in the Royalist army during the Civil War, and accompanied the Queen to France on her banishment. Amongst other accounts of his actions is that of a quarrel with a gentleman named Crofts. To the resulting duel the latter came armed with a squirt; a second meeting having been arranged with pistols on horseback, Crofts was slain on the first discharge. Jeffrey Hudson in 1679 was sent to the gatehouse Westminster, charged with complicity in the popish plot. He died in receipt of a royal pension at the age of 63 years. His portrait[2] is at Hampton Court and at Buckingham Palace, and an engraving is in the Print Room of the British Museum. (Fuller details are to be found in the *Dictionary of National Biography.*) About the same time there lived (1615—1690) at the Court of King Charles I a dwarf named Richard Gibson, of very diminutive size, 3 feet 10 inches. He was a celebrated miniature painter and subsequently became the drawing-master of the Princesses Mary and Anne, daughters of James II. He married Anne Shephard, a dwarf of Queen Henrietta Maria. Gibson lived to the age of 75 years and his wife to 89 years. There were nine children, five of whom lived to maturity and were of normal growth, by this marriage. A head of Gibson drawn by himself is in the Print Room of the British Museum, and an engraving of miniatures of Gibson and his wife is in the edition of Walpole's *Anecdotes of Painting,* issued in London, 1849 : see Bibl. No. 97.

Other well-known dwarfs were :—Wybrand Lolkes, son of fisher-folk at Jelst, Holland, a skilful jeweller. He amassed a large fortune by exhibiting himself in Holland and England, and died in 1800, aged 70 years. (He has been classed by Regnault as achondroplasic.) Nicholas Ferry, also known as Bébé, was born in the Plain of the Vosges in 1741. His height was 29 inches. He was presented at the age of 5 years to King Stanislas of Poland. He was mentally deficient. He died at the age of 22 years. His family and personal history and the photograph of his skeleton are given under Ateleiosis. (See Plate Z (39) and (40), and for further details Buffon and I. Geoffroy-Saint-Hilaire[3].) Joseph Boruwlaski was a dwarf very well known in his time, of whom the Comte de Tressan and Caroline Hutton[4] have

[1] See Bibl. No. 23[b]. Hay was probably deformed, not a real dwarf. EDITOR.]

[2] A portrait of Jeffrey Hudson, dwarf of Henrietta Maria, painted by a Dutch artist has recently been added to the National Portrait Gallery: see our Plate HH (66).

[3] Bibl. Nos. 33 and 75, T. I. p. 140.

[4] See Bibl. Nos. 28 and 88.

written accounts and whose autobiography exists[1]. Born in Poland in 1739, he died in England aged 98 years. He was a good linguist and above the average in intelligence. His family and personal history are given under Ateleiosis. We reproduce Bonomi's excellent cast taken from this dwarf while alive: see Plate II (67) and (68). Mary Jones of Shropshire was of height 32 inches. She died in 1773, aged 100 years. Richebourg, a servant of the House of Orleans and afterwards one of its pensioners, was of height 23 inches. He died in 1858, aged 90 years. During the French Revolution he passed in and out of Paris disguised as an infant in its nurse's arms, and so was enabled to convey important information to his friends within and without the city (Garnier). Nannette Stocker who died at Birmingham in the 18th century, aged 39 years, was said to be "the smallest woman in the kingdom and one of the most accomplished." Born at Kammer in Northern Austria, her height was 33 inches, growth having ceased at the age of 4 years. "She was a good pianist and otherwise accomplished." See Plate II (69). Robert Skinner was of height 0·43 m. and his wife Judith about the same. They were exhibited in London in 1843 and made a small fortune. In twenty-three years there were fourteen robust and well-formed children of this marriage. (Gould and Pyle[2].) Another most remarkable example of dwarf growth was Carrie Akers. Her height was only 2 feet 10 inches, but she weighed, it is stated, 309 pounds (22 stone 1 pound)! Her portrait is shown in Gould and Pyle's book. She does not appear from this to belong to any of the commoner varieties, and appears to be almost unique[3]. "General Tom Thumb" (Charles Stratton, born 1832) was another well-known dwarf. He ceased to grow at the age of 5 months and his height was less than 21 inches. With him were associated, in his professional career, three other dwarf colleagues, "The Sisters Warren" and "Commodore Nutt." "General Tom Thumb" gave his hand in marriage to one of the sisters, and it is said that she bore a child of normal proportions, which, however, died in infancy. Other well-known dwarfs seen in exhibitions in modern times were "General and Mrs Mite," of the U.S.A., and "General and Mrs Small" (a dwarf named Morris of Blaenavon, North Wales, and his wife). Morris's height was 35 inches. His wife was still smaller. She was the mother of twins of normal growth in 1895. (Gould and Pyle.) The Russian dwarf, Dantlow, considerably antecedent in point of time to these, showed dwarf growth associated with congenital deformities[4]. This dwarf showed evidence of rickets in his limbs (all that he had) and vertebral column. He had only four toes on each foot, and the arms were almost entirely lacking. At the age of 30 years, however, he was "of a pleasant appearance" and "of a happy disposition." "He feeds himself with his left foot and writes a round hand, clearly legible, with the same member, both in Latin and in Russian." In the same journal another Russian dwarf is, it is reported, described by an anonymous author (account not found). He, it is stated, was elected by the Empress of Russia to the Academy of Arts. Other dwarfs (classed as achondroplasic by Regnault[5], see Achondroplasia) were: Owen Farrel, "the Irish dwarf," of height

[1] See Bibl. No. 43.
[2] See Bibl. No. 332.
[3] Cf., however, Iconography No. 63 and the dwarf "Barbino" in the section on Statuary. EDITOR.]
[4] See Bibl. No. 34.
[5] See Bibl. No. 411.

1·137 m. (4 feet 5·9 ins., Hunter had one of his femora which measured 9·5 ins.), very well known in his time, he died in 1742; he was noted for feats of strength and endurance much above the average; his portrait is in the Royal College of Surgeons and in the Print Room of the British Museum: see Plate KK (74). Wybrand Lolkes, already referred to, of height 0·648 m., who died in the year 1800, aged 70 years. Tom Pouce, who died in the year 1843. The dwarf of Broca[1] (1877). Balthazar Zimmermann of de Quatrefages[2] (1881), and the dwarf of Sabudini (1887)[3].

There are many other accounts of dwarfs besides those referred to in the above short summary. But this seems enough to indicate that these individuals are sometimes capable of achievements above the average, though this is exceptional and is probably confined to three or four classes, the achondroplasic, the rickety dwarfs and a few examples of the class "ateleiosis," "true dwarf growth" ("echter Zwergwuchs," "nanisme vrai"). To these may perhaps be added the very undersized individuals, referred to under the heading of racial dwarfs, in which the query arises whether they have a racial or a pathological cause. Nearly all the other varieties are, from their nature, deficient: *e.g.* cretinism, etc.

There has recently, 1909 to 1910, been an exhibition of some fifty-three dwarfs[4] of both sexes at Olympia, in London: see Plates Q, CC, DD, EE and FF. In addition to holding stalls, where commodities of various kinds were sold, there were a variety theatre and a circus in this exhibition. The performance was of much the usual type—in miniature. Some of the ponies in the circus seemed to show the same condition of ateleiosis (*i.e.* disease) as did the human dwarfs, while others were racial dwarfs (*e.g.* Shetland). Many varieties of acrobatic feats were to be seen, *e.g.* that of the lady who manœuvred the rolling ball upon an inclined plain, and of the gymnast who stood in an inverted position upon his hands on the upraised soles of the feet of a colleague; there were also the humorist, the vaulting equestrian athlete and the dwarf representative of Hercules. The last, aged 23 years, weighed 40 pounds. He supported by "the bridge" performance a weight of between 280 and 300 pounds (two men of above medium weight), a feat which in proportion to weight seems to compare not altogether unfavourably with that of many other "strong" men. He had the general appearance of a child of 10 years, and the musculature in its lineaments showed the form of that of childhood. Most of these performances, though remarkable, conveyed the impression of automatism, like the tricks of performing animals, and seemed to lack spontaneity and to need the presence of full-grown assistants for their performance. But this was not the case with some of the equestrian feats by one of the larger dwarfs; this dwarf was probably rickety.

In the Print Room of the British Museum there are prints ranging in date from about the year 1620 to 1860. These show many notable and notorious individuals of their day, such as noted pugilists and other athletes, giants, eccentric individuals and human curiosities, etc. Amongst these prints are those of the following dwarfs: (1) Jeffrey Hudson, already referred to, ateleiotic. (2) "The

[1] See Bibl. No. 169. [2] See Bibl. No. 187 [probably myxoedematous. Editor.] [3] See Bibl. No. 233.
[4] At a banquet of Cardinal Vitelli in the 16th century, *thirty-four* dwarfs are said to have waited on the guests: Blaise de Vigenère, cited by Garnier, Bibl. No. 205, p. 106. Cf. also ftn. 2 on p. 359 above.

wonderful and surprising English dwarf, 2 feet 8 inches high. Born at Salisbury, 1709. Has been shown to most of the Nobility and Gentry of Great Britain." This dwarf, a female, appears to be ateleiotic, but the print is somewhat crude and bears no name, and the features of the condition, whatever it may have been, are not well shown. (3) Owen Farrel, already referred to, achondroplasic. (4) "Mr Coan, the Norfolk Dwarf, 3 foot high. Died aged 36 years"; "with Mr Bamfield, the Staffordshire giant, 7 foot 4 inches high." Date 1771. This dwarf appears to have been ateleiotic. (5) "John Tarr, aged 57 years. Height 4 foot 3 inches. Born in the village of Bampton. For fifteen years ostler at the White Horse. For twelve years, boots at the Three Pans, Tiverton." Period 1787 to 1830. He appears to have been achondroplasic. (6) Andrew Whitson—a crippled dwarf concerning whom no details are given, but he looks like a case of birth palsy and was probably mentally deficient. (7) A Show Bill representing "Richard Garnsey, 'The Miniature John Bull'—Born at Kittsford near Taunton in 1831. Height at the age of 16 years was 34 inches. Has not grown since he was four years old. 'The miniature John Bull' is pronounced by Sir James Clark, M.D., Dr Locock, Dr Fergusson and Dr Dalrymple to be the most symmetrical dwarf in the world." "Exhibited before the Society of British Artists." This appears to have been a case of ateleiosis. (8) An individual named George Trout, "one of the Porters to the Honourable the House of Commons," no date given, but about 1840—60, was probably achondroplasic. In a print recently on view in London, a Persian dwarf of the 18th century was shown, who was stated to have possessed great strength, lifting large stones with his hair, and who spoke seventeen languages[1].

There was at the Savoy Hotel, London, December, 1910, a Hungarian dwarf named Mademoiselle Anita. She was said to be aged 25 and 25 inches tall. She spoke English, French, German and Hungarian, and was said to be perfectly formed. According to the daily papers she was engaged to be married and had come to London for the purpose of buying her trousseau and arranging an insurance for £50,000 on her life. She has since been on exhibition in the provinces. She is probably ateleiotic. We give a portrait of her on Plate HH (65).

II. CLASSIFICATION OF VARIETIES OF DWARF GROWTH[2].

The term "dwarf" is a general one applied to all individuals who are markedly smaller than the average. The influences which may produce this effect, as well as the anatomical conditions that occur, are many. Methods of examination were for long confined to measurement. More recently, however, cases have been investigated anatomically, and it has been shown that many different abnormal processes may cause the defect. Investigations by radiography, by which means alone diagnosis of some of these bone conditions in adult life has been rendered possible, have enabled notable advances to be made. The relationships of the different varieties of dwarf

[1] This is probably the dwarf exhibited in 1740: see *Daily Advertiser*, Aug. 18, 1740, and Bibl. No. 138, p. 310.

[2] [For this discussion (pp. 363—370) the writer, with Professor Sternberg's consent, is indebted, materially and literally, to *Nothnagels Specielle Pathologie und Therapie*, Wien, 1899, Band VII. 2 Halfte: see Bibl. No. 364. EDITOR.]

growth to one another have now been shown, chiefly by the researches of Virchow, Kundrat, Paltauf, Kaufmann, Porak, and others. The condition in dead born infants, formerly known as "foetal rickets," "micromelia," etc., and the relation of defective growth to disturbances in function of the thyroid gland, have now in part been explained. The relation of the condition known as "infantilism" to dwarf growth has also been determined.

Following the classification of Kundrat[1], growth depends upon the following factors :—(1) Inherited disposition or tendency; (2) Nutrition; (3) Conditions of life—surroundings or environment; (4) Habits of life. Pathological influences may affect :—(1) The inherited disposition; (2) Intra-uterine development; or (3) Growth in infancy and childhood. In cases of dwarf growth it is not yet possible in all cases to locate exactly cause and effect, but a general view of the condition as a whole has been attained, so that from many varieties several natural groups can be formed.

A. General Classification of Varieties.

Group I. Cases which may possibly be caused by either (1) changes of conditions (environment) within physiological limits[2], or, as seems more probable, by (2) inherited tendency alone; but, at least in some instances, possibly by both causes working together. Under (1) would be such factors as : (*a*) deficient or unsuitable food, especially insufficiency of albuminous content; (*b*) cold; (*c*) excessive and long-continued overexertion, beginning in early childhood and continued throughout life. To this Group I would belong the real dwarf races referred to, "Akkas" or other negrillo races (the Bushman of South Africa, *e.g.*), and relatively small peoples such as Andamanese, Eskimos, Laplanders, Gurkhas, Japanese, etc., and the individuals of exceedingly stunted growth seen in European cities, the victims of want and misery (whether as cause of dwarf growth or effect of it).

Group II. Cases in which, it is supposed, the protoplasm has a special tendency to defective development. This condition affects individuals, or it may be stocks, but not the race. To this group belong cases of general hypoplasia, found in one or more individuals of a family, side by side with well-grown brothers and sisters, in twin children, or in cases of multiple births, and the defective growth that sometimes occurs in hereditary syphilis and is said to occur in the children of drunkards, or of those affected by plumbism, other metallic poisons or intoxicants, and here belong, *perhaps*, cases of ateleiosis or true dwarf growth.

Group III. In this group, which to a large extent merges with the last, the influence adverse to growth is brought to bear during intra-uterine life. Examples : the dwarf growth of microcephaly, porencephaly, etc., and the defective growth sometimes seen in children born before full term. In all these cases, dwarf growth is by no means invariable; and they merge largely with those of Group II.

[1] See Bibl. No. 267.

[2] This factor seems to be an improbable one. It is common to find a dwarf race and another race side by side under much the same conditions, *e.g.* "Bushman" and "Kaffir," "Akka" and "Masai." Again, amongst the Japanese, if accounts may be trusted, it is, on the whole, amongst the highest classes of the race that the smallest stature is observed; but the environment of these must be supposed to be the most advantageous. It seems more reasonable to suppose that heredity alone is the cause of the particular average stature in these cases.

Group IV. Cases in which the adverse influence has been exerted during childhood. Here development at first apparently normal has, after an illness, ceased to advance at the usual rate. This has been observed in acquired hydrocephalus, chronic meningitis, pneumonitis, malaria cachexia, granular kidney of childhood, congenital heart disease, acquired heart disease with valvular failure early in life, in other disorders of the circulatory system, and the chronic alcoholism of childhood, and disorders of suprarenal body, pancreas and intestines. Trauma has been regarded as an occasional cause, and Rohrer's case[1] is quoted as an example, but the case was very insufficiently described and the recorded syphilis of the father must be taken into consideration (Sternberg).

Group V. This group is formed by cases in which, owing to absence, disease or operative removal of the thyroid gland, development has ceased. (True cretinism and infantile myxoedema and surgical cachexia strumipriva occurring in childhood.) "Besides cases such as these there seems to be other evidence of the influence that the thyroid gland exerts upon development, gained from clinical experience of the effects of thyroid extract upon dwarf growth in cases other than the last" (Sternberg).

Group VI. In this group are placed general disturbances of growth which show some affinities with the conditions of the last group, and have sometimes been grouped with them. Such are: rickets, the so-called "real dwarf growth" or "ateleiosis" ("echter Zwergwuchs" or "nanisme vrai")—in which epiphyses and diaphyses remain ununited throughout life, as in the thyroid gland group; and the dwarf growth of defective development and premature junction of epiphyses to diaphyses—"achondroplasia" (Parrot[2]) or "chondrodystrophia, dystrophia hypoplastica or foetalis" (Kaufmann[3]). "The conditions included under this head are really heterologous, but cannot at present be further classified aetiologically and even from those of the thyroid gland group they cannot, aetiologically, be definitely marked off at present" (Sternberg). Compare Plates Y, JJ (71), LL—PP.

B. Pathological Anatomy of Varieties of Dwarf Growth compared.

Kundrat, who was the first to deal with this aspect of dwarf growth comprehensively, differentiated two varieties of changes: (1) Quantitative. Here the processes of ossification are normal, as far as they go, but are retarded. (2) Qualitative. Here these processes are altered by disease. To this group belong Groups V and VI of the last heading. Though this is a useful general classification it must be largely modified when applied to individual cases, and not only the changes that take place in the skeleton but the time at which these occur must be considered.

Varieties of Dwarf Growth.

I. Dwarf growth, the result of insufficient development and premature ossification and union of epiphyses with diaphyses ("Chondro-dystrophia hypoplastica," Kaufmann; "Achondroplasia," Parrot). The individuals of this class have short

[1] See Bibl. No. 222. [2] See Bibl. Nos. 161 and 172. [3] See Bibl. 279.

arms and legs, relatively to the size of the trunk, which is of approximately normal growth. This shortness of extremities is indicated by the term "micromelia" ("little limbs"). This term, however, expresses nothing of the underlying cause, and "micromelia" may arise in more than one way, either as a result of diminished growth or of other disturbances in the region of growth of bones. See Plates U and Y illustrative of rickets and achondroplasia and the fuller treatment below under Achondroplasia.

II. *True Dwarf Growth.* (Kundrat and Paltauf[1].) Ateleiosis (Hastings Gilford[2]). These dwarfs are to be recognised by the fact that the cartilage discs persist throughout life. This can be shown by X-ray examination during life or in the skeleton after death. The epiphyses of the bones of the extremities, if present, do not join the diaphyses, but are separated from them, as in infancy, by cartilage, or are, at most, united to them by thin bridges of bony tissue. In many bones the epiphyses do not appear at all, being represented by cartilage, as in the infant. The skeleton in general is weak, but it shows no such disproportion in length between the trunk and extremities as occurs in the first group. Individual centres of ossification, as those on the spinous and transverse processes of the vertebrae, or the trochlear nucleus of the humerus, which normally appear late may be entirely lacking. The vertebral bodies are depressed. The sternum remains throughout life as several pieces of bony tissue united by cartilage. In the pelvis the tri-radiate cartilage of conjugation persists throughout life. The face is often "cretin-like," or for this appearance Sternberg suggests as a better term "infantile." This peculiarity is caused by the broad saddle-shaped depression of the bridge of the nose associated with a skull of brachycephalic form. The form of the nose depends upon defective development of the bones of the base of the skull. The defect is not one of premature union by bone, as in achondroplasia, but is produced, as in cretinism, by lack of growth of bone, the cartilage of conjugation persisting throughout life. Dentition is delayed and the milk teeth are not unusually retained until late in life. The genital organs are little developed, they are often in an infantile condition. For the most part the secondary sex characteristics, such as hair about the face, pubes and axillae, are also wanting. In the female the pelvis and mammary glands are incompletely developed. Such individuals are usually sterile. The face retains through life an infantile or "puerile" appearance. This "puerile" condition, which may affect females as well as males, sometimes produces in the former a somewhat masculine appearance. The genital organs may remain in the early foetal condition. Schaaffhausen found bilateral cryptorchism in his first case. Incomplete descent of the testes is usual and is usually bilateral. The intelligence is slightly defective as a rule. This is shown by a childish, shy or timid disposition and lack of self-reliance. The visual organs are almost uniformly normal in this class of dwarfs.

III. *Dwarf Growth associated with Lesions of the Thyroid Gland.* These conditions need only be outlined here for comparison with and distinction from other groups. Typical cases of the last group (the "real dwarfs") are easily recognisable. In certain cases, however, it is difficult to draw a sharp line between them and cases

[1] See Bibl. No. 262. [2] See Bibl. 403.

of disturbed bone growth of the present group due to thyroid gland lesions. (*a*) On the one hand the skeleton in the latter cases presents just the same diminution of its development as in the real dwarfs, and the cartilage of conjugation persists, the epiphyses remaining ununited to the diaphyses throughout life. (This occurs in operative removal of the gland before growth is complete, in infantile myxoedema and in real cretinism.) According to Sternberg, Paltauf has erroneously included several of these cases amongst the "true dwarfs" described by him, but at the time at which he wrote the functions of the thyroid gland and its influence upon growth were not understood as they have since become. (*b*) On the other hand the "true" dwarfs (ateleiosis) present a number of features by which they seem to be allied to myxoedema. Such are the peculiar conformation of the skull, which is the same as is seen in cretins, and the "cretinoid" facial appearance. Defective development of the genital organs and the "puerile habitus" are other features common to both, and in the "true dwarfs" the defect of intelligence occasionally goes as far as idiocy. In some of them the thyroid gland seems to be abnormal. The two groups, infantile myxoedema or cretinism, and "real dwarfism" ("echter Zwergwuchs"; "nanisme vrai") or ateleiosis, seem to merge into one another, and it is not impossible that they have relations in common, though what these are does not definitely appear, in the present state of knowledge. "Brissaud[1] has classified in one group that of myxoedema, the 'true dwarfs,' and 'infantilism.' He considers all to be cases of myxoedema in which the symptoms usually associated with it are little marked, and terms the condition 'myxoedeme fruste.' In the present state of knowledge this view cannot be refuted and must stand until more is known of these conditions[2]."

IV. *Rachitic Dwarfs.* Rickets is the commonest cause of dwarf growth (Sternberg), and many exhibition dwarfs have been rickety. These individuals often show deformities, and in many cases the diagnosis can be made at a glance; in other cases a thorough examination is necessary to establish it. Upon the subject of X-ray investigations opinions differ. Sternberg considers radiography to be of little value in distinguishing rickets from other conditions. Other authorities, notably Porak[3], hold an opposite view.

As this paper is only concerned with the varieties of pathological dwarf growth, which at the present time show undoubted evidence of heredity in causation, rickety and cretinoid dwarf growths are only referred to in order to show their relationships to these other varieties. See Plates GG and NN.

Dentition. The germination and eruption of the teeth are liable to be disturbed in all varieties of dwarf growth except that of achondroplasia. Persistence of the milk dentition and delayed eruption of the last molar are common in the "true dwarfism" of Kundrat and Paltauf (ateleiosis of Hastings Gilford), and in cretinism, microcephaly, etc.

Age of Onset. The age at which "dwarf growth" begins, or at which growth becomes retarded, varies in the different conditions and in different cases of these. Certain dwarfs, such as the achondroplasic variety, enter the world as such (though

[1] See Bibl. No. 334. [2] See Sternberg, Bibl. No. 364. [3] See Bibl. 491.

some observers state that the condition may be acquired after birth). The diagnosis can with those exceptions be made at birth. The study of achondroplasia was begun in new-born infants, mostly still-born, whose peculiar shape had attracted attention; and a good deal of the knowledge of the anatomy of the condition was gained from these before the identity of the condition with that of the short-limbed dwarf adults, familiar from remote times, was realised. In other cases only the history can determine this point; growth in normal children is not continuous in the various segments, first one part then another is advanced[1]. It may be impossible to determine the time of onset exactly. In some cases of "true dwarf growth" it is clear that the influence, whatever this may be, was brought to bear *in utero*. [Such a case is that of Caroline Crachami, "the Sicilian dwarf," whose skeleton is in the Museum of the Royal College of Surgeons (Sir Everard Home[2] and Hastings Gilford[3]). Bébé (Buffon and Geoffroy-Saint-Hilaire[4]) may perhaps be another.] In other cases the history given is that these individuals were of about ordinary size at birth, but ceased to develop at the ordinary rate in the early months or years of life. In other cases cessation of growth occurred in relatively later years, up to the time of puberty. For these facts numerous old but well authenticated accounts speak (for instance those by I. Geoffroy-Saint-Hilaire) and many modern ones (accounts by Hastings Gilford, Sternberg and others). The same observers, as well as Schaaffhausen, Paltauf and others, have shown that in a number of cases growth is merely retarded to a minimum, but very slowly continues until quite late in life (up to the 30th year or later). In other cases growth, having apparently ceased for years, may recur and slowly continue until still later in life; or in such a case there may be in about the same short period of time a relatively large increase in growth. This peculiarity of course only affects the varieties of dwarf growth in which the epiphyses remain ununited to the diaphyses through life.

Duration of Life. This is relatively short; comparatively few reach old age. A reference to the account of individual dwarfs given in the introduction will, however, show that such ages as 100 years, 90 years, 80 years and 75 years have been attained, and many have reached the sixth decade.

The Prognosis of Disease, with the exception of achondroplasic and rickety dwarfs, is to be made guardedly, relatively to that for other individuals; "dwarfs present, for the most part, pathological bodily conditions and have slight powers of resistance" (Sternberg).

Infantilism[5].

Since many varieties of dwarf growth show the condition known as "infantilism" or "the puerile habitus," the condition seems to require definition. This occurs, either occasionally or constantly, in all varieties of dwarf growth except achondroplasia[6], but its occurrence is not confined to dwarf growth, and, as stated, it

[1] Humphry: *On the Skeleton*, Cambridge, 1858, pp. 97 *et seq.*

[2] See Bibl. No. 58, Vol. v. p. 191, Ed. 1828. [3] See Bibl. No. 403, p. 305. [4] See Bibl. Nos. 33 and 75.

[5] The mode of classification here followed is that of Sternberg (*loc. supra cit.*) and of Thomson (*Allbutt's System of Medicine*, 2nd edition, Vol. IV. Part I. p. 486). [An interesting account of the views on Infantilism of Lasègue, Lorain, Ausset, Breton and Hutinel will be found in *Archives de Médecine des Enfants*, T. v. pp. 488—90, Paris, 1902; but see especially Ettore Levi's important memoir of 1908, Bibl. No. 588 (with bibliography). EDITOR.]

[6] Achondroplasia, like other morbid conditions, may be associated, in the same individual, with others, *e.g.* mental deficiency (as in a family shown in the pedigrees), and with some of these "infantilism" may be associated.

does not occur in all varieties or cases of that condition. The explanation of "infantilism" seems to be as follows: When an individual has ceased to be a child and has become an adult this fact is shown, not by increase of stature and power, but by maturity of the genital organs, *i.e.* by acquisition of "the primary sexual characteristics" of adult life. With these "the secondary sexual characteristics," bodily and mental, are acquired; the outlines of the figure become more marked and definite, it becomes that of a man or of a woman rather than of a child; in the male the larynx enlarges and the voice changes; in the female the breasts enlarge; hair appears on the pubes and axillae and in the male also upon the face; the epiphyses of long bones join the shafts and growth of these in length practically stops; the mental outlook changes. By the term "infantilism," devised by Lasègue, is implied the maintenance of the genital organs in the infantile state of development with a lack of secondary sex characteristics, *i.e.* there is failure of the primary and secondary sex characteristics to appear at the proper time; and this may occur in individuals of dwarf growth, in those of ordinary growth, or in giants. Any of these may show "infantilism." This term denotes, therefore, merely a group of symptoms and no more. In many cases individuals thus affected, of male sex, show some resemblances to women, and females of this kind somewhat resemble males; hence the synonymous terms "Masculism" or "Androgynism" and "Feminism" have arisen (Meige: Bibl. No. 306). "Infantilism" may be associated with disturbances of growth of various kinds and seems to have a cause in common with them. It occurs in the following conditions: (I) In the dwarfing of growth which results from intoxications such as (1) chronic tuberculosis[1], *e.g.* hydrocephalus and spinal caries; (2) congenital syphilis (Fournier[2]); (3) malaria cachexia (Borelli[3]); (4) leprosy, pellagra and some of the commoner infective diseases (Lancereaux); (5) in very severe rickets (Sternberg); (6) chronic alcoholism of childhood (Sternberg); (7) prolonged and excessive doses of such materials as mercury, lead, morphine, tobacco and bisulphide of carbon (Thomson). (II) In the abnormalities of growth which seem to depend upon gross lesions or defects of important internal organs such as: (1) thyroid gland; examples, cretinism and infantile myxoedema; (2) pancreas (Bramwell[4], etc.); (3) liver, hypertrophic biliary cirrhosis (Lereboullet[5]); (4) suprarenal body (Morlat[6]); (5) kidney, *e.g.* congenital granular kidney (Stephen Mackenzie, Schorstein, and others); (6) pituitary body, as in gigantism and acromegaly and other apparently allied conditions (Sternberg, Ferrier). (III) In disturbances of growth associated with deficiencies of the circulatory system, either congenital or beginning very early in life, such as congenital stenosis of the aorta and stenosis of the mitral and aortic valves of the heart, and general defects of the circulatory system. (IV) In

[1] As an example of dwarf growth produced by the influence of the tuberculous toxin, the small stature sometimes seen in cases of tuberculous disease of the spine (Pott's Disease) is quoted. Individuals thus affected show in their proportions the converse of achondroplasia. That is, they have long limbs and a short trunk and a head of ordinary size. Such individuals form broadly two groups of cases: (1) Those in which, in adult age, the limbs are of the average length and the head is of average size; they owe their defective stature to the shortness of trunk produced by their spinal disease and resultant deformity. Such cases are those, for the most part, in which the ouset of disease occurred relatively late in childhood. They are not, properly speaking, dwarfs at all, though they may show a stature sufficiently small to be classed as such. (2) Those in which, as well as the spinal deformity and shortening of trunk relatively to length of limbs, the growth of all parts is retarded to a minimum by the influence of the tuberculous toxin during the period of growth. Such cases are, for the most part, those in whom the onset of disease occurs in relatively early life. Though they present the same relative proportions as (1), they are dwarfs strictly speaking, and it is in them more particularly that "infantilism" occurs.

[2] See Bibl. No. 228. [3] See Bibl. No. 190. [4] See Bibl. Nos. 458 and 481.

[5] See Bibl. No. 431. [6] See Bibl. No. 460.

conditions such as congenital adiposis, progressive muscular dystrophies (Sternberg), in the dwarfing of growth that occurs with microcephaly, spastic diplegia and in some cases of the dwarf growth of obscure origin called "ateleiosis," or "true dwarf growth" already referred to and, occasionally, in individuals of ordinary height who present no other peculiarity.

"Infantilism" is not invariably present in the above conditions. For instance, of two ateleiotic dwarfs, brothers, of the same height, who show otherwise the same features exactly, one shows "infantilism" and the other does not. With "infantilism" there is usually associated defective function of the sexual glands, but this does not appear to be either invariable or, when present, always complete. Apart from such conditions as cretinism (amenable to treatment and in which treatment seems to improve all symptoms, including "infantilism," equally) there are grounds for supposing that occasionally as age advances "infantilism" becomes less marked or tends to pass away.

III. ACHONDROPLASIA[1].

A. Introduction.

In some instances achondroplasia shows the influence of heredity in its causations. It is with this aspect of the subject that this paper is concerned; but in order that the mode of working of its heredity be understood, a short description of achondroplasia seems desirable. Numerous achondroplasic dwarfs have already been referred to. That achondroplasia is not a "new disease" has been proved by Parrot[2], Pierre Marie[3], Porak and Durante[4], and Regnault[5], who have shown that it has been represented in the statuary and pictures of ancient artists. The figures shown in the illustrations on Plate S (14) of the Egyptian gods[6] Ptah-Sokar and Bes and the caricature statuette of Caracalla, as well as existing representations of the gladiator dwarfs of Domitian, show this[7]. In the ancient Greek picture, reproduced by Garnier in his book[8], the dwarfs are obviously achondroplasic. According to Porak and Durante (*loc. supra cit.*) many of the clowns of courts, of grotesque figure but of marked intelligence, have probably been of the same nature. Regnault believes that Owen Farrel (see Plate KK (74)), Wybrand Lolkes, Tom Pouce, Broca's dwarf and Sabudini's dwarf were achondroplasic. Reference has already been made to portraits of achondroplasic dwarfs by great masters. It seems on the whole probable that famous men of action, who were of dwarf stature, were either rickety or achondroplasic; for instance, Attila, King of the Huns, Prince Eugen, Wladislaus Cubitalis of Poland and the Spanish Admiral Gravina. But details of their proportions are not precise, and therefore their nature can only be surmised. At the present day achondroplasic dwarfs are not infrequently seen playing clowns' parts at fairs and circuses; *e.g.* two

[1] [Pp. 370—384 are largely translation and adaptation of the classical memoir (see Bibl. No. 491) of Porak and Durante, whose consent to this use of their work has been most generously given. Editor.]

[2] See Bibl. No. 172. [3] See Bibl. No. 371. [4] See Bibl. No. 491. [5] See Bibl. No. 411.

[6] The humerus of an achondroplasic dwarf was found in the tomb of King Zer (1st Dynasty), also a stele near the tomb with a very achondroplasic looking figure. See Petrie: *Royal Tombs of Earliest Dynasties*, 1901, Part II., Plates VI. A. 14 and XXVIII. 58. Two steles of dwarfs, with skeleton of one and bones of another dwarf, were obtained by Petrie from the tomb of Mersekha-Semempses, 1st Dynasty; see *Royal Tombs of the First Dynasty*, 1900, Part I. pp. 13, 27 and Plate XXXV.

[7] See Note A, p. 386, on early achondroplasic figures. [8] See Bibl. No. 205, p. 3.

of Apert's cases[1] were circus clowns or "eccentric comic artists" as they describe themselves. One of the individuals, whose pedigree is described below, was a music-hall artist of the humorous type. Photographs are also shown (Plate R (11)—(13)) of a Chinaman, a native of Hankow, 500 miles up the river Yang-tse-kiang, of achondroplasic proportions, who was discovered at Shanghai by Dr Gordon Moir, Surgeon of the Royal Navy[2], earning a livelihood by dancing and buffoonery. Another Chinese case, that of Li, is published by Molodenkoff[3]; he is a nomadic conjuror and juggler.

The congenital bony "dystrophies," supposed to arise more or less early in the development of the embryo, were for long all confused under the name "rickets." Gradually, however, within recent years, the differences of some of these conditions from rickets and from one another have been made out. Some of them show, amongst other characters, shortness of limbs (micromelia). The term micromelia, however, expresses nothing of the pathology, and micromelia occurs in several conditions, which differ from one another clinically as well as in their pathological anatomy. Amongst these are true achondroplasia and "the periosteal dystrophy" (Porak and Durante[4]), the latter was formerly included with the former under the name achondroplasia. Some of the congenital bony dystrophies (cleido-cranial dysostosis, congenital rickets and congenital syphilis) and various kinds of dwarf growth need no further discussion here; others, such as congenital osteoporosis or congenital fragilitas ossium, and congenital malformations (discussed elsewhere in *The Treasury*), belong to a different group, and need also only be mentioned here. Chaussier[5] (1819), Romberg[6] (1817), M. J. Weber[7] (1829), Busch[8] (1836), and Dumenil[9] (1857) described foetuses with short but thick limbs, which they distinguished from rickets, but did not know how to name. Depaul[10] (1851 and 1878) did the like, and took the possibility of hereditary syphilis into consideration. Virchow[11] (1856) described a foetus with short limbs and large head as a case of congenital rickets. H. Müller[12] (1860) distinguished a case of this kind from the rickets of infancy. He believed it to be an example of rickets arising *in utero*, and described the characteristics of the condition as "default of ordination of cartilage cells, with premature synostosis of the bones of the base of the skull." Winkler[13] (1871) proposed for this condition the name "Rachitis micromelica"; Urtel[14] (1873) advanced the hypothesis that the condition was one of intra-uterine inflammation of cartilage (chondritis foetalis). But other authors such as Scharlau[15] (1867) and Fischer[16] (1875) regarded it as true rickets, either simple or complicated by cretinism. In 1876, however, Parrot[17] drew attention to the differences between this condition and true rickets on the one hand, and hereditary syphilis on the other, outlining its clinical and pathological features as they are now known. These he stated to be (1) micromelia, or shortening of the long bones of the limbs, plus (2) abnormal shape of the cranium, (3) absence of thoracic deformity, (4) marked thickening of the skin. But his researches passed unnoticed, and the condition was still looked upon as a rickety or syphilitic

[1] See Bibl. No. 386.
[2] See Bibl. No. 547.
[3] See Bibl. No. 550, p. 43.
[4] See Bibl. No. 491.
[5] See Bibl. No. 63.
[6] See Bibl. No. 61.
[7] See Bibl. No. 73.
[8] See Bibl. No. 78.
[9] See Bibl. No. 111.
[10] See Bibl. Nos. 100 and 165.
[11] See Bibl. No. 107.
[12] See Bibl. No. 120.
[13] See Bibl. No. 143.
[14] See Bibl. No. 144.
[15] See Bibl. No. 135.
[16] See Bibl. No. 153.
[17] See Bibl. No. 161.

manifestation, Eberth[1] (1878), M. A. Smith[2] (1880), Neumann[3] (1881), Spiegelberg[4] (1882), Kassowitz[5] (1879) and several other observers continuing to hold the old opinion that it was an expression of true rickets. In 1889, Porak[6] recorded some new facts, and entirely confirmed Parrot's views. He also described a hereditary case, demonstrated the occurrence of the condition in lower animals, and showed that the disease had really been familiar from very remote times. (See our illustrations, Plates S and LL.) In the same year Kirchberg and Marchand[7] and Stilling[8], in independent researches, came to conclusions much the same as Porak's. They concluded that it was of a different nature from rickets, but each gave the condition a different name. These papers, however, are only of historical interest. After this, confirmatory observations rapidly accumulated. Amongst these were those of Kaufmann[9] (1893), who described 14 cases, S. Müller[10] (1893), Lugeol[11] (1892), Salvetti[12] (1894), Thompson[13] (1893), Porak and Durante[14] (1894) and others. Although several cases in adults had been described, until the year 1900 for the most part only the achondroplasic foetus had been studied. But in 1900, P. Marie[15] clearly described the condition in the adult for the first time. In 1901 and 1902, Apert[16], and in 1902 Méry and Labbé[17], also dealt with the condition in adults. They showed the distinguishing features between achondroplasia in adults and cretinism and myxoedema, which had hitherto been to a great extent confused. Apert and others insisted on the existence of hereditary myxoedema, and the importance of modification of species. Regnault[18] (1901) examined 14 achondroplasic skeletons in the Dupuytren Museum with the object of determining the macroscopic morbid anatomy of the condition. Its radiographic characters were established by Johannessen[19] (1898), Joachimsthal[20] (1899), Cestan and Infroit[21] (1901), and Molin[22] (1901). In 1902, Durante[23] showed that two conditions had hitherto been included under the term achondroplasia, namely (1) true achondroplasia, characterised by sclerosis of the epiphysial cartilages in early life, and (2) "the periosteal dystrophy," in which there is abnormality of the osteoblasts, while the epiphysial cartilage is normal. More recently, the researches of Porak and Durante[24] and many others have been published, but they are too numerous to be all given here. Achondroplasia is now recognised as a clinical entity. But its pathogenesis and the mode in which the bony lesion is produced are still uncertain. The variety of the conditions that occur in it explains this. These have been described as: Rachitis annularis or micromelica, Chondritis foetalis, Osteogenesis imperfecta, Pseudo-chondritis, Cretinoid Dysplasia, Micromelia chondromalacica or pseudo-rhachitica, Osteoporosis and osteosclerosis congenita, Periosteal Aplasia with osteopsathysosis, Chondrodystrophia foetalis, divided into three varieties, hyperplastica, hypoplastica and malacica by Kaufmann[25] (because of the large volume and dense consistency of the epiphyses), Micromelia, etc.

[1] See Bibl. No. 173.
[2] See Bibl. No. 180.
[3] See Bibl. No. 191.
[4] See Bibl. No. 194.
[5] See Bibl. No. 217.
[6] See Bibl. No. 247.
[7] See Bibl. No. 243.
[8] See Bibl. No. 245.
[9] See Bibl. No. 279.
[10] See Bibl. No. 282.
[11] See Bibl. No. 276.
[12] See Bibl. No. 298.
[13] See Bibl. No. 281.
[14] See Bibl. No. 299.
[15] See Bibl. No. 371.
[16] See Bibl. Nos. 380 and 413.
[17] See Bibl. No. 410.
[18] See Bibl. No. 389.
[19] See Bibl. No. 351.
[20] See Bibl. No. 363.
[21] See Bibl. No. 388.
[22] See Bibl. No. 387.
[23] See Bibl. No. 412.
[24] See Bibl. Nos. 490, 491.
[25] See Bibl. No. 279.

In Germany the name "foetal rickets" is generally used for it, but leads to confusion. In France "achondroplasia" has been generally adopted, and this term is the one commonly used in this country. But the term "achondroplasia" has hitherto been used to describe what appear to be two different conditions as shown by Durante (*loc. supra cit.*). In one there is abnormal division of cartilage cells, with normal periosteal bone formation. In the other there is a defect of periosteal cell division with normal chondral cell division. Kaufmann, Porak and Durante (*loc. supra cit.*) and others have shown that the latter condition is a lesion of compact bone, and that there are two varieties of this congenital bony dystrophy, both of which were hitherto known as "achondroplasia": (1) true achondroplasia and (2) the periosteal dystrophy (dysplasie periostale).

(1) *True Achondroplasia* is a developmental disease of the skeleton appearing in the course of intra-uterine life, affecting chiefly the long bones of the limbs, the pelvis and the base of the skull. Histologically it is characterised by sclerosis of the cartilages of conjugation of the epiphyses. Clinically, its manifestations are: marked shortening of the limbs, which are very thick and heavy, deformity of the pelvis, and depression and broadening of the bridge of the nose, dependent upon premature synostosis of the cartilage-bones of the base of the skull, a fully developed cranial vault, a trunk of almost normal growth, intelligence equal to the average, and genital organs normally developed.

(2) *Periosteal Dysplasia* is a defect of development of the skeleton affecting especially the diaphyses of the long bones of the limbs, the ribs and the bones of the cranial vault. Histologically there is defect in the formation of periosteal compact bone. This is also shown by fragility, multiple fractures being of frequent occurrence. "There is no premature synostosis of the bones of the base of the skull, but the presence of shortened, curved limbs is usual, if not constant" (Porak and Durante[1]). This variety of congenital bony dystrophy seems clearly to be rarer than achondroplasia. Its essential pathological features are normal chondral ossification and defective periosteal ossification. Micromelia is the only feature that it has in common with achondroplasia. As this account is concerned with achondroplasia, the condition does not require further reference here.

B. Clinical Symptoms.

(1) *In the Newborn.* The diagnosis of the achondroplasic foetus can be made, in a typical case, at a glance. The infant is one with a trunk of normal size, short, very thick limbs, markedly curved with convexity outwards, head apparently rather bigger than usual, with bulging frontal and parietal eminences, and nose the bridge of which is depressed. The shortening of limbs, which are very thick, so that the girth of the segments is often equal to or greater than their length, usually affects all four equally. The finger-tips, which in normal individuals reach to the middle

[1] See Bibl. No. 490.

third of the thigh, reach, in these cases, no further than the great trochanter of the femur, or, it may be, the iliac crest. The shortening of bones which produces this peculiarity is more marked in the proximal than in the distal segment. It is more marked in the thigh and arm than in the leg and forearm, and it affects the foot and hand still less than the last two. In other words, while in normal newborn infants the thigh and arm are longer than the leg and forearm, in achondroplasic infants the reverse is true. But this condition is not quite constant in achondroplasia. The growth of bone in length and the general development from embryonic life to adult age do not occur in the normal in regular progression, but first one segment or part and then another is in advance, as was first shown by Humphry[1]. Again the two ends of the diaphysis do not take equal shares in producing the increase in length of a long bone, the one growing much more extensively than the other. Therefore the above peculiarity may be more marked at one age than another and is said to be more usual in the adult than in the infant (Porak and Durante[2]). The *skin* is usually thickened, the subcutaneous tissues increased, from which transverse folds in the skin in the neighbourhood of the articulations result. As a consequence of this there is produced in the continuity of segments of the limbs, *e.g.* thigh, leg, and foot, or arm, forearm and hand, an appearance of several short cylinders, superimposed upon one another. The limbs have a peculiar pudding-like appearance (*aspect boudiné*) as a result. (See illustrations, Plate S (14).) Besides shortening there are other more or less obvious *deformities* of the limbs. The thigh is shortened with a concavity on its postero-internal surface (the foot is as a rule extended and rotated inwards). These deformities are not usually of high degree, but they form angles rather than general curves, and affect the points of union of epiphyses and diaphyses rather than the shafts of the long bones. The muscles seem to be more developed than in normal infants.

The hands and feet are broad and thick, like the other segments of the limbs, but present peculiarities of their own. The middle digit of the hands is short. Its length does not exceed that of its fellows on either side. The digits are roughly conical in form and diverge from one another at their extremities. They form the "hand like a trident" ("main en trident") as it was described by Pierre Marie[3]. Others have described the fingers as radiating like the spokes of a wheel. The face is relatively small, the root of the nose flattened and the frontal eminences prominent. The cranium is large and brachycephalic. The fontanelles and sutures are regular, or a little exaggerated, and the consistency of the bone of the vault is normal. While the cranium is large, the face is relatively small, and thus the head as a whole has a roughly pyriform shape as described by Porak and Durante[4]. The trunk is well formed and of normal size for the term at which delivery occurs;

[1] Humphry (Sir G. M.): *On the Skeleton*, Cambridge, 1858, pp. 97—98.
[2] See Bibl. No. 491.
[3] See Bibl. No. 371.
[4] This appearance is also the rule in hydrocephalus. In that condition, however, it is generally much more obvious than in achondroplasia. It is probably owing to this appearance that some cases of achondroplasia have been described as hydrocephalic as was the case in the foetal skeleton in Royal College of Surgeons reproduced on Plate W. Cestan (*loc. supra cit.*) has shown that the peculiarity is due to the conformation of bones. Nevertheless cases of achondroplasia in the foetus, the child, and the adult, are still frequently described as hydrocephalic.

the lower border of the thorax (costal cartilages) may be prominent, the lower aperture of the thorax being slightly enlarged. The vertebral column is straight, the back flat, there is little real lordosis, but even in the newborn infant, probably owing to forward tilting of the pelvis, the buttocks are prominent and there is a more or less marked (apparent) lumbar curvature and a prominent abdomen. This forward tilting has been attributed by some writers to the smallness of the hip-bones, as a consequence of which the acetabula are displaced backwards. Thus the abdomen and buttocks become prominent and an appearance of lordosis results. (See illustrations, Plates P, S and Y.)

(2) *In the Child.* Most infants of this kind are born dead or die soon after birth, or in the first year of life. But if they survive, and it is not quite clear why they should not, they usually do well. Their processes of ossification, as far as they go, seem to be rather in advance of the normal than otherwise. This agrees with the premature synostosis of the bones of the base of the skull that has been described, but the period at which this occurs is uncertain. At birth the child is fat, but as it grows up it usually becomes very muscular. Such children generally begin to walk and talk at the ordinary age. The teeth appear normally, and the intelligence is also normal.

(3) *In the Adult.* The clinical study of achondroplasia in the adolescent and the adult was begun by Porak[1] and Kaufmann[2], in women, from the obstetrical point of view. It was continued by Pierre Marie[3] and Apert[4]. The features of the condition have been shown by them to be: (1) Smallness of height. Thus, in the case of Don Ward, aged 28 years, whose photograph is shown on Plate Q (6)—(8), this is 4 feet 0 inch. The Chinaman, aged 58 years, shown on Plate R (11)—(13), measures 3 feet $6\frac{3}{4}$ inches. Elisabeth Dörffler, *née* Kipke (Boeckh's case), aged 42 years, whose photograph is shown on Plate Q (9), is 3 feet 2·18 inches; her daughter, aged 17 years, the same, and her sister, aged 43 years, about the same; while in Bailly's case (quoted by Marie[5]), a woman, 27 years of age, this was 107 cm. (3 feet 6·12 inches)[6]. (2) A normal trunk. (3) A large head. (4) Excessive muscular development. This is even more marked in the adult than in the infant. The arms are muscular and held a little abducted from the trunk as a result of the disproportionate size of the head of the humerus. But (5) they are very short and when the fingers are fully extended their tips do not reach beyond the great trochanters of the femora, or, it may be, the crests of the ilia, whereas in the normal individual they reach as far as the middle third of the thigh. (6) The lower limbs are short and massive. They show angular deformities, more or less marked, just above and below the knees. These occur at the site of union of epiphysis and diaphysis and do not involve the shafts, as they do in rickets. This *appearance* of curvature is increased by the great development of muscles on the anterior and external aspects of the thigh. (7) As a result of the shortening of the lower limbs

[1] See Bibl. Nos. 247 and 252. [2] See Bibl. Nos. 275 and 279. [3] See Bibl. No. 371.
[4] See Bibl. No. 386. [5] See Bibl. No. 371.
[6] Other cases are shown in the illustrations.

the midpoint between the vertex and the soles of the feet may be as much as a quarter of the total height above the symphysis pubis (Porak and Durante[1]). (In the normal individual the upper border of the symphysis pubis is about midway between the vertex and the soles of the feet.) In these individuals the length of the trunk, measured from the episternal notch to symphysis, is of about the normal length for age, and the proportions to each other of the measurements between the vertex and symphysis and episternal notch and symphysis are normal. (8) The above shortening of limbs is of the "rhizomelic" as opposed to the "mesomelic" type, the proximal segment being more shortened than the middle segment. (9) The hands and feet are short, thick and broad, the fingers of equal length; the same condition is shown in the feet. (10) The digits of the hands diverge at their extremities in extension, showing the "main en trident" (see Plate P (5) and Plate R (11)) as in the infant. The metacarpal bones and phalanges are shortened, but this shortening is relatively less than that of the bones of the other two segments of the limb (thigh and leg; arm and forearm). The head, proportionately, is larger than normal, and in some cases the increase is absolute (one case described by Apert[2] had to have his hats made to measure owing to the large size of the head). In shape it is round, or brachycephalic. The frontal and parietal eminences are prominent. The face is relatively small but with large features. The bridge of the nose is broad and flat. The nose is retroussé, the nostrils are large. The shape of the head as a whole presents some rough resemblance to an inverted pear, as described by Porak. The teeth are normal. The palatine vault, though sometimes of the high-arched or Gothic type, is usually regularly formed and of the normal shape. The thorax is normally formed, but the lower border is often prominent. The scapulae are of defective development, but are proportionately less deficient than the humeri, because, in the former, chondral ossification commences later in life than in the latter. The spine shows no abnormal curvature. But the normal lumbar curve is increased, or is made to appear so, by the excessive development of the buttocks. The general muscular development is great (see illustrations). "They often perform in circuses as 'Auguste,' and amuse the audience by dangerous feats and exercises that demand strength for their execution. These are rendered grotesque by the diminutive stature of the performers" (Porak and Durante[3]). Obesity is common in the female sex (Porak and Durante[4]), but rare in the male (Marie[5], Apert[6]). "The weight of achondroplasiacs accords with their general stoutness and is much above that of children of the same height, for their trunks are of normal size and their bones thick" (P. Marie[7]). The genital organs are normal. The female may become pregnant, and this fact makes the pelvic deformity of great importance. Achondroplasiacs differ greatly from cretins and most other dwarfs in their mental qualities. They are of average intelligence, but often show minor peculiarities in their mental attributes.

[1] See Bibl. No. 491.
[2] See Bibl. No. 386.
[3] See Bibl. No. 491.
[4] See Bibl. No. 491.
[5] See Bibl. No. 371.
[6] See Bibl. No. 386.
[7] See Bibl. No. 371.

C. Characters of the Skeleton.

(1) *In the Foetus* the long bones of the limbs are short, thick and show angular deformity. The shortening is rhizomelic as a rule, but this is not invariable. The epiphyses are much enlarged in all their dimensions and afford a marked contrast to the shortness of the diaphyses. The latter are firm and hard and relatively thickened. The markings of muscular attachments are exaggerated. Between the epiphysis and the diaphysis a strip of connective tissue can on section be seen to extend, partially separating the one from the other. This has been regarded as an ingrowth of perichondrium, as an infolding of perichondrium, and as the result of inflammation. It has also, by some, been supposed to be a membrane. The tibia is more shortened than the fibula, which usually reaches to the knee joint, and its head forms part of the lower articular surface of this joint. The tibia and fibula are both deformed. An "angular" curvature is produced at the site of junction of epiphyses with diaphyses in both these bones at both extremities. In the case of the tibia the angle opens outwards, in the case of the fibula inwards. The interosseous space is thus increased. These angular curvatures are symmetrically placed, usually at the level of junction of epiphysis and diaphysis. This condition is not always seen in the forearm, but is occasionally present, as in Dixon's case[1]. Growth has not ceased, but is retarded. The sclerosis is not equally marked at all points. In some parts cell proliferation occurs a little less badly than in others (Porak and Durante[2]). From this a deviation of the axes of the epiphysis and the diaphysis (instead of continuation of these in one straight line) results and an angle is formed. This is originally juxta-epiphysial, but if the deformity occurs long before growth is complete, growth continues in the new direction, and the angle finally occupies a variable position in the shaft. "In the adult its distance from the line of ossification affords an approximate indication of the time at which it arose" (Porak and Durante[2]). In most cases in the infant other curves can be observed in the diaphysis. (See bones of achondroplasia, Plates S (15), T (16), U (18).) The cranium is large, brachycephalic, with prominent frontal and parietal eminences; the face is small, receding and narrow. The bones of the nose, broadened at the base, are retracted, large, triangular or quadrilateral, and completely united. The upper jaws are approximated to the vertebral column more nearly than normal.

The bones of the base of the skull show arrest of development and premature union of the basi-sphenoid and basi-occipital bones. As a result of this the sphenoidal angle is more acute than usual (Kaufmann[3]). The occipital condyles are prematurely united to the basi-occipital bone. The basi-occipital is markedly inclined; it draws down the petrous, the posterior surface of which becomes more nearly vertical. The occipital fossa appears deepened with vertical walls, 40 mm. above the level of the occipital foramen (Regnault[4]). Occasionally there is premature union of the bones of the anterior region of the base of the skull as well. Though these premature

[1] See Bibl. No. 606. [2] See Bibl. No. 491. [3] See Bibl. No. 275.
[4] See Bibl. No. 389, pp. 190 and 425.

unions are the rule, they may exceptionally be absent (Porak and Durante[1]). The thorax. This is usually normal, but may be enlarged at the base. In some cases the ribs are enlarged and thickened, their grooves are exaggerated and there are small discrete nodular thickenings on their inner surfaces. But there is usually no thickening at the costo-chondral junctions as occurs in rickets. The clavicle is sometimes normal, but may be thickened and show exaggeration of all its curves and muscular markings. Vertebral column: the laminae and spinous processes are usually enlarged but there is no curvature. The spinal canal and occipital foramen are sometimes narrowed (Porak and Durante[2]).

(2) *In Adults* the same skeletal changes are present, but all are more marked than in the infant. The radio-humeral index, 100 Length of radius/Length of humerus, in normal Europeans varies from 82 to 88[3]. In achondroplasia it is raised to 100 or even 140. The tibio-femoral index, 100 Length of tibia/Length of femur, is normally between 84 and 90. It may here be 105 to 120 (Porak and Durante[4]). In some cases, however, the index is normal; in others the radio-humeral may be as low as 66, 58, 53, the tibio-femoral 78 or 64 (Regnault[5]). Other features are the same in the adult as in the foetus. The apparent exaggeration of the lumbar curvature, which causes the appearance of lordosis, is produced by the following factors: (1) tilting of the sacrum, forwards of the upper end and backwards of its lower end. This is owing to the fact that the innominate bones are of deficient growth, while the sacrum is less so. Hence the sacro-iliac articulation occurs in a different plane from the normal, the sacral promontory becoming tilted forwards and downwards. (2) Prominence of the buttocks. This Poynton[6] attributes to the fact that, since the innominate bones are defective in size, the ilio-pectineal lines are relatively shorter than normal. The acetabula, through which, when the individual is standing, the weight of the body is transmitted, are thus displaced relatively backwards, *i.e.* nearer the sacro-iliac articulation than normal; hence the buttocks become very prominent. (The prominence of the abdomen seems probably owing to the fact that the ilia, as well as the other parts of the innominate bones, are deficient in size. Hence the false pelvis as well as the true pelvis is small, and the abdomen projects unduly in consequence.) The pelvis is characterised by tilting of the sacrum forwards, marked absolute diminution of all diameters and relative diminution in the antero-posterior diameter of the inlet. According to Poynton[7], the true conjugate seldom exceeds $2\frac{3}{4}$ inches, and may be as small as $1\frac{5}{8}$ inches. The pelvic defect does not affect all the pelvic bones equally. The innominate bones are small, the sacrum not. As a result of the relatively great development of the trunk and head, and because the weight is transmitted through the acetabula, the pelvis becomes tilted, and the sacral promontory is displaced[8]. The upper end of the sacrum is displaced forwards,

[1] See Bibl. No. 491, p. 18. [2] See Bibl. No. 491, p. 19.

[3] [Porak and Durante must be in error here; the radio-humeral index has for mean value in modern French 72 and for mediaeval French 71. The tibio-femoral index for modern French has a mean value 80 and for mediaeval French 83; all values less than the total range given above. Editor.] [4] See Bibl. No. 491, p. 19.

[5] See Bibl. No. 389. [6] See Bibl. No. 534. [7] See Bibl. No. 534.

[8] [This explanation seems hardly consistent with the fact that the conjugate diameter of the pelvis, especially the ilio-pectineal portion, is greater in the female than the male, and the inclination of the pelvis is generally greater in consequence: *Quain's Anatomy*, 10th Edn. Vol. II. p. 117. Editor.]

and its lower end backwards. The antero-posterior diameter of the inlet thus becomes markedly diminished, relatively more than the other diameters. *Radiographic Appearances.* These features are shown in the illustrations: see Plates U, V and X. In the foetus the long bones are defective[1]. They are short, thickened and squat, and sometimes distorted. The epiphyses are very large, and are separated from the diaphyses by a very large clear space of transparent material. The phalanges, metacarpal and metatarsal bones are also shortened and of almost square shape instead of the usual form of elongated rectangles. The clavicles and ribs are short and thickened, but the vertebrae are diminished in thickness, and appear more distant from one another than normal (Bouchacourt[2]). These changes are, however, not constant. The bones are more transparent than in normal individuals (Levi and Bouchacourt[3]). In older children the epiphyses are clearly marked and of great size. Their opacity is greater than in the normal child (Cestan and Infroit[4], Méry[5]). Ossification and calcification are more advanced than is usual for their age. The diaphysis is short, sometimes of normal thickness, sometimes thickened, either straight or curved, but if present the curvature is situated less in the diaphysial body than at the junction of epiphysis and diaphysis. Cartilaginous development is retarded. The diaphysis grows in length and thickness slowly, remaining separated from the epiphysis by a transparent line until adult age. Union is said to be usually premature. In one of P. Marie's[6] cases, aged 18 years, the head and shaft of the humerus were ununited as is usual for that age. But in another, aged 40 years, union was still incomplete. In a third, aged 25 years, union had occurred. (The average age at which, in normal individuals, junction becomes complete at this end of the humerus is the 20th year.) Thus the rule as to premature union is certainly not absolute.

D. Pathological Anatomy.

Only the skeleton is affected. All bones developed from cartilage show the same histological changes, but these are best seen in the long bones. The costo-chondral line of ossification, however, usually involved in rickets, is free in the case of achondroplasia. The microscopic lesions are more pathognomonic than the clinical signs and may be the only means of diagnosis in atypical and doubtful cases (Porak and Durante[7]).

Before describing the microscopic changes of achondroplasia, a brief outline of the development of normal bone will be given for purposes of comparison.

In the normal development of bones, ossification occurs by two processes: (*a*) *Chondral Ossification.* This determines growth in length at the junction of epiphysis and diaphysis. (*b*) *Periosteal Ossification.* This determines growth in thickness, of the shaft or diaphysis in long bones, and of the bone as a whole in flat bones. In all the latter, except those of the cranial vault, however, both processes take part. The changes that occur may be broadly outlined as follows:—(*a*) *Chondral Ossification.* In the zone of ossification three successive layers may be distinguished

[1] See Bibl. No. 491, p. 15. [2] See Bibl. No. 479. [3] See Bibl. No. 480. [4] See Bibl. No. 388.
[5] See Bibl. No. 410. [6] See Bibl. No. 371. [7] See Bibl. No. 491, p. 15.

in passing from the area of undifferentiated cartilage of the epiphysis to the diaphysis. (1) The zone of proliferation of cartilage cells. Here the cells of the cartilage are increasing in numbers, and extend in a vertical direction, but irregularly. They are close together, but are separated by a noticeable quantity of clear, intercellular, hyaline cartilage—a matrix of clear hyaline cartilage. (2) The zone of columns of cartilage cells. The cells, increasing in number and in size, here become more transparent and are formed into regular columns. The cells nearer to the next zone, the line of ossification, are larger than those more remote. The columns are all exactly parallel to one another; they are very near together, but are separated by a small amount of clear hyaline substance (hyaline matrix). Towards the next zone, however, a deposit of calcareous material is to be observed in this matrix, *i.e.* it begins to calcify. These two zones, numbers (1) and (2), together form a thin blue, translucent zone of 1 to 2 mm. in thickness, the chondral layer. (3) The line of ossification or bony layer. This shows itself as a thin zone of a dull yellow colour about $\frac{1}{2}$ cm. thick. Each of the columns of cartilage cells extends here as far as the medulla or bone marrow. From the latter vascular loops grow, eroding the columns of cartilage cells quite regularly, and each to the same level. The septa, which separate these columns, thus eroded and denuded of their cells, become somewhat irregular and incrusted with calcareous salts; they form the scaffoldings of the future bony trabeculae. On their surfaces osteoblasts are deposited by the vascular loops, and for these they serve as supports or buttresses. The osteoblasts lay down true bone by successive deposits of bony lamellae, while the calcareous material of zone 2 is absorbed by osteoclasts. A microscopic section at the region of growing bone under a low power, therefore, shows the following zones: (1) zone of proliferation of cartilage cells; (2) zone of columns of cartilage cells; (3) the "line of ossification"; (4) marrow and bony trabeculae, with the minute changes above described.

(*b*) *Periosteal Ossification.* The osteogenic layer of the periosteum is composed of a fibrous tissue matrix and of special cells—the osteoblasts. As the diaphysis is approached, the fibrous tissue matrix becomes impregnated with calcareous salts more and more thickly. It separates the columns of cartilage cells. It also serves to support the osteoblasts, which secrete ostein or true bone substance. This surrounds the cartilage cells, and transforms them into bone cells. Trabeculae are formed, and elongated spaces are produced, which afford a path for vessels from the periosteum. Ossification is completed by successive deposits by the vessels of ranges of osteoblasts to these primitive trabeculae. The osteoblasts lay down true bone. Concentric osseous lamellae are in this way produced, and the Haversian systems of compact bone are formed. In *achondroplasia* periosteal ossification is normal, or even, if anything, of increased activity in some cases. In the periosteal dystrophy periosteal bone formation is almost completely lacking; only slight, irregular and discontinuous lamellae are present. There is very little compact bone. That laid down is in part destroyed by resorption by osteoclasts. Chondral ossification is normal. (Section Plate U (25), reproduced by the courtesy of the Editor of *Le Nouvelle Iconographie de la Salpêtrière*, Paris.) In achondroplasia longitudinal

section of an area of chondral ossification such as the upper end of the humerus shows the following changes from the normal: (1) The area of inactive hyaline cartilage is unaltered, but between this and the zone of columns of cartilage cells (Zone 2 of the normal as above described), separating the two, there is, occupying what should be the zone of proliferation of cartilage cells (Zone 1 of the above), a fibrous band or area. This is more marked in some places than in others, and may occur on one side only; it is vascular and enclosed within it are small areas of cartilage. It is directly continuous with the perichondrium or lining membrane of the epiphysis, and thins out towards the centre of the bone end. It has by some been looked upon as a membrane. (2) The arrangement of cartilage cells in columns (in Zone 2 of the above description of the normal) is here completely lacking; the cells are scattered and dispersed without order, and are few in number. The matrix or intercellular ground substance of this zone is not hyaline, but shows fibrillation, and where it approaches the perichondrium, it takes the form of white fibrous tissue. In places this matrix shows mucoid degeneration and vacuolation. Where this fibrillar, or, perhaps, fibro-cartilaginous, matrix approaches the next zone (Zone 3, the "line of ossification" of the above description of the normal), there is a deposit of calcareous salts in places, but this is not uniform as in the normal. (3) The line of ossification is thin and may be somewhat irregular. (4) In some cases the formation of bony trabeculae is normal, and the medullary spaces are normally constituted; in other cases, Zone 3 is replaced by mucoid tissue; sometimes, again, all the changes in Zone 4 are in excess, but are otherwise normal. (See Section, Plate U (23).) The epiphysial cartilages, as a whole, are very vascular. Such are, broadly, the microscopical changes in outline. Kaufmann[1] divided cases into three groups: "hypoplastic," in which growth is merely retarded along the lines described above; "hyperplastic," in which the above changes are in excess; and "malacic," in which, still further, the cartilage softens. Others have followed this classification. Regnault[2], however, regards all three of these as grades of one and the same condition, of which only the first is compatible with life. There are no special changes in parts other than bone. Such changes as occur may be found alone or in association with any other pathological condition or abnormality. Emerson[3] gives the following associated abnormalities: hypospadias, cervical cysts, spina bifida, defective auricles, defects of development of ensiform cartilage, umbilical hernia, inguinal hernia, cleft palate, genu valgum, new growths of various kinds. Porak and Durante[4] record congenital dislocation of the hip, adenoids, enlarged uterus, enlarged thyroid, multiple cystic disease of the kidneys. Nathan[5] records congenital hernia and high-arched palate. Myxoedema, in association with achondroplasia, has been recorded more than once, and is shown in a case on the pedigree plates. Mental deficiency is also shown there by Dr Hunter's Case. Porak and Durante[6] found in two cases in the infant "congestion, recent haemorrhages and small celled infiltration in the spinal cord, liver, muscles and kidneys," and consider that this favours the view of infection rather than auto-intoxication as a cause.

[1] See Bibl. Nos. 275 and 279.
[2] See Bibl. No. 389.
[3] See Bibl. No. 604.
[4] See Bibl. No. 491, p. 26.
[5] See Bibl. No. 475.
[6] See Bibl. No. 491, p. 26.

E. Forms and Complications of Achondroplasia.

The following varieties have been described: (1) The classical type. (2) Incomplete forms, in which a certain number of the typical features are modified or absent, *e.g.* micromelia may be only slightly marked (as in Houston Porter's cases in the pedigrees, or the head not very large or not of typical shape, see adult skeleton Plate U (18) and Dr Hunter's Case in the pedigree plates), or the nose atypical, or the hands and feet normal. (3) The ribs and vertebrae are affected, the former showing excessive grooving and thickening at their points of union with one another (a feature shown by the above adult skeleton), the latter a thickening of lamellae and spinous processes (Bouchacourt[1], Legry[2], Regnault[3]). (4) The premature synostosis of the bones of the base of the skull is lacking (von Franqué[4], Lampe[5], and Salvetti[6]). (5) Micromelia of the lower extremities alone (Variot[7]), humerus alone shortened (Regnault[8]). (6) Osteoporosis of the diaphysis with lesions otherwise those of true achondroplasia (Porak and Durante[9]). (7) Complication by other conditions such as rickets (Plates S and Y) and cretinism or myxoedema (Porak and Durante[10] and others). (8) True achondroplasia, as well as micromelia alone, is sometimes seen in lower animals (Leblanc[11]). See section below on Heredity.

Diagnosis. In typical cases the diagnosis of achondroplasia either in the foetus, child, or adult, presents no difficulties; its characters, which seem to have been sufficiently described, are distinctive at a glance. But not one of these features *alone* is diagnostic, for each may occur in other conditions. The features of most of the other commoner varieties of dwarf growth are outlined either in the illustrations or in the general classification of dwarf growth. From these the diagnosis can be made on general principles, and presents no difficulty in typical cases. In atypical cases of achondroplasia confusion appears most liable to occur with certain cases of rickets and with periosteal dysplasia. Such cases are rare. According to Porak and Durante[12] in some instances the differential diagnosis is only possible on histological examination. The three conditions are fairly clearly shown in the illustrations. With regard to the clinical features in doubtful cases Porak and Durante[12] point to the following as in favour of the diagnosis of achondroplasia:—(1) Extreme muscularity; (2) character of curvatures (as described); (3) absence of fractures; (4) regular dentition; (5) shape of skull (as described); (6) micromelia (*i.e.* real shortening) as opposed to pseudo-micromelia (the result of curvatures); (7) radiographic examination showing epiphysis much enlarged, with regular or premature appearance of centres of ossification, diaphyses short and thickened and of normal consistence. Achondroplasia and rickets may co-exist, as occurs in the twin child shown on Plate P. The characters of the pelvis differ in these two conditions; in achondroplasia all diameters of the pelvis are diminished more or less equally, because, though all the bones entering

[1] See Bibl. No. 479.
[2] See Bibl. No. 422.
[3] See Bibl. No. 389.
[4] See Bibl. No. 278.
[5] See Bibl. No. 303.
[6] See Bibl. No. 298.
[7] See Bibl. No. 440.
[8] See Bibl. No. 411.
[9] See Bibl. No. 491.
[10] See Bibl. No. 491.
[11] See Bibl. No. 421.
[12] See Bibl. No. 491.

into it are small, they are never at any period softened. Owing, however, to tilting of the sacrum, the inlet of the pelvis is diminished, though the antero-posterior diameter itself is not reduced more than the others. In the rickety pelvis, arrest of growth to any great extent does not occur, but owing to the softening that has occurred, bending of bone under superincumbent weight takes place at points of pressure. The sacrum is pushed forwards, its curve increased. The antero-posterior diameter is thus diminished, but the transverse is sometimes increased. Another cause of multiple fractures in the newborn, besides the above conditions, seems to be fragilitas ossium congenitalis, a condition in which the tendency to fractures persists through life; the condition has been reported to be hereditary, and to be, in some cases, associated with haemophilia. It is a rare condition, for only one case attended the Orthopaedic Department of the London Hospital in two years.

Pathogenesis. Many of the older views as to the pathogenesis have been referred to on page 371. Of more modern ones there are the following: (1) Trauma. There is no evidence for this, and there seems no possibility of this factor playing any part here. (2) Parrot[1], who first employed the name "achondroplasia," regarded the condition as a local one, a "congenital dystrophy of primordial cartilage," *i.e.* a congenital abnormality of the cartilage of growth. Kaufmann[2] also believed the condition to be due to a dystrophy of cartilage, characterised by irregularity and retardation of growth of epiphysial cartilage with proliferation of the bone marrow to invade this and the hard bone of the shaft. (3) The process is one of "arrest" of chondral ossification during intra-uterine life. (4) It is a "sclerosis" of the zone of ossification at the epiphysial line. (5) The latter is an effect of some cause unknown. (6) Most authorities at the present day appear to favour the view that the primary cause of achondroplasia is some maternal intoxication, the nature and origin of which is unknown. This view is supported by: (*a*) the histological appearances of the lesions in the region of cartilaginous bone growth; (*b*) the condition has been found in mother and child and in twins; but analogous facts are recorded for nearly all congenital defects of whatever kind; (*c*) in some few cases the mother has been syphilitic, in others tuberculous; this, however, appears to be of no significance whatever, since in the majority of cases this has not been the case. This seems to be all the evidence there is in favour of this view. As to the nature and mode of action of this intoxication, it has been supposed (i) to be a placental defect; (ii) that the intoxicating agent is localised in the cartilaginous area of the growing bone. (7) It is a "trophic" disturbance, of nervous origin. For this view there seems to be no evidence worthy of attention at all. (8) General maternal debility, not exceeding physiological limits, is the cause. The evidence for this view is defective. (9) The condition is caused by an auto-intoxication of glandular origin. This view was put forward by Marie[3], Leblanc[4], Joachimsthal[5], etc., but appears to confuse the condition with cretinism. Though removal of the thyroid gland in infancy produces dwarfing of growth, cretinism differs completely from achondroplasia. Other glands under

[1] See Bibl. Nos. 161, 172. [2] See Bibl. Nos. 275, 279. [3] See Bibl. No. 371.
[4] See Bibl. No. 421. [5] See Bibl. No. 363.

suspicion are: thymus, testis and pituitary, which seem to be suspected not only in all forms of growth in which the primary cause is unknown, but in many other conditions the origin of which is obscure. In this case there is no reliable evidence whatever in favour of any of them. (10) The condition is due to an infection. (11) It is due to some auto-intoxication other than of glandular origin. At the present time of the suggested causes the most plausible seem to be (10) and (11).

Of (11) nothing is known; the matter seems to be one of pure speculation. With regard to (10) it can only be said that this seems to be the most reasonable at the present day. The sclerosis, which is present in all cases[1], may be produced by a toxin; this may be supposed to be of bacterial or other origin. The most markedly sclerogenic of such toxins at present known seems that of syphilis, others are those of tuberculosis and alcohol. It seems improbable that the syphilitic toxin, or either of the other two, is this particular sclerosing agent. The condition might be supposed to be produced by one organism specific for this dystrophy or by several indifferently, either those suggested or others at present unknown[2]. In the case of the periosteal dystrophy there is no sclerosis, and the view is held (notably by Porak and others) that the histological changes follow the type of some glandular or trophic disturbance, and that hereditary or auto-intoxication must be considered possible. "The condition has analogies with the myopathies" (Porak). It seems that, whatever the exciting cause, this morbid susceptibility of cartilage is hereditarily transmissible in some families much in the same way as is the "pre-disposition" or "vulnerability" to tuberculous or rheumatic infections, etc. The various hypotheses that have been advanced on this subject in recent years are nearly all expressions in one or other form of the idea of lowered resistance locally.

F. Heredity in Achondroplasia.

It would seem that the condition may appear "accidentally" in a family or it may be "hereditary." The achondroplasic individual has normal reproductive powers. Heredity has shown itself in: the father; the mother; in more than one member of the same generation; father and daughter; grandfather, father, brother and sons; in twins, both of which were achondroplasic. (For these cases see pedigrees.) In a new case of twin progeny shown with the plates (Dr R. Hutchison's case, Plate P) one twin was achondroplasic and the other normal. S. Müller[3] and Klein[4] have recorded similar instances. An achondroplasic parent of either sex may have normal children; and this seems to be the usual occurrence. The circumstances in which heredity can show itself directly in this condition are limited, because no achondroplasic woman can come to a normal confinement, and formerly, before the

[1] "La lésion essentielle de l'achoudroplasie est une *sclérose, puis une dégénérescence calcaire du cartilage de conjugaison* avec intégrité complète ou relative de l'ossification périostale." Porak and Durante: see Bibl. No. 491, p. 44.

[2] It seems possible that the sclerosing agent may be none of those that have been suggested. For it is conceivable that it may differ in character as widely from sclerosing agents at present known as does the spirochaeta pallida from, say, alcohol or the bacillus tuberculosis and other canse of chronic inflammation. Until the spirochaete was demonstrated, conceptions as to what the syphilitic organism might be naturally pictured it for the most part on the lines of pathogenic organisms already known, *e.g.* as a bacillus or a coccus. It proved to be none of these. It may be conceived that the agent of achondroplasia, at present unknown, may afford an analogy, and be one of which present knowledge allows no precise conception.

[3] See Bibl. No. 282. [4] See Bibl. No. 397.

operation of Caesarean section had been devised, such women in labour at full term must have died, or if not the child must have been delivered after craniotomy. The female sex seems to be more predisposed to achondroplasia than the male. Kassowitz[1] found amongst 29 cases, 25 girls and 4 boys. Most of these children are either born dead or die soon after birth, and in former times if the mother was achondroplasic she died as well for reasons stated. These facts seem to refute the view which would regard achondroplasic individuals as examples of "atavism," the persistence of an old dwarf race, or the evolution of a new one. This view would consider such individuals as analogous to the "Akkas" and other dwarf races (see Plate O). The differences between the former and the latter are marked, for the "Akkas" and all other existing dwarf races are of normal proportions (dwarf races may very probably show individuals affected with rickets, congenital syphilis, etc., or any of the other varieties of dwarf growth due to disease; but these are only diseased individuals or stocks and seem to have no race characters). In the animal kingdom under natural conditions it would appear that when new qualities occur in individuals it is only the advantageous, those of assistance in the struggle for existence, which become fixed in the race, because they confer advantages. Others tend to die out, with the individuals possessing them, because they are disadvantageous. Micromelia would seem to be of the latter kind. It may be, however, that under civilised conditions, the effect of physical disadvantages being largely eliminated, achondroplasic individuals may be analogous to the races of short-limbed animals of various kinds produced by breeders under artificial conditions, *e.g.* short-limbed hounds and other varieties of short-limbed animals. Here the objection that normal accouchement is impossible in achondroplasia seems very important. Again, achondroplasia seems to have been observed in varieties of domestic animals living under their usual environment; yet no achondroplasic race has been produced. Achondroplasia is a disease, as its histology shows, and though it sometimes shows hereditary transmission seems no more likely to produce a race than tuberculosis, to which in some ways it seems analogous. The superficial resemblances to achondroplasia shown by short-limbed domestic animals are obvious, *e.g.* dachshunds, Aberdeen terriers, Skye terriers, bull dogs, Pekinese spaniels, Basset hounds and other short-limbed dogs of various kinds. But the resemblance is not real. Regnault[2] has studied the skeletons of some of these and has demonstrated in them shortening of the bones of the limbs of rhizomelic type with excessive markings for muscular attachments. The trunk and cranium are normal, the pelvis flattened in its antero-posterior diameter. But there does not appear to be the difficulty in parturition such as occurs in achondroplasia. I. Geoffroy-Saint-Hilaire[3] has described incomplete or partial examples of these short-limbed animals, occurring under domestication, the fore limbs alone of which are shortened. Leblanc[4] and several other observers have described the "basset" condition in such animals as cattle, sheep, pigs, dogs, fowls and pigeons. Achondroplasia occurs in cattle. Thus the "bull-dog calf" has been said to be truly achondroplasic (Emerson[5]).

[1] See Bibl. No. 251. [2] See Bibl. Nos. 289 and 423. [3] See Bibl. No. 75. Vol. I. p. 173.
[4] See Bibl. No. 421. [5] See Bibl. No. 604.

These all show a short upper jaw and micromelia. Regnault[1] states that achondroplasic and "bull-dog" cattle form separate groups; the former show all the pathological changes that occur in human achondroplasia, and instances of it occur at times in all kinds of cattle[2]. In the latter the base of the skull, in particular, is normally developed. Further, short-limbed dogs occur in dogs of all types; coursing dogs, terriers (Aberdeen terriers, Skye terriers, etc.), poodles, spaniels, *e.g.* Pekinese spaniels, hounds (otter hounds and dachshunds), etc. They do not represent a *race* but peculiar varieties of several different races produced by artificial selection. In the aetiology of some varieties of dwarf growth of pathological origin heredity seems to play a part. Of these achondroplasia is one. It seems likely that the heredity may be one of vulnerability, of toxins (bacterial or other) or of a "dystrophy." But it does not seem possible to say for certain, at the present time, which of these views is correct.

In the plates of family histories many pedigrees are included which show no evidence of heredity. This, however, does not preclude its possibility. It is not of course contended that all cases show the hereditary influence. Negative as well as positive facts are here recorded. It does not seem feasible to determine, at the present day, the proportion between "hereditary" and other cases. In this condition as in others it seems desirable that the whole family should be considered, normal as well as deformed individuals being included, if any advance in the knowledge of its heredity is to be made.

Frequency. No reliable information is at present available.

[Note A. *Achondroplasic Forms in primitive Sculpture.* We have already referred to the Egyptian gods Ptah-Sokar and Bes (pp. 357 and 370, Plate S). Owing to the kindness of Professor Petrie we are able to reproduce (Plate QQ (94)—(96)) a series of jugs with more or less achondroplasic figures. Other examples occur in the British Museum collections (*e.g.* Jug No. 30459, which may be compared with the Bes and Ptah-Sokar figures 15291, 22610, 22930, 1419 etc.). The combinations of apparent lordosis and steatopyga (see our p. 375) with such figures are not uncommon. The representation of the Queen of Punt, B.C. 1516—1481 (Plate QQ (96)) is a remarkable instance of this kind, though the exact nature of her dwarfism, if it be such, is obscure (see account of Plates); the bas-relief of her daughter, which has perished, presented similar unusual features. Prehistoric Mexican pottery, and even modern Burmese fetish-like figures, are in many respects comparable with the Egyptian products. A complete study of primitive sculpture and its relation to achondroplasic forms would be of great interest. Editor.]

Note B. *Periosteal Dysplasia* (see p. 373). Various names seem to have been applied to this condition, *e.g. osteo-porosis congenitalis*, achondroplasia being termed under this system *osteo-sclerosis congenitalis*. Such terms as *micromelia chondromalacica* or *pseudo-rachitica*, *osteo-genesis imperfecta*, etc., appear to have been indiscriminately used by some of the relatively older writers for both achondroplasia and periosteal dystrophy, the distinction between the two conditions not having been drawn. It seems probable that under the term *fragilitas ossium congenitalis* (or, as it is sometimes now described for want of exact knowledge by the general term, *osteo-genesis imperfecta*) more than one condition is included. These have as a common feature the occurrence of multiple and repeated fractures from comparatively slight trauma, and seem to present, in this and other respects, resemblances to the periosteal dysplasia on the one hand and to rickets on the other. But the real nature of these conditions is obscure and the clinical resemblances are possibly fallacious.

[1] See Bibl. No. 423.

[2] H. Müller, 1860 (see Bibl. No. 120), first considered at length the "hull dog" calf; he attributed the deformity to a "cretinoid habitus." Leblanc, 1902 (see Bibl. No. 421), found achondroplasia, myxoedema, but more especially defect of the thyroid gland in "bull-dog" cattle. Apert, 1902, asserted (see Bibl. No. 413) that the two first are totally different affections, and that the "bull-dog" cattle are achondroplasic. Legry and Regnault, 1902, maintained (see Bibl. No. 422) that the thyroid gland is normal in achondroplasia and in the same year the latter differentiated "bull-dog" and achondroplasic cattle. Two years later, 1904, Seligmann supported (see Bibl. No. 470[bis]) the thesis that the "bull-dog" cattle are cretins, with an examination of the thyroid. Emerson in 1909 (see Bibl. No. 604) returned to the achondroplasic view. A full explanation must account for Dexter cattle a "dwarfed" race, producing one calf in six of "bull-dog" type. Editor.]

IV. ATELEIOSIS (HASTINGS GILFORD); "TRUE" DWARFISM[1] ("ECHTER ZWERGWUCHS"; "NANISME VRAI").

It has been shown that in the case of achondroplasia its study was begun by the *clinician*, impressed by the peculiarities of newborn infants (usually of female sex and born dead) with a big head, a body of ordinary size, but with short, thick, curved limbs and hands of peculiar form, which he considered to belong to a group apart. Instances of similar cases having accumulated and the pathologist having taken up the study, a torrent of facts and names poured in; and considerable knowledge of the morbid anatomy and pathology of such infants was acquired before the study of achondroplasia (as it is now termed) in the adult had been begun. It was again the *clinician* who began this, in the adult female from the obstetrical standpoint of the contracted pelvis. The identity of these infants, of the peculiar form described, with the large-headed, short-limbed dwarf adults known in everyday life from time immemorial, and so well known to obstetricians, was not immediately realised, probably in part because such infants are nearly always born dead, and partly because they are not, properly speaking, "dwarfs" at all at birth. It was only subsequently that the fact emerged that those few who live grow into those well-known dwarfs, and the study of the subject as a whole was brought into line.

The study of ateleiosis, on the other hand, may be said to have been begun in the post-mortem room. For although there are in the literature some few accounts dealing with difficult labour in dwarfs which can now be judged to have been ateleiotic, yet these accounts are written from the obstetrical standpoint, and though a few particulars of general characteristics are given, no inquiry into the nature of the dwarf growth or its anatomical features, apart from those of the pelvis, was ever made. There was no attempt to study the dwarf growth as such. Again, though there are numerous accounts and representations of ateleiotic dwarfs (as we can now perceive them to have been) in literature and in art, of the dwarfs of courts and those shown in exhibitions, etc., no attempt was made to discover the nature of the defect from which they suffered nor to determine their place in Nature.

In 1868 Schaaffhausen[2] of Bonn described an autopsy performed upon a dwarf who had died in Coblenz at the age of 61 years. The height was 94 cm. (37·6 inches), the total body weight was 45 "Pfund" (46·4 pounds). He had lost several front teeth, but showed no grey hair and was not bald. The permanent teeth began to appear in the 22nd year, and the first to erupt was the first incisor. (Normally the first permanent tooth appears in the sixth year, and the first to erupt is the first molar.) The head and most of the bodily parts had retained the size and proportions of childhood. The dwarf showed no indication of having reached manhood. There

[1] "True" dwarfism or "ateleiosis." The term "true" dwarfism only indicates one symptom, namely dwarfing of growth, of an abnormality of which the cause and the mode of action of this are unknown; and "ateleiosis" is almost as vague. But it serves, until this mode is known, to distinguish the condition from others that have as a symptom dwarfing of growth. An analogous condition in this respect is "cretinism." The dwarfs known as "cretins" were long familiar before the cause of their dwarfing of growth and other symptoms were discovered to be defect of thyroid gland secretion. The term "cretinism" expresses nothing of aetiology, pathology or symptoms, but still survives. And in the case of "ateleiosis" a no less vague term can of course be employed until the pathogenesis of the condition is known. The morbid anatomy is not, as it is in the case of achondroplasia, sufficiently distinctive for a succinct name to be applied to the condition.

[2] Bibl. No. 136.

was bilateral cryptorchism. Spermatozoa could not be demonstrated in the testes. There was no pubic hair. The face, though showing evidence of age and with numerous furrows and wrinkles, was that of a child. The broad bulging forehead, the flat undeveloped nose, broad lower lip and weak chin assisted in producing this appearance. The head circumference of 520 mm. corresponded to that of a boy of five years of age. The head length was 170 mm., which occurs in the first years of life, the breadth 150 mm., a measurement which is found in adults. The brain weighed 1183 grammes and was normal with regard to furrows and convolutions; the brain weight was $\frac{1}{19}$ of the total body weight. In the newborn the normal average of these proportions is $\frac{1}{5}$, in normal adults $\frac{1}{40}$ to $\frac{1}{44}$. The cranium itself was somewhat asymmetrical, with prominent parietal eminences. The sutures, including the spheno-occipital fissure, were all ununited, showed few serrations and these as little marked as is usually seen in skulls of the first years of life. Further post-mortem findings showed: The viscera were of the size of those of a child of about six years of age. The heart showed hypertrophy of both ventricles, with aortic and pulmonary stenosis. There was "chronic endarteritis deformans" and "inter-meningeal haemorrhage."

Some fourteen years later (1882) Schaaffhausen[1] examined the exhumed skeleton of this dwarf. The measurements were:—Femur 220 mm., tibia 160 mm. These correspond to those of a normal child of four and a half years. Length of skull 164 mm., breadth 147 mm., height 121 mm. The first measurement corresponds to that of the skull of a child of six or seven years of age; the two last occur in normal adults. Skull capacity 1390 cm. This figure occurs frequently in lower races and in European children between four and five years of age[2]. Nearly all the epiphyses were still ununited to diaphyses; many could be easily detached. Only two parts of the skeleton were of normal size for adult age, namely, the teeth and the ossicles of the ears. This individual had one normal sister and seven brothers of whom four were dwarfs like himself, and two, who died before six years of age, yet it was clear they would have remained small. The parents were of normal growth and without apparent abnormality of any kind. (The family is described in the Pedigrees. See Schaaffhausen's Case, Pedigree No. 705.)

Thus, on the whole, the appearances presented by this dwarf were those of a growth which had ceased in childhood from about the fourth to seventh year. But the teeth were of adult growth and on this a contradiction to that view occurs; while the fact that some of the cranial measurements were those of childhood and others those of adult years, as well as the date of eruption of the permanent teeth and their order of appearance show an abnormality or irregularity of development beyond simple retardation of growth as a whole.

In 1891 Paltauf[3] described a similar case in his monograph *Ueber den Zwergwuchs*. This was a dwarf named Mikolajek, 49 years of age, born in Andrichau in Galicia. He had been, for 21 years, servant to a colonel of the Austrian Army and had served through two campaigns in this capacity (1859 and

[1] See Bibl. No. 195.

[2] This is hardly correctly put by Schaaffhausen, many female and even male adult European skulls have a capacity under 1400 cms. EDITOR.]

[3] See Bibl. No. 262.

1866) and had subsequently worked as a gardener. He had twice suffered from "rheumatic affections of the knees" and subsequently on two occasions from "general oedema." In both instances this was relieved after a few weeks. Three weeks before coming under notice, however, the general oedema returned, and for it he was admitted to hospital. He died 12 days later. He showed the following peculiarities:—Height (at the age of 49 years) 112·5 cm. taken during life; for the head: horizontal circumference = 540 mm., bi-parietal breadth = 150 mm., mento-occipital diameter = 225 mm.; well proportioned, bones thin and light, but musculature fairly well developed. There was a slight scoliosis with convexity to the left in the upper dorsal region; the lower dorsal and first lumbar vertebrae showed a slight compensatory curve, convex to the right, while the lumbar column showed, in addition, marked lordosis. The development of the external genitals was that of childhood; the prepuce was phimotic; the left testis was in the scrotum, the right in the inguinal canal. Autopsy showed, in addition to dwarfing of growth, "chronic lymphatic glandular tuberculosis, acute disseminated pulmonary tuberculosis, hypertrophy with dilatation of the right side of the heart, fatty degeneration of the myocardium, and recent haemorrhage into the pons Varolii." The total body length after death was 111 cm.; the body was rather thin and markedly oedematous; the bones were very thin and light. The head was relatively big, the face short and broad, with prominent malar bones; the bridge of the nose was depressed, broad and saddle-shaped; the nose itself was blunt. The neck was short, the thorax fairly convex, at least in no wise flat; the abdomen was hemispherically arched forwards. The external genitals showed the development of childhood; the prepuce was very long, the urethral orifice much contracted. The feet and hands were very small. The cranial measurements were:—horizontal circumference 506 mm., horizontal length 169 mm., maximum transverse diameter 142 mm. It was roughly rhomboidal on transverse section, but slightly asymmetrical, the left half being somewhat in advance of the right. All the normal sutures were present; the frontal suture was still present. The cranial contents and the abdominal and thoracic viscera showed the appearances already stated. Apart from general oedema and other changes stated there was no noteworthy abnormality in any part except the skeleton. The thyroid gland was, however, "very small and pale red."

A complete description of the skeleton with very full measurements of all its parts is given by Paltauf. These need not be reproduced here. It is enough to say that, as a whole, compared with the normal, the skeleton corresponded to that of a boy of seven years of age. The skull at first sight appeared disproportionately large, but the appearance was deceptive. All the fissures, emissary foramina and canals at its base were abnormally patent or large. *The sella turcica (or cavity of the hypophysis cerebri or pituitary body) was very large in all dimensions*, not only as compared with that of a child of the same stature, but the measurements were considerably greater than the average of those of several adults of normal growth. The sphenooccipital junction was still un-ossified and was cartilaginous. Both the sphenoid and the basilar portion of the occipital bone showed measurements markedly smaller than

those of a child of the same height. The foramen magnum, from front to back, was on the contrary very large. The upper jaw was relatively big and markedly prognathous, the alveolar processes very powerfully developed and in the formation of the teeth-sockets as well as of the bony lamellae was that of an adult. The lower jaw was also comparatively strongly developed, with well-marked ridges for muscular attachment. In the upper jaw the third molar on both sides was partially erupted; in the lower no trace of these was to be seen, the second molar lying in contact with the coronoid process of the jaw. The teeth (all of the permanent set) were all sound, without evidence of rickety or other malformation, and were proportionately large, as of an adult.

In the vertebral column the centres of ossification were ununited by bone and the epiphyses for the spinous and transverse processes had not appeared at all, these parts being still cartilaginous. The sternum consisted of several plates of bone united by cartilage, as in the child. The clavicles, like the lower jaw, were relatively strongly developed, short, thick and curved with for their size pronounced markings for muscular attachments. The scapulae showed the condition of childhood, the epiphyses being united to the body by cartilage, or in some instances no centre of ossification had appeared in these. The bones of the arm and forearm, though relatively a little shorter and a little thicker than those of a child of seven years, presented in other respects the appearance of that age. Epiphyses and diaphyses were still united by cartilage and not by bone. The same was true of the metacarpal bones and those of the digits, of the thigh and leg and foot. The bones of the carpus and tarsus, however, all present in normal number, suggested adult growth by reason of the sharpness of their moulding. Both patellae were present and of normal development for size and age. In the pelvis the sacrum was markedly inclined forwards. The first sacral vertebra, however, did not take part in the curvature of the sacrum but was, as it were, a continuation of the line of the lumbar vertebrae, the promontory was formed by the second sacral vertebra, which was separated from the first by a cartilaginous disc of 13 mm. in thickness. The ilium, ischium and pubis were ununited to each other, and the pubo-ischial junction, which after the sixth year normally becomes united by bone, was still cartilaginous. All epiphyses were united by cartilage alone.

These two cases, which are alike in all essential particulars, appear to be the first instances of this variety of dwarf growth to be completely investigated from the standpoint of science. Paltauf quotes Schaaffhausen's case as resembling his own and refers, in more or less detail, to other cases, described by various observers, which show dwarf growth associated with delay in processes of ossification, as shown by lack of union of epiphyses and diaphyses in adult years. Amongst these was Bobbie Fenwick, whose skeleton is in the Museum of the Royal College of Surgeons of Edinburgh and is clearly of the same nature as his own case; but one of the others was an instance of cachexia strumipriva (Grundler's case[1]); another appears to have been a cretin (His's case[2]); and a third (Schauta's[3]) seems very doubtful. Paltauf clearly distinguished between the condition illustrated by his

[1] See Bibl. No. 212. [2] See Bibl. No. 123[b]. [3] See Bibl. No. 249.

case and such conditions as rickety dwarfism, achondroplasia, etc., and the classical type of cretinism (as regards their skeletal features with which he is mainly concerned); but it is not clear that he knew how to distinguish all types of the thyroid gland group from the condition represented by his case, and, indeed, the thyroid gland group was, apparently, at that time not so fully understood as it has since become. In 1891 A. Schmidt[1] described a case (Theresa Fend) which appears to be certainly of the same kind as those described by Schaaffhausen and Paltauf. In 1896 Manouvrier[2] described what appears to be another, and in 1899 Joachimsthal[3] published details of several more. There are a good many other instances of the same condition in the literature, many of which are shown in the Pedigrees, but the above appear up to the present (by reason of the completeness of their descriptions) to be the most important as enabling us to form a picture of the condition. Thus far cases had been described from time to time; but it was not until 1902 that these were grouped together, and the condition and its relationships were indicated. This was done by Hastings Gilford, and his memoir is no doubt an important contribution to this subject. He examined two skeletons and four living cases, and made one post-mortem examination. It was he who first defined the condition in all its details and gave it a name ("ateleiosis"; Greek ἀτελείωσις, "not arriving at perfection"). He grouped the cases, showed the clinical history of the disease and pointed out the manner in which the condition differs from other varieties of dwarf growth and established the diagnosis. It is owing to his work that the present section of this paper has been written, and I should wish to state this as clearly as possible and express my great indebtedness to this author[4].

Hastings Gilford (*loc. supra cit.*) discussed four new cases. In the first, which had already been described by Home, but not as ateleiotic, the growth change began during foetal life and the child was greatly undersized at birth—a dwarf infant. The second, a male aged 28 years, was 3 feet 7 inches in height at the time of description. He was of average size at birth but it was observed that he was not growing when he was between one and two years of age; there was no illness or other known cause to account for this. At 23 years of age his height was 3 feet 6 inches, one year later it was $\frac{3}{4}$ inch more; and three years later it had increased to 3 feet $7\frac{1}{8}$ inches. One year later his height was still the same. He was of average intelligence, but of the general appearance of a boy of about six years; showing, however, bronzing, wrinkling and other signs of age. Both testicles were undescended, occupying the inguinal canal; he showed infantilism, etc. Ossification, at least of the bones of the hand and forearm, radiograms of which were taken, corresponded to that of a child of about 10 years of age; none of the epiphyses were united to diaphyses by bone. At the age of 23 years all the permanent teeth on both sides of both jaws were present, except the wisdom teeth. The permanent canine teeth, however, appeared to have only just erupted and the temporary canines were still present. Five years later the condition was the same except that the temporary canine teeth had been shed.

[1] See Bibl. No. 270, S. 59. [2] See Bibl. No. 324. [3] See Bibl. No. 363.

[4] See Bibl. No. 403. [Gilford's views are developed more at length in a recent treatise: see Bibl. No. 604. They should be studied in conjunction with the very fine work of the French school on infantilism and true dwarfism. EDITOR.]

The third case was a female aged 18 years, whose height was 2 feet 9½ inches. She was small at birth but grew at an ordinary rate until she was between two and three years of age. After this she still continued to grow, steadily but at a much diminished rate. She resembled a child in nearly all respects, general appearance, infantilism, etc., but her intelligence was normal for age. Teething began in the eighth month, but it was not known when the permanent set began to appear. The teeth were very irregular and some were of the temporary and some of the permanent set. Radiograms showed that ossification of the hands was a little more advanced than in those of a child of six years.

These two cases of Hastings Gilford clearly represent the same type of case of ateleiosis as is shown by Schaaffhausen's and Paltauf's cases. This group, in which the growth change began in infancy or early childhood, he terms the second, and it appears to be much the largest group of all. Cases show, in adult years, the stature, general proportions and facial appearance of young children. The first group is that formed by cases in which the growth change began in foetal life. This is instanced by the case of (1) Caroline Crachami, the photograph of whose skeleton is shown, Plate AA. She was first described by Sir Everard Home[1], but it was Hastings Gilford who located her amongst the ateleiotics, as an instance of the most extreme type (or Group I) of this condition. This observer regards (2) Frank Flynn, "General Mite" (examined by Virchow and by Ranke and von Voit[2]), (3) Millie Edwards (Ranke and von Voit), (4) Pauline Muster (Virchow, Bouchard[3]) as possibly of the same nature. The greatest measurements recorded of these individuals were: (1) At nine years of age, 19⅓ inches (approximately), a little less than that of a normal child at birth but with head a little bigger. (2) In adult years (exact age uncertain) 32½ inches, weight 14 lbs. 7½ ozs. (3) At 12 years of age 24¼ inches, weight 27 lbs. (4) At five years of age 21⅞ inches. These measurements, though very incomplete, suffice to show that the members of this group have on the whole considerably greater defect of growth than those of the second. In the case of (2), Frank Flynn, the only adult of the group, it was said by Virchow that his head was too big for his body but that he was otherwise well proportioned.

Hastings Gilford's third group is composed of cases in which the developmental change first becomes apparent between the ages of infancy or young childhood and puberty. An instance of this is Hastings Gilford's fourth case, in which it was observed by the mother that growth seemed to have stopped (without known cause —he had had no illness and showed no taint) at about the age of 13 years. The height, at the age of 28 years, was 4 feet, 9½ inches, and the weight 79 lbs. "He showed the proportions and appearance of a lad of 14 years, but the skin of his face was more rough and weather-beaten than one ever sees in a youth." The results of a post-mortem examination are recorded. The only essential differences between these and those shown by Schaaffhausen's and Paltauf's cases were (in addition to those stated, *e.g.* appearance of boyhood or adolescence rather than of infancy or early childhood, considerably greater stature, etc.) that ossification and

[1] See Bibl. No. 58. [2] See Bibl. Nos. 200 and 216. [3] See Bibl. Nos. 196 and 214.

general development, though delayed, were considerably more advanced (to the normal for 14 years of age, rather than that for seven years, *e.g.*). Again, though there was "infantilism," the right testis was completely descended and the left occupied the inguinal canal, whereas in Schaaffhausen's case there was bilateral cryptorchism. The defects were precisely the same in kind but less marked in degree in all respects. Hastings Gilford suggests that there is possibly a fourth group in which the developmental change first shows itself after puberty; that, if it exists, it will be a less clearly marked group than the third and will tend to become more obscure as age advances and difficult to distinguish from individual variations of growth and development within the normal limits.

These cases demonstrate clearly the features which serve to distinguish the three groups of Hastings Gilford from one another; Group I in which the growth change begins during foetal life and is apparent at birth: Group II in which it becomes apparent in infancy or early childhood, and Group III in which this occurs later but before puberty. They show further that the facial appearance, height and general development, in adult years, correspond, broadly, to those of a child of the particular age at which ateleiosis appeared.

Many examples of these groups are shown in the illustrations (Plates AA—FF, HH), and these, with the cases of Schaaffhausen, Paltauf and Hastings Gilford above described demonstrate the features of ateleiosis sufficiently fully for a picture of the condition to be formed. There are many other cases, either certainly or probably ateleiotic, in the literature. These have been summarised by Hastings Gilford (*loc. supra cit.*) and need no further reference here. Some of these are described in our Pedigrees.

The following short description of the features of the condition seems, however, desirable; and in this the common, second, group alone will be dealt with; for the manner in which examples of the other groups differ from those of this group has been shown.

The facial and general appearances are those of infancy or early childhood[1]. The face is short and broad, the head proportionately large, high and quadrate with prominent eminences. The bridge of the nose is somewhat depressed, broad and saddle-shaped, the nose short and undeveloped. The lower jaw and chin are small as in childhood. There is usually no hair about the face; when, however, this is present the growth is thin and weak and the facial appearance apart from this is the same. The neck is short, round and thick, as is seen in the young child. The general bodily proportions are those of normal childhood; that is to say the limbs are a little shorter than in the normal adult so that the midpoint between the

[1] With regard to the facial appearance of this variety of dwarfism, Hastings Gilford (*loc. supra cit.* pp. 345—6) says:— "The facial type is so distinctly childish that it is probable that ateleiotic dwarfs of the second class may be distinguished from all other dwarfs by their physiognomy alone." Probably anyone who has seen many of these cases would agree that a typical case presents no resemblance to any other variety of dwarfism. The face has been described as "cretinoid," but if this is meant to imply that these dwarfs look like cretins it conveys a wrong impression. A glance at the illnstrations will show this. The use of the term probably arose from the general similarity in the form of the head in the two conditions (dependent no doubt upon persistence of immaturity in both). There are doubtful cases, in which the distinction is difficult to make; but the face of typical cases of ateleiosis presents no resemblance to that of cretinism, infantile myxoedema or any other form of dwarf growth.

vertex and the soles of the feet is a little above the upper border of the symphysis pubis, whereas in the normal adult it is at that point. The proportionate length of the segments of the limbs to one another is also normal, that is the proximal segment (femur or humerus) is longer than the mesial (tibia or radius). The hands and feet, and the nails of these, have the shape and appearance of those of childhood. There is no deformity of any kind. The larynx is small and undeveloped as in childhood, the voice high-pitched, thin and sometimes squeaky, but with more timbre than that of a child of the same size. The thyroid gland can be felt. The skin though it shows the markings of age (wrinkling, bronzing or weather effects) is not thickened, dry or in any way abnormal, nor are the subcutaneous tissues increased. The hair of the head is also in no way abnormal. There is usually infantilism (cryptorchism, etc.), but this is not invariable, and there are several authentic cases of these dwarfs having produced offspring. The intelligence is normal and of the average for age. It is sometimes stated that these individuals are of shy and diffident disposition and lacking in self-reliance. But this is not invariable nor perhaps more frequent or more marked than is seen in individuals of ordinary growth. When present it seems, very probably, to be the result of the peculiar environment in which these dwarfs spend their lives, and in reflex response to these surroundings. For it appears probable that most people would show evidence of shyness, diffidence, lack of confidence, etc., if they had been objects of curiosity or hilarity all their lives and were only of a stature of, say, 3 feet and of weight, say, 40 pounds. That is to say their mental attributes are probably not infrequently, when present, acquired characteristics and not, properly speaking, part of the disease at all. The musculature, though proportionate to size, is weak and shows the lineaments of childhood. (The skeletons that have been examined also show that the ridges, grooves and points of muscular origin and insertion are very feebly marked, though more pronounced than in the skeleton of a child of the same size.) The physical strength of these dwarfs is necessarily very small though probably, on the average, greater than that of children of the same size; and their endurance is certainly, on the average, considerably greater; for they frequently perform daily and regularly as acrobats, dancers, etc., and this no child of, say, four years of age could do. This greater endurance may, however, be the result of careful training in isolated cases. One case that I have seen had been trained as a "strong man." With a total body weight of 40 pounds he could support some 250 to 300 pounds by "making a bridge." He was very muscular (the outlines of the muscles were, however, even in this case, those of childhood and not of adult age. His age was 24 years). Paltauf's case (Mikolajek) is also stated to have been very muscular, and Hastings Gilford (*loc. supra cit.*) quotes another case of a "strong man" who was very muscular. This is certainly very unusual and probably the effect of occupation or of special training when it occurs.

Dentition. In all the few cases, some eight in number, that I have been able to examine on this point, dentition appeared to be normal, as it frequently is in ateleiosis. But in the above eight no reliable history as to the time of eruption of either milk or permanent set could be obtained. In three of these cases, each of about 60 years, all

the permanent set were present, none were carious and they appeared little worn. Hastings Gilford considers that the explanation of this occurrence (which is at least unusual in full-grown individuals of this age, but which has been noted as frequent in ateleiosis) is to be found in the fact that they have appeared late, and this explanation was suggested in Paltauf's case. "The teeth are, as a rule, decidedly backward in development, though they are of ordinary size" (Hastings Gilford). In Schaaffhausen's case the permanent dentition was delayed and irregular but the teeth were of ordinary size. In Paltauf's case none of the third molars were completely erupted but the teeth were all sound and of ordinary size. In the case of "Tom Thumb" (quoted by Hastings Gilford) "there was a double row of teeth all round." In that of Bobbie Fenwick "dentition was very irregular owing to the late appearance of several of the teeth." In Hastings Gilford's own second case, quoted above, "the milk teeth persisted side by side with those of the permanent set."

Naked-eye Appearances and Characters of the Skeleton. These, at the present day, are perhaps the most important points for study if we wish to attain any knowledge of the essential nature of the condition, or at least as showing how markedly it differs from achondroplasia, rickety dwarf growth, etc. But these features are more easily and advantageously studied in the actual accounts of cases given by Schaaffhausen, Paltauf and Hastings Gilford, quoted above, than in a general description of the osteology; in order to emphasise the importance of this aspect of the subject those cases were quoted at the beginning of this paper and quoted fairly fully.

Microscopical Appearances of the Skeleton. Paltauf's description of the microscopical appearances (*Ueber den Zwergwuchs*, S. 46—52) is as follows: "For microscopical examination plates were sawn out of the fresh bone to include the epiphyseal line and the parts on either side of this; these were decalcified and imbedded and sections were then cut. Sections from the following bones were examined: Humerus, head of femur, lower epiphysis and great trochanter of femur, tibia. Stains: Haematoxylin, Eosin and Carmine. Examining with the naked eye a section, coloured by the first two stains, from the epiphysis of the femur for example, a double coloration of the epiphyseal line is to be seen. This line is represented for the most part by a band staining red, but this is accompanied by a very fine bluish border which is not equally broad throughout, is finely indentated and finally, in some specimens, breaks through the red band here and there. Under the microscope there is on the whole a similar division of the staining of the preparation but naturally in addition many other appearances are seen.

"The cartilage of the epiphyseal line, followed from the outer end of it, that is, corresponding to the surface of the bone, presents most externally a layer of loose wavy connective tissue, which soon becomes denser, firmer and fibrillar. These two connective tissue layers are continued from the part of the preparation belonging to the diaphysis and are continued further over the zone, which represents the cartilage of junction, to end finally on the articular cartilage of the joint, and this, finally, marks the contour of the section. The dense-fibred connective tissue covering becomes looser in texture where it covers the diaphysis, becomes richer in cells and

more vascular, becomes in short the periosteum of the cortical layer of the immediately underlying bone-shaft. Just at the level of the zone in which the epiphyseal cartilage becomes marked off there appear in the fibrous tissue elongated spindle-shaped cells parallel to the long axis of the bones, at first single, then in groups, each with an elongated granular nucleus and clearly defined outline. The further inwards one follows, the larger do these cells become and the larger do their nuclei become; the cell as a whole appears to lie in a cavity. The surrounding intercellular tissue has at the same time lost its fibrillar character and has assumed that of a perfectly hyaline intercellular tissue staining diffusely bluish red. Still further towards the axis of the bone, where the epiphyseal cartilage begins to show itself in the preparation as free trabeculae between the substance of the bone marrow of the epiphysis and diaphysis, the above cells are still more numerous and lie in twos, threes and fours; they are larger, spherical, possess an obvious cell membrane and lie in cavities in the intercellular substance. Where several cells lie together they are flattened at their points of contact and form small groups or almost rows of oval cells, the long axes of which are transverse or at least somewhat oblique to the inner border of the cartilage. The intercellular substance here shows, with clearly marked cell-systems, a homogeneous area around the cell itself, but otherwise a fine fibred or reticular or granular appearance. An intense blue clearly defined staining of the intercellular substance, which can be traced back to the decalcification of the cartilage bordering on the bone marrow, suddenly appears. This blue margin is thin, often interrupted and contains collections of cartilage cells clearly recognisable as such, which in some cases are hyaline and glistening and in others nucleated and stained blue; these cells contain, in addition to the relatively large, in part oval and in part serrated nuclei, small particles which very deeply absorb the stains. The cartilage cells are, in this situation, not more densely crowded, but are of greater size than in the regions hitherto described.

"Following the section further it is seen that the substance of the investing cartilage is continued without interruption into that of the epiphyseal cartilage and that both are of similar microscopical structure. The histological nature of the cartilage of the epiphyseal line (or line of conjugation) is not, however, uniform, but shows different features at different parts of the disc, or of the section. The intercellular substance of the cartilage is, on the whole, of hyaline character; but this hyaline material is in numerous places displaced by a fibrillar or granular structure. The fibrillation of the cartilaginous matrix is in places very faint, in others clearly shown. The fibrils are not always regular in arrangement but in places wavy or reticular; in other places the fibrillation is coarser and changes the whole appearance of the matrix. The least marked deviation from the purely hyaline condition of the matrix is to be seen in a very fine granulation of this. It is only around the groups of cells and in the middle of the cartilage of the epiphyseal line as a whole that the cartilaginous matrix has a hyaline structure. The epiphyseal side of the cartilage discs shows the changes in the ground substance that have been indicated, in slighter degree, but especially there is in rare places a fibrillation with direction at right angles to the long axis of the cartilage. The side facing the diaphysis shows, on the contrary, a much more mixed

and more changeable form of structure of the ground substance, the hyaline cartilage is here solely confined to the neighbourhood of the cells and cell groups that here occur, it forms concentric areas of small thickness around these and these are isolated from one another by a matrix or ground substance which may be granular, fine, striated or fibrous and is in places so coarse-fibred that it has much the characters of fibrillar connective tissue. The fibres are for the most part directed in the long axis, but are throughout accompanied by oblique and transverse fibres, so that an irregular field is produced in which the cartilage cells are scattered. These transverse fibres appear in the centre of epiphyseal cartilage but are there faintly indicated; towards the bony margin they become more numerous and coarser, to form, here, coarse fibrillar arrangements between which cartilage cells are enclosed. These fibres pass, at the border of the layer of calcified cartilage, directly into the ground substance of it.

"The cartilage of the epiphyseal line is throughout studded with cells and cell groups, which according to the region under examination show numerous differences in size, form and arrangement. In the middle region of this cartilage of the epiphyseal line the cells much resemble those of the cartilage covering already described; elongated, spindle-shaped cells with granular nuclei which for the most part lie in groups and which become the bigger the nearer they approach the surface of the cartilage, and also undergo such an obvious change of form that one can here recognise in them the typical form of cartilage cells: globular, glistening cells with somewhat distorted nuclei and obvious, clearly defined cell borders; the cells lie in places singly, in places in oval groups or rows, in common cavities with hyaline or granular blue stained cell substance and with large nuclei. On the side of the cartilage of the epiphyseal line which faces the epiphysis the above cell-formation only occurs rarely and is not well shown. On the opposite, diaphyseal, side there are more groups, and these contain more cells. Amongst the well-formed cartilage cells there are also some to be found which are scarcely of this nature, since they are elongated, spindle-shaped, indentated and show processes. For the most part, also, they lie several in a group, and this circumstance, as well as the fact that they lie free in the cartilage matrix, indicates that they only represent altered cartilage cells, and this is also shown by the peculiarity that cell groups of this kind, when they lie at the margin of the layer of calcified cartilage, show portions which are without doubt those of cartilage cells.

"From these malformed cells all transition stages to such as can be regarded as normal and still capable of function can be seen. Cells of the latter kind are very numerous towards the diaphyseal side of the cartilage of junction, and form here groups and columns which can scarcely be distinguished from the columns of cartilage cells which distinguish the layer of proliferation of cartilage cells in the epiphyseal cartilage of the growing child.

"The ends of the cartilaginous part of the epiphyseal junctions are invested in their whole extent by a border of calcified cartilage, which has already been described. The marking off of the two otherwise regular layers is extraordinarily sharp, and they can very easily be separated from one another, of which one has evidence only too

frequently in the preparation of the microscopic section. The calcified cartilage on either side of the cartilage of the epiphyseal line shows the same qualitative relations; the only differences are that whereas this border on the side of the epiphysis is thin, often interrupted and especially poor in cell elements, is indeed almost lacking in columns of cells, that facing the diaphysis is very rich in cells, much broader and rich in columns of cartilage cells, and these sometimes consist of twenty or more cartilage cells regularly arranged. (In this way, also, the thickness of the layer is indicated.)

"I should like here to state that this calcification of cartilage is not in the least to be confused with that impregnation with calcareous salts in which, during the process of ossification, the precursor of cartilage transformation is to be seen, but that it is much more to be compared to the calcification which is well known as an age-change of cartilage (which occurs in the costal cartilages, *e.g.* in aged persons); what has already been said will suffice to prevent such a mistake arising; but there are other indications of this.

"The side of the border of calcified cartilage which lies next the bone is serrated or indented; it shows lacunae and crypts, possesses the contour that we know in normal processes of ossification, but from which the condition of the underlying cartilage clearly distinguishes it, so that neither marrow nor cartilage cells but only a proliferative change can be recognised. The cartilage is covered over with osteoid and real bone tissue, the marrow spaces—of primary marrow spaces one cannot speak—are filled with marrow substance like any part of the bone. The bone lamellae of the marrow (trabeculae) are thin, delicate, show many lacunae between them, and enclose, for a distance of 1 cm. or thereabouts, from the cartilage of the epiphyseal line, calcified cartilaginous rests with or without cells.

"At the site where the cartilage of the epiphyseal line becomes attached to the bony cortex the margin of calcified cartilage is continued along the cortex of the diaphysis for a short distance.

"The picture of the histological appearances of the cartilage of the epiphyseal line here described holds in general for all the bones examined, so that in order to describe the condition shortly, but as far as possible completely, I may confine myself thereto, merely showing further peculiarities of this or that region.

"Thus the cartilage of the epiphyseal line of the lower epiphysis of the femur shows a thickness greater than any other, as great as 3 mm., and a markedly zigzag form. The whole epiphyseal cartilage lacks the small cells almost entirely, containing throughout large cartilage cells which, on the diaphyseal side of the cartilage of the epiphyseal line, are in such numbers and are arranged in such clearly defined columns, that one might suppose the section to be one of an epiphysis of a normal child, if it were not that in this case, more prominently than in all other preparations, the calcification occurs and occupies here half the breadth of the entire cartilage of the epiphyseal line. This is, in the same way as in the other dwarf specimens, bridge-like and completely transverse; and these appearances show themselves with the same regularity as in the other preparations. The striated appearance of the intercellular

substance is here more marked; the bundles of fibrils form columns and arches along and over the columns of cartilage cells and the immediately surrounding matrix, and form a system, somewhat resembling that of the lamellae of the bone marrow, and possibly like it, of some mechanical importance." (The epiphysis of the great trochanter, and of the tibia, showed minor differences from the above, which need not be detailed here.)

"The following comparison between the appearances shown at the epiphysis of an ateleiotic dwarf and that of a child of normal growth of the same size is important.

"The cartilage disc is, absolutely, broader in the child than in the dwarf. The general arrangement of the cartilage, with its intercellular substance and cells, is the same in both; the region of union is, however, different in that that of the child is much more cellular. The matrix shows a slight fibrillation between those cell columns which are nearest to breaking down, a process of disintegration which also occurs in the dwarf. In the case of the child, however, the cells show an appearance suggestive of active life and growth. The contrasts with reference to the epiphyseal and diaphyseal sides of the cartilage of the epiphyseal line are of a similar kind to the above in the child; but diffuse impregnation with calcareous salts occurs in the case of the dwarf. The cartilage cells arranged in typical manner in rows, according to their different degrees of development in the changes of bone formation with, as the last steps of that preparatory change, swelling, calcification, and finally invasion by marrow capillaries and erosion or solution; this is the condition in the normal child.

"A comparison of preparations from the dwarf and from children shows that the zone of calcified cartilage, in part occupied by columns of cells and in part disintegrating, corresponds to that layer of a bone undergoing normal ossification which directly adjoins the margin of the cartilage; but that it is distinguished from the normally developed in this: that in the case of the latter the cartilaginous remnants are inferior in quantity to the already formed bone substance, so that between the trabeculae thin serrated calcified cartilaginous remnants can be discovered; while in the case of the former, the dwarf, these calcified cartilaginous remnants have been preserved to a striking extent. While normal ossification at once lays down real bone substance on the remnants of cartilaginous matrix which for the time are stationary, in the case of the dwarf we find a thin interrupted layer of osteoid tissue on which afterwards real fibrillar bone forms itself.

"A further marked difference between the histological conditions shown by the epiphysis of the dwarf and that of the child is that in the latter the advancing development of the cartilage cells can be followed step by step, while in the case of the dwarf there is seen, amongst the small-celled middle layer of the cartilage, large spherical cartilage cells or only larger rounded cells, mostly in groups; there are thus, in the cartilage of the epiphyseal line of the dwarf, cells belonging to different stages of development mingled together, while in the case of the normal child of the same size, in the normal processes of cartilaginous development, a definite train of changes

in developing cells can be followed until the last stage of transformation to bone is reached. Cartilaginous inclusions in the bony trabeculae of the spongiosa occur in both cases, as the continuation of the last layer of the cartilage of junction with the first layer of the diaphysis; the description given above applies also to the cells found here."

Essential Nature of the Condition; its Aetiology, etc. As concerns causation nothing certain is at present known; but in a considerable proportion of cases evidence of the "hereditary" or "family" influence is forthcoming, as a reference to the pedigrees recorded below will show. All known causes of secondary dwarf growth and the causes or associations of other conditions of "infantilism" discussed on page 368 can be excluded. As we have stated, however, "infantilism," though usual, is not invariably present in ateleiosis: see p. 394).

The following views as to the nature of the condition have been held:—

I. The defect of development is "germinal." That is to say the developing ovum possesses an inherent general, but possibly minor, defect, so that, although at birth and, for a time subsequently, growth and development appear perfect, yet the constructive anabolism of the organism does not continue to prevail over katabolism during post-natal growth and development; so that, after a period, and without adverse influence of any kind, growth and general development become quiescent and cease to continue through puberty and adolescence until adult years, as they normally do. The average normal development of adult years is thus never attained. Normal development may be supposed to depend upon three motive forces:—(1) architectural or plastic, determining the growth in form of individual parts; (2) that which determines growth of these in size; (3) that which determines the continued growth of the organism as a whole. In this view defect of (1) or (2) will produce deformity; defect of (3) will produce dwarfism.

In the above view ateleiotic dwarfs are children or infants that have never grown up. This in itself constitutes an abnormality, but the view seems to presuppose that growth and development, as far as they go, are normal but have stopped short before their time. This, however, is disproved by the following facts, which show that the condition is an abnormality of development in a still further sense; for while some parts have ceased to develope, others have continued to do so:—(*a*) these dwarfs are in adult years, intellectually, not children but adults; (*b*) they do not always show "infantilism"; (*c*) in cases where they do show this, with *e.g.* cryptorchism or incomplete descent of testes, this condition corresponds to a pre-natal stage of development, whereas other features correspond to those of a later age. For instance, in Paltauf's case the right testis was in the inguinal canal, the left was completely descended. Development thus corresponded, in this respect, to that of a foetus of about eight months (but was still further abnormal in the fact that the right testis normally descends about one month before the left), while the general features of development were those of a child of seven years. Schaaffhausen's and Hastings Gilford's cases already quoted also illustrate this point. (*d*) As regards growth, particularly that of the bones of the skull, to take Paltauf's case, while those of the

base showed measurements less than those of a child of the same size, those of the vault showed in some cases measurements such as are found in adults, which indicates that growth was irregular and abnormal; (*e*) the frequent irregularity of dentition suggests abnormality; the usual final appearance of all the permanent set except the third molars shows, in this respect at least, a continuation of development past that of childhood; (*f*) the microscopical appearances of the bones at the epiphysial lines, etc., are not those shown by the normal child of corresponding size, but are abnormal. Again, the ridges and grooves for muscular attachment on the bones are more marked than on those of a child of the same size. The development of some parts is thus more advanced than that of others.

Since, therefore, some parts show the development of adult age, some that of childhood, others that of foetal life, while the microscopical appearances of the line of ossification suggest abnormality, the condition cannot be regarded simply as one of retarded development, but must be supposed to be one of abnormal development, retarded as a whole though it be.

II. The condition is essentially one of defective bone growth and development. It is a bony "dystrophy" or "dysplasia"; that is to say it is, strictly speaking, a local condition. This view cannot be definitely refuted at present for lack of full knowledge. It appears, however, improbable, and the evidence for it is not apparent.

III. The skeletal peculiarities are primary, the others secondary to this condition. In this view there is some primary disease of bone which exercises a deleterious influence upon development as a whole. Against this view are: (1) The fact that none of the changes seen in the bones are pathological. The bone marrow and the cortex of the shafts are normal. The changes seen about the epiphyses and beneath the periosteum of the shaft are the same in kind as those which occur in childhood (seen, however, in these cases in individuals of adult years and with minor differences in the direction of defect), or as the calcification of cartilage that normally occurs in the aged (occurring here, however, in cartilage which has persisted abnormally unossified). None of the changes are pathological. (2) The fact that some features, *e.g.* cryptorchism, are present at birth, but no evidence of bone defect occurs until later.

The above views can probably all be dismissed as fallacious. One can only suspect that the features, one and all, of this condition are secondary effects. The primary cause might be supposed to be (IV) a bacterial infection. For this there is no evidence, and none of the features of the condition suggest it.

V. A maternal intoxication, placental or other. If this occurs its action must be, usually, delayed for years. Nothing is known upon this point, and there is no recorded evidence of placental abnormality in these cases. Occasionally ateleiosis is present at birth, and this, together with the fact that the condition fairly frequently occurs in more than one member of the same generation, is the only feature suggestive of this view. But cases in which ateleiosis occurs in father and son (as in two instances in the pedigrees) cannot be explained in this way. The view is entirely fanciful.

VI. Some intoxication from without or an auto-intoxication other than such as is due to abnormality or defect of some internal organ. Nothing is known of any such influence.

VII. All the features of the condition are produced by defect or abnormality, of one or another kind, of some internal organ. Analogies drawn from conditions of dwarf growth such as cretinism and such conditions as show "infantilism" produced by such defects suggest plausibility for this view. That ateleiosis is secondary to abnormality or defect of one of the viscera seems the most probable explanation of it at the present day. It appears possible to exclude, however, the pancreas, intestine, spleen, suprarenal body, kidney, thymus, and most other causes, enumerated under the heading "Infantilism." These produce infantilism and defect of growth, but not ateleiosis. Defects of thyroid gland in infancy produce cretinism and infantile myxoedema, conditions which in some of the gross features resemble ateleiosis; and occasionally cases occur in which the diagnosis between infantile myxoedema and ateleiosis cannot at once be made. This is, however, very exceptional and as a rule no such resemblance exists; and although in Paltauf's case it is stated that "the thyroid gland was small and pale red," this cannot be held to imply that it was in any way deficient in action. The case showed no evidence suggestive of this. It is the only undoubted ateleiotic case at all fully described in which the thyroid gland could be under suspicion. There remain the testis (or ovary) and the pituitary body or hypophysis cerebri. With regard to the generative organs the fact that "infantilism" with (in the male) cryptorchism so frequently occurs in ateleiosis seems suggestive, because cryptorchism is the first indication of any abnormality; it is present at birth while other defects are only observed later. But that this, the first apparent defect, is a cause rather than one of the effects of some other obscure cause seems improbable. The effect upon growth and development of removal or defect in early life of the sexual glands is not clearly known. "Infantilism" is produced; but that dwarfing of growth and development in other directions always follows does not appear to be the case. Nor are the skeletal features apparently like those of ateleiosis. (The beneficial effect of ovariotomy in mollities ossium or osteo-malacia does not appear, for obvious reasons, to have any bearing here.) Paltauf discusses this question at some length, but without bringing its answer any nearer. On general grounds it seems fair to suppose that the defective development of the sexual system that is usually present in ateleiosis is, as in cretinism, one of the general effects rather than a cause in itself.

There remains, as the possible primary cause of ateleiosis, abnormality or defect of the pituitary body or hypophysis cerebri; and this, at the present time, seems on the whole the most probable. On the one hand the condition seems to represent the opposite pole of abnormality as regards *growth* to that shown by pituitary gigantism, such as that shown by the skeleton of O'Brien in the Museum of the Royal College of Surgeons of England, while in other features, such as "infantilism," defect or delay of union of epiphyses, persistence of growth after normal growing

years, asthenia, somnolence, etc., the two conditions appear to show similarity. On the other hand, in Paltauf's case it is clearly stated that the cavity of the sella turcica was much enlarged, and that the pituitary fossa showed measurements which were, in all directions, greater than those of the mean of several adult skulls of average size, whereas the rest of the sphenoid bone showed measurements which were less than those of the skull of a child of seven years (of a child of a height, that is, equal to that of Paltauf's dwarf). It is to be supposed, therefore, that in this case the pituitary body was considerably enlarged; but there is no note of the pituitary itself having been examined. This the only definite evidence that there is upon this aspect of the subject. The number of autopsies made has been very small, and in none of them except the above instance was this point investigated. In Hasting Gilford's autopsy the pituitary fossa appeared normal, but the hypophysis cerebri was not examined. So that the view that ateleiosis is due to abnormality or defect of the pituitary gland must at present remain hypothetical; but it appears probable at the present day.

As regards growth and development as a whole it seems clear that neither is absolutely stopped but both are indefinitely retarded or reduced to a minimum. Hastings Gilford (*loc. supra cit.*) quotes several cases in which growth, having apparently ceased, began again and slowly continued, although at a much diminished rate, until late in life. Ateleiotic dwarfs appear to be capable of growth until quite late in life—up to 30 years or over. (See Jeffrey Hudson (p. 360), Joachimsthal's cases (Bibl. No. 363), etc.)

Abnormalities found in Association with Ateleiosis. It has been stated that what would appear to be *causes* of dwarfism and of "infantilism," as discussed on pages 364 to 365, can in all cases be excluded in ateleiosis. There are, however, some cases recorded (doubtful, it is true) in which what may possibly have been ateleiosis was associated with some other condition, possibly without any relationship of cause and effect.

(1) Paltauf (*Ueber den Zwergwuchs*, S. 41) quotes a case (Helm Gottfried), which possibly showed ateleiosis with osteo-malacia. This may have been an independent association of two conditions; but it appears much more probably to have been one of dwarf growth and "infantilism" secondary to osteo-malacia with onset early in life, *i.e.* in childhood. (2) Paltauf (*loc. supra cit.* S. 37 and S. 38) quotes a case (König's) which was probably one of ateleiosis. Death occurred at the age of 18 years from cerebral tumour (cysticercus cerebri). There were osteo-chondromata on ilia, ischia and pubes. (3) Sir Jonathan Hutchinson[1] has described a case of what appears to be ateleiosis which showed marked hypertrophy of the gums, of unknown cause. (4) Hastings Gilford (*loc. supra cit.* pp. 358 and 359) quotes Thomson's case, which showed hypertrophy of the thymus gland. This may possibly have indicated a condition of "lymphatism"; but, whether or no, the case appears just as likely to have been one of dwarf growth, with "infantilism" secondary

[1] Hutchinson, Sir Jonathan, "A case of hypertrophy of the Gums with General Dwarfdom." *Edin. Med. Journal*, n. s. vol. I. No. 2, p. 117.

to the thymic hypertrophy, as one of ateleiosis with the former as an independent association. (5) Hastings Gilford's third new case (Martin Lane) "showed a persistent, though not patent, ductus arteriosus." (6) Bobbie Fenwick above referred to shows some abnormality of the development of the lower jaw of uncertain nature. These appear to be all the cases that have been recorded which illustrate this matter. Of these cases (1) and (4) are probably not ateleiotic and (3) is doubtful. It is, in cases like (1) and (4), difficult to exclude some secondary form of dwarfism with "infantilism."

The above cases show nothing of any special significance. Ateleiotic dwarfs appear to be subject to the same diseases and to show the same age changes and causes of death as do individuals of normal growth.

Duration of Life in Ateleiosis. Sternberg (*loc. supra cit.*) states that dwarfs die, on the average, younger than do ordinary individuals. This does not appear to be true on the average of the ateleiotic variety. Boruwlaski lived to be 98 years of age, Jeffrey Hudson to 62, others, *e.g.* the Gibsons, to over the seventh decade and one, probably ateleiotic, to 100 years (see general account). I know of three cases of 60 years or over, all in apparent health, and of three over 50 years of age in the same condition. The span of life does not appear to be really altered, on the average, by ateleiosis.

Diagnosis. Differentiation from other varieties of dwarf growth and from the various other conditions associated with "infantilism" (and usually, also, showing dwarfing of growth) has been considered in the general discussion—pp. 393 *et seq.* Diagnosis can only be made, of course, upon general principles. In typical cases it presents no difficulties. The most important indications of ateleiosis appear to be: (1) Very low stature. In the commoner group of cases, at least, the degree of dwarfing is only equalled by that of extreme grades of cretinism and infantile myxoedema. (In the less common third group it is of course not very marked.) (2) Proportions, those of childhood, normal, with the exception of the cranium, which is relatively large. (3) Absence of deformities of any kind. (4) Facial appearance child-like or infant-like, without any of the symptoms such as macro-cheilia, macro-glossia and increase of subcutaneous tissues found in cretinism and infantile myxoedema. (5) Intelligence normal. (6) No cause (such as those enumerated in the general discussion) apparent. (7) Radiographic examination of the skeleton, showing delayed union of epiphyses, their delayed appearance or their absence.

In certain cases, however, the distinction between ateleiosis and infantile myxoedema is nearly impossible to make, at least in the earlier years; for, on the one hand, cases of ateleiosis do occasionally show mental backwardness almost amounting to idiotcy, and on the other hand, in cases of infantile myxoedema mental backwardness may not amount to idiotcy and symptoms other than dwarfing of growth may be very slightly marked. Nor does the influence exerted by the administration of thyroid extract always remove the difficulty, because, as is well known, thyroid extract sometimes appears to act beneficially on other varieties of dwarf growth, such as do not appear to have any relationship to thyroid gland defect. Such

symptoms as "infantilism," "cryptorchism," etc., are shown by both conditions and even the radiographic appearances of the skeleton may be indecisive in earlier life in distinguishing the one condition from the other. From such conditions as achondroplasia, rickety dwarf growth, mongolism, etc., the diagnosis can be made at a glance; these conditions present no real resemblances to one another, except defect of growth or development as a whole.

Heredity. As is shown by the pedigrees, ateleiosis has been known to occur, fairly frequently, in several members of a family in the same generation. Schmolck's case, see Pedigrees, Fig. 689, shows the condition in two branches of such a family. With few exceptions families described with the pedigrees show the condition in only one generation. In one of these exceptions (Pedigrees, Figs. 608 and 620) an achondroplasic mother produced an ateleiotic son by an ateleiotic father. The latter did not show "infantilism," the son, however, does (see Plates DD (51) and FF (62)). In a second case, that of Levy, Pedigrees, Fig. 708, ateleiosis occurred in father and son. The grandfather was also probably ateleiotic.

The ateleiotic individual usually shows "infantilism" and is usually sterile, but this is by no means invariable. There are a fair number of cases recorded in which offspring have been borne to parents one or both of whom were ateleiotic. These, however, with the exception of the cases quoted have grown to a normal size if they survived to adult years. The history of the descendants or collaterals in the latter instance, if any, does not appear to have been followed up and recorded; so that nothing is known of that aspect of the heredity of this condition.

Dangers in Delivery. Comparing these dwarfs with achondroplasic individuals, in whom the process of child-birth is always one of vital danger, the question arises whether in them also dangers arise in delivery. This seems to be unusual, but sometimes to occur. The comparative structure of the two classes of dwarfs shows why this is so. The achondroplasic female is one who, though she possesses a trunk and cranium of practically normal dimensions, owes her lack of stature to the fact that the bones of her limbs are defective in growth. This defect is more marked in those of the segments nearer to the trunk than in those more remote; affecting most those in which ossification in cartilage begins early *in utero*. That is to say long bones in which ossification becomes relatively advanced during life, *in utero* are the most defective, and to this group the bones of the arm and thigh belong. The bones of the limb girdles, though affected to a less degree than these, are relatively defective. A pelvis which is small in comparison with the rest of the trunk and the head results. On the other hand these individuals have normal powers of reproduction. An achondroplasic female may have a child of normal proportions, or one like herself; in either case the cranium is of at least average dimensions; her pelvic inlet and outlet are, however, too small to permit of the passage of a child at anything approaching full term; hence normal labour is here impossible. The ateleiotic dwarfs on the contrary are puny individuals. Their bodily development with the exception of the cranium is defective as a whole; they have small bodies as well as small limbs, and though the pelvis is contracted, it is not

contracted proportionally to the rest of the skeleton. It maintains the relative size and shape present in the young child of normal growth. Further, their powers of nutrition are probably not great enough to maintain, nor is their abdominal capacity great enough to contain, as a rule, a child which would be full sized at full term. Their infants are therefore usually undersized at birth in due proportion to their own size. Hence as a rule no trouble in delivery occurs. It appears that in some cases, however, the child is of normal size and the head occupies the true pelvis in the later months of pregnancy. In other cases the child, full sized at birth occupied a normal position in the abdomen and delivery by Caesarian section or embryotomy has been necessitated. (See accounts of such cases with the pedigrees.)

Frequency. Ateleiosis appears to be a rather rare condition, but no exact estimate of its frequency seems possible at present. "Johann Ranke found amongst 45,000 Bavarian conscripts, who were mustered for military duty in the year 1875, 43 dwarfed individuals, or a percentage of 0·095, whose height varied from 1·40 m. (4 feet 7 inches) to 1·15 m. (3 feet 9 inches), and were for this defect rejected as unfit for service. The average height for males in Bavaria is 1·62 m. (5 feet $3\frac{3}{4}$ inches)" (A. Schmidt[1]).

It is possible that some of Ranke's cases may have been examples of ateleiosis; but if so we do not know how many; and such conditions as rickety dwarfism (so much more common), achondroplasia, etc., would probably account for the majority of cases that were due to disease. In a military muster of conscripts under compulsory service, which appears to be "no respecter of persons," even some mentally deficient cases, such as examples of cretinism, infantile myxoedema, etc., would possibly be required, unless obviously idiotic, to attend among the number for inspection, before being rejected as unfit. So that the above figures do not afford any means at all of estimating, even approximately, the frequency of ateleiosis.

In the recent exhibition of some 53 "dwarfs" at Olympia, London, October 1909 to January 1910 the ateleiotic dwarfs appear to have been in a majority: see Plate FF (60). The names and ages of these "dwarfs" are appended[2].

It was reported that 200 more "dwarfs" in various parts of Europe were known

[1] See Bibl. No. 270.

[2]

Name.	Age.	Name.	Age.	Name.	Age.
Diedrich Ulpts	49	†Angelica Dorffler	17	Karl Liebisch	18
Meyer Blaser	40	Reinhold Tschuschke ...	36	Alois Sauer	16
Sally Gabriel	27	Helene Tschuschke (Kulawy)	40	Thomas Thon Jones	16
†Albert Huebler	45	Heinrich Glauer	24	Josef Weisseneder	14
Andreas Wruck	23	Bruno Glauer	20	Louise Leynard	29
Gustav Geschke	52	Paul Hennersdorf	28	Andreas Leynard	26
†Don Ward	28	Adolph Pospiech	30	Christian Hansen	31
Otto Beskow	20	Liesbeth Botschen	15	Juvenal Dhelin	19
Otto Bottcher	21	Franz Jungling	56	Auguste Geoffroy	37
Baron Ernesto Magri	62	†Lilly Warton	25	Smaun Sing Hpoo	26
Count Primo Magri	59	Anna Mayor	32	Wilhelm Moller	56
The Countess Mercy Lavinia Magri ("Mrs Tom Thumb")	67	Anna Angerer	24	Charlotte Moller (Braker) ...	45
Annie Nelson Laible	45	Paula Angerer	22	Martha Schwarz	22
George Laible	48	Ilonka Blaschek	24	Marie Meister	16
†Dagmar Huther (Kipke) ...	43	Isabella Otocka	18	Agnes Jankowska	23
†Ludwig Merz	18	Ignar Haun	27	Wizentina Jankowska ...	49
†Elizabeth Dorffler (Kipke) ...	41	Albert Grunner	23	Karl Hesselbart	37
		Arthur Huhle	21	Johanna Freyer	25

† Signifies achondroplasia.

to the promoters of this exhibition, but here again, examples of such conditions as cretinism, etc., would probably be included. Ateleiosis is, however, probably more common than is recognised; the fact that so large a number of "exhibition" cases exists tends to confirm this. It must be remembered that a considerable proportion of cases do not join "exhibitions," and so remain little noticed. These would usually only come under the observation of medical men for complaints other than ateleiosis and so would in some instances possibly escape record as cases of ateleiosis. If these suppositions be correct the condition is probably more common than would be, at first, supposed.

The condition occurs in the lower animals, at least in the equine species (see Plate FF (61)).

BIBLIOGRAPHY OF DWARFISM.

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Bibliography. This bibliography applies in the first place to true dwarfism and achondroplasia. The references to myxoedematous dwarfs and to cretinism are only intended for the purpose of comparative citation or illustration. Cretinism and Infantilism will be treated at length in other sections of the *Treasury*. It was intended at first to divide the Bibliography into two sections, (i) True Dwarfism (Ateleiosis) and (ii) Achondroplasia, but it was found that (*a*) many of the works dealt with both groups and (*b*) in most of the earlier records of historical dwarfs it was not possible to be absolutely certain of the class to which the dwarf really belonged.

An asterisk attached to the index number denotes that the bibliographer has not been able either to discover or to consult the paper referred to.

1. Cardanus, Jacobus: *De Subtilitate*, p. 357. Basiliae, 1554. [He states that, in the previous year, a man of adult age and a cubit in height had been carried round in a parrot's cage.]

2. Thurneisser zum Thurn, Leonhart: *Pison, Das erste Theil. Von kalten, warmen, mineralischen und metallischen Wassern, sampt der Vergleichung der Plantarum und Erdgewechsen.* Buch 7. Cap. 84, S. 358—359. Franckfurt an der Oder, 1572. [He states that he was told that pygmy bones and an entire pygmy skeleton, which was only "2 Werkschuh, 3 Zoll lang," had been dug up near the town of Lubin.]

3. Guyon, L.: *Les diverses Leçons*, T. i. Chap. vi. pp. 789—795. Lyon, 1604. [Several dwarfs are mentioned, among them one in Cairo in 1559, an Abyssinian aged 68, not above "3 pieds" in height, who said his father was smaller than he "d'un demy pied."]

4. Talentoni, Giovanni: *Variarum et reconditarum rerum Thesaurus.* Liber iii. Cap. xxi. pp. 543—553. Francofurti, 1605. [This chapter is on the pygmies of the ancients. He quotes many classical writers.]

5. Purchas, S.: "The strange adventures of Andrew Battell of Leigh in Essex sent by the Portugals prisoner to Angola, who lived there and in the adjoining regions neere eighteene years." *Purchas, His Pilgrimes*, Vol. ii. Chap. iii. pp. 981—983. London, 1625. [He tells of a pygmy race called Matimbos, who lived to the North-East of Mani Kesock. He says, "they are no bigger than Boyes of twelve years old, but are very thicke and live onely upon flesh, which they kill in the Woods with their Bowes and Darts."]

6. Bartholinus, Casparus: *Opuscula Quatuor Singularia.* I. De Unicornu ejusque affinibus et succedaneis. II. De Lapide Nephritico et Amuletis praecipuis. III. De Pygmaeis. IV. Consilium de Studio Medico inchoando, continuando et absolvendo. Hafniae, 1628. [De Pygmaeis consists of eight chapters on dwarfs and on the pygmies of the classical writers. pp. 3—5 give a long list of celebrated men who were small of stature.]

7. Platerus, Felix: *Observationum in hominis affectibus plerisque, corpori et animo, functionum laesione, dolore, aliave molestia et vitiis infensis Libri Tres.* Liber iii. pp. 581—582. Basiliae, 1641. [An account of three dwarfs is given: (i) Johann d'Estrix, a native of Mechlin, who was with the Duke of Parma. He was seen in Basel in Nov. 1592, aged 35 years, height "tres pedes." He had a full beard, could speak three languages, and was ingenious and industrious. (ii) John Ducher, an English dwarf, seen in 1610. Judging from his wrinkled face and

long beard he was aged about 45. "Longus erat pedes duos cum dimidio saltem." He was well formed, with straight thick limbs. (iii) A dwarf seen at the wedding of the Duke of Bavaria 40 years previously. No height given.]

8. Aldrovandi, Ulyssis: *Monstrorum Historia*, pp. 38—40 and p. 602. Bononiae, 1642. [He gives an account of Michele Magnan, aged 41, height about 30 "uncias," whose portrait is in the museum of the Senate at Bologna (see Iconog. No. 72) and also of the two dwarfs in Pedigree No. 733. There are pictures of the two last.]

9. Glisson, Francis, Bate, George, and Regemorter, Ahasuerus: *A Treatise of the Rickets*, published in Latin in 1650 and translated by Phil. Armin, pp. 151—153. London, 1651. [Probably the first general account of the subject; pp. 151—153 are on heredity.]

10. Jornandes: *De Getarum sive Gothorum origine et rebus gestis. Historia Gotthorum, Vandalorum et Langobardorum ab Hugone Grotio*, Cap. xxxv. p. 661. Amstelodami, 1655. [Gives description of Attila. "Forma brevis, lato pectore, capite grandiori, minutis oculis, rarus barba, canis aspersus, simo naso, teter colore, originis suae signa restituens."]

11. Dobrzensky, Jac. Joh. Wenceslaus: De Artificiale Pygmaeorum Efformatione. *Miscellanea Curiosa sive Ephemeridum Medico-physicarum Germanicarum Academiae Naturae Curiosorum*, Annus Primus, Obs. lxxix. pp. 160—161. Francofurti et Lipsiae, 1670. [He states that Dr Joannes Marcus of Prague had been asked by a religious man whether the method of making pygmies was natural or an art of the Devil. For a certain poor man had anointed his offspring from the day of their birth with an ointment made from dormice, bats, and moles and this ointment had dried up their spinal cord and prevented their bones from growing. They remained small and were presented to great people, and, dwarfs being fashionable, were a means of support to their parent.]

11b. Zwinger, Theodor: Pumiliones, Parvi, Pusilli, Nani. *Theatrum Vitae Humanae*, Vol. xvii. Liber vi. p. 2576. Basiliae, 1671. [Gives a list of various small men, starting with Zacchaeus the publican.]

12. Thevenot, J.: *Relation d'un voyage fait au Levant*, 2e Partie, Chap. 68, pp. 475—476. Paris, 1674. [Gives an account of the arrival of the ambassador from Ethiopia at Cairo with presents for "le Grand Seigneur." Among them were diminutive black slaves from Nubia and other confines of Ethiopia.]

12b. Wanley, Nathaniel: "Of Pygmies and Dwarfs and men much below the common height," *The Wonders of the Little World or a General History of Man in Six Books*, Book i. Chap. 23, pp. 36, 37. London, 1678. [Refers to dwarfs mentioned by Pliny, Plater, Zwinger, etc.]

13. Dapper, Olfert: *Naukeurige Beschrijvinge der Afrikaensche Gewesten van Egypten, Barbaryen Lybien, Biledulgerid, Negroslant, Guinea, Ethiopien, Abyssinie, vertoomt in de Benamigen, Grenspalen, Revieren, Steden, Gewaffen, Dieren, Zeeden, Drachten, Talen, Rijkdommen, Godsdiensten en Heerschappyen, met Lantkaerten en Afbeeldingen van Steden, Drachten, etc. getrokken nyt verscheyde bedendaegse Lantbeschrijvers en geschriften van bereisde Ondersoekers dier Landen door Dr O. Dapper*, Second Edition, pp. 166, 216, 218. Amsterdam, 1676. [p. 166, describing the kingdom of Loango or land of the Bramas, he says that there were dwarfs "met den rugh na hem toe gekeert," who were short in person but had large heads. The negros said there was a region full of forests, where only these dwarfs lived, and that most of the elephants were killed by them. These dwarfs were generally called Bakke-Bakke (? = Akkas), but also Mimos; p. 216 again refers to the elephants being killed by the dwarfs, who the Jagos said made themselves invisible and could thus get close to the elephants; p. 218 states that the kingdom of Makoko was north of the river Zaire and 200 or 250 "mijlen" inland from the coast of Lovango or Kongo and the dwarfs lived in the forests of this kingdom.]

14. Clauderus, F. W.: Nanorum generatio. *Miscellanea Curiosa sive Ephemeridum Medico-physicarum Germanicarum Academiae Imperialis Leopoldinae Naturae Curiosorum*, Annus Octavus, Obs. ccxxii. p. 543. Norimbergae, 1689. [Pedigree No. 839.]

14b. La Motiie, Marie Catherine, Comtesse d'Aulnoy: *Relation du Voyage d'Espagne*, 2 Ed. T. iii. pp. 169, 199, 225. La Haye, 1692. [Comtesse d'Aulnoy went to Spain in 1679. She visited the Queen Mother at Toledo and there "Une petite Naine, grosse comme un tonneau et plus courte qu'un potiron, toute vetue de brocard or et argent, avec de longs cheveux qui lui descendaient presque aux pieds, entra et se vint mettre à genoux devant la Reine pour lui demander s'il lui plaisait de souper." Cf. Iconog. Nos. 64a and 64b. On p. 199 reference is made to a giantess who held two female dwarfs on her hands; these were seen during a visit paid to the Queen Mother at Madrid. p. 225 describes the King's dwarf Louisillo. "Il est né en Flandre, et d'une petitesse merveilleuse, parfaitement bien proportionné. Il a le visage beau, la tête admirable et de l'esprit plus qu'on ne peut se l'imaginer; mais un esprit sage et qui sçait

beaucoup." He had a dwarf horse as well made as its master, "Quand il est monté dessus, ils ne font plus de trois quartiers de hauteur."]

15. Tyson, E. *The Anatomy of a pigmie compared with that of a monkey, an ape, and a man.* London, 1699. [Followed by the well-known "Philological Essay concerning the Pygmies of the Ancients": see No. 294 for reprint.]

16. Banier (Abbé): Dissertation sur les Pygmées. Memoires de Litterature tirez des Registres de l'Académie des Inscriptions et Belles Lettres depuis 1718—1725, pp. 101—116. *Histoire de l'Académie des Inscriptions et Belles Lettres*, T. v. Paris, 1729. [Title describes subject.]

17. Nicephorus, Callistus: *Ecclesiasticae Historiae*, Liber xii. Cap. xxxvii. p. 307. Paris, 1730. [He states that he saw a dwarf in Egypt, "tam brevis fuit, ut perdici persimilis esset." He was about 25 years of age. His prudence was that of a well-formed man and his conversation showed superiority of mind. Garnier (Bibl. No. 205) reproduces a picture of this dwarf by Lycosthenes. Cf. Iconog. No. 126.]

18. Vallisneri, Antonio: *Opere fisico-mediche*, T. iii. p. 455. Venezia, 1733. [Vallisneri states he had spoken to a well-made dwarf, with a long beard, who was exhibiting himself. He pretended he had come from India but his accent betrayed his Parmesan origin.]

18b. Du Plessis (James Paris, servant to Samuel Pepys): *Collections of Wonderful Prodigies.* 1730—1733. *Sloane Manuscripts*, Nos. 3253 and 5246. British Museum, London. [No. 3253 gives picture and history of John Grimes. Pedigree 760. No. 5246 gives pictures and accounts of Anne Rouse, John Worrenbergh (Wormberg), the Black Prince and his Lilliputian horse and Hannah Warton. MS. 5246 contains account of collection and how it passed into Sir Hans Sloane's possession. See Iconog. Nos. 171—175.]

19. *The Spectator*, Vol. iv. 1711—1712, pp. 75—76. 12th Edition. Dublin, 1737. [There is a letter dated Jan. 10th, 1712, giving an account of two dwarfs, man and wife, the latter 2 feet high and enceinte; they had a dwarf horse and were exhibited in London. See No. 18b.]

20. Geoffroi, C. J.: *Histoire de l'Académie des Sciences avec les Mémoires de Mathématique et de Physique*, 1746, pp. 44—45. Paris, 1751. [Account of Bébé, Pedigree No. 745.]

21. Arderon, Wm.: Extract of a letter from Mr Wm. Arderon, F.R.S., to Mr Henry Baker, F.R.S., containing an account of a dwarf, together with a comparison of his dimensions with those of a child under 4 years old by David Erskine Baker. The letter is dated Norwich, May 12th, 1750. *Philosophical Transactions of the Royal Society*, Vol. xlvi. pp. 467—470. London, 1752. [An account of John Coan of Twitschall, Norfolk. Height, 38 inches with hat, wig and shoes on. Weight, 34 pounds, including clothes. See p. 363 *supra*. No family history. Measurements given.]

22. Browning, John: Extract of a letter from John Browning, Esq., of Barton Hill near Bristol, to Mr Henry Baker, F.R.S., concerning a dwarf, Sept. 12th, 1751. *Philosophical Transactions of the Royal Society*, Vol. xlvii. pp. 278—281. London, 1753. [Pedigree No. 701.]

23. Klein, L. G.: Rhachitis congenita atque hermaphroditi rumor. *Nova Acta Physico-medicae Academiae Caesareae Leopoldino-Carolinae. Naturae Curiosorum*, T. i. Obs. xxxviii. pp. 146—148. Norimbergae, 1754. [Description of an achondroplasic (?) twin. No family history.]

23b. Hay, W.: *Deformity, an Essay.* London, 1754. [p. 4, "I am scarce five feet high, my back was bent in my mother's womb, and in person I ressemble Esop, the Prince of Orange, etc.": see p. 360 *supra*.]

24. Birch, Thos.: *History of the Royal Society of London*, Vol. iv. p. 500. London, 1757. [Letter from Mons. Justel read Nov. 3rd, 1686, giving an account of a dwarf from Quimpercorantin, Lower Bretagne, aged 37, height 16 inches, who had a great beard. No family history.]

25. Morel: Diversités anatomiques. *Vandermonde's Recueil périodique d'observations de médecine, chirurgie, pharmacie, etc.*, T. vii. pp. 432—441. Paris, 1757. [Description of the skeleton of a rachitic child, still-born at full term, which Morel had in his collection. Length of skeleton from crown of head to level of heels "12 pouces."]

26. Sandel, Samuel: Account of two children of an uncommon small stature. *Medical, Chirurgical and Anatomical Cases and Experiments*, communicated by Dr Haller and other eminent physicians to the Royal Academy at Stockholm, translated from the Swedish, pp. 68—71. London, 1758. [Pedigree No. 833.]

27. Marin: *Histoire de Saladin, Sulthan d'Egypte et de Syrie*, T. i. pp. 88—89 and 158. Paris, 1758. [Note on p. 89 refers to Schirkouh; the name of this Kurd is written in different ways, Schirucouh, Schiracus, Schiragouh, Schirgouh. Historians write it Siracon. It means "lion of the mountain." Schirkouh was uncle of Saladin, who reaped the fruit of his victories. He is thus described on p. 158: "Schirkouh étoit petit de taille et fort gros, d'une figure

austère, d'un caractère dur et farouche, et d'un esprit peu éclairé." Apparently it is this Schirkouh who appears later as the dwarf Characus: see p. 359.]

28. *La Vergne de Tressan, Louis E. de: Mémoire sur un Nain. *Académie des Sciences.* Paris, 1760. [A Memoir on the dwarfs Boruwlaski and Bébé. Said to be presented to Académie, it cannot be found in the *Mémoires*: see, however, No. 41.]

29. Houtluyn, F.: *Natuurlyke Historie of uitvoerige Beschrijving der Dieren, Planten en Mineralien, volgens het Samenstel van den Heer Linnaeus. Mit naauwkeurige Afbeeldingen*, Deel I. p. 148. Amsterdam, 1761. [States the Archduke Frederick had a dwarf at his court 3 "Spannen" long and mentions Bébé and Boruwlaski. A note at foot of page says a Frieslander had been in Amsterdam, aged 21, on Mar. 2, 1751, and no higher than "negen en twintig Amsterdamsche Duimen, en biggevolg nog omtrent anderhalf Duim kleinder."]

30. Klein, J. H.: *Dissertatio inauguralis medica sistens casum rhacitidis congenitae observatae in infante varie monstroso.* Argentorati, 1763. [This is a description of the case recorded by his father, L. G. Klein. See Bibl. No. 23.]

31. Bordenave: Description d'un foetus mal conformé. *Mémoires de mathématique et de physique présentées à l'Académie des Sciences*, T. IV. pp. 545—550. Plate. Paris, 1763. [A male foetus of 7 months, length "9½ pouces" from the crown of the head to the heels, which touched the buttocks.]

32. Article: Nains. *Diderot et d'Alembert's Encyclopédie*, T. XI. pp. 7—8. Neuchatel, 1765. [Signed D. J., written in 1760 and giving an account of Bébé and Boruwlaski.]

33. Buffon, G. L. L.: *Histoire Naturelle générale et particulière*, T. XV. pp. 176—185. Paris, 1767. [Description and measurements of the skeletons of Bébé and of a rachitic girl of 10 or 11 years of age.]

34. Lettre envoyé par M. D. à Mons. Le Comte de —: Sur un nain monstrueux existant actuellement dans la Ville de Lubni. *Introduction aux Observations sur la Physique, sur l'Histoire Naturelle et sur les Arts*, T. L. pp. 295—296. Paris, 1771. [An account of the dwarf Pierre Danlow Bereschny. Height 29¾ "pouces." He had no arms, no neck, and no joints to his knees.]

35. *Mussot, Arnould François: Spectacles des Foires et des Boulevards de Paris. *Calendrier historique et chronologique des théâtres forains.* Paris, 1776. [Quoted by Garnier (Bibl. No. 205). Pedigree No. 748.]

36. Buffon, G. L. L.: Nains. *Histoire Naturelle générale et particulière*, T. IV. Supplément, pp. 400—404 and 505—512. Paris, 1777. [A few remarks on some well-known dwarfs, Bébé, Boruwlaski, the Norfolk dwarf, etc.; pp. 505—512 refer to "Les Nains de Madagascar." See p. 356 *supra.*]

37. Lavater, J. C.: *Physiognomische Fragmente zur Beförderung der Menschenkenntniss und Menschenliebe*, Theil IV. S. 72. Leipzig und Winterthur, 1778. [Short note on a picture of C. H. Stöberin, aged 16, and not more than "2 Fuss" in height. See Iconog. No. 138.]

38. Changeux, P. C.: Dissertation sur les nains et les géants, et sur les vraies limites de la taille humaine. *Observations sur la Physique, sur l'Histoire Naturelle et sur les Arts,* Supplément, T. XIII. pp. 167—171. Paris, 1778. [The chief subject of this dissertation is, the author says, "Rapports singuliers qui se trouvent entre les Nains et les Géants."]

39. Küiin, D.: Kurze Geschichte einer Zwergfamilie. *Schriften der Berlinischen Gesellschaft naturforschender Freunde*, Bd. I. S. 367—372. Berlin, 1780. [Pedigree No. 794.]

40. van Wurmb, F.: Kitip, een klein en simeetrisch wanschapen Mensch. *Verhandelingen van het Bataviaasch Genootschap der Kunsten en Weetenschappen*, Deel 3. pp. 339—344. Batavia, 1781. [An account and full measurements of Kitip, a six-fingered and six-toed achondroplasic (?) dwarf. Height "2 voeten, 11 duim, Englische maat." He was a native of Bali. His parents and several brothers and sisters were normal.]

41. Article: Sur les Nains. *Histoire de l'Académie des Sciences de Paris*, pp. 62—71, 1764. Also *Collection Acad. de Mémoires, etc.*, T. XIII. pp. 347—355. Paris, 1786. [Résumé of Bibl. No. 28 and of Morand's paper read Nov. 1764 on Bébé and Boruwlaski.]

42. Walpole, Horace: *Anecdotes of Painting in England*, 4th Edition, Vol. III. pp. 116—188 and p. 257. London, 1786. [An account of Gibson the dwarf artist is given. Pedigree No. 699.]

43. Boruwlaski, Joseph: *Memoirs of the celebrated Dwarf, Joseph Boruwlaski.* London, 1778. [Written originally in French by himself, translated by M. des Carrières. The French and English versions are intermixed. Portrait of Boruwlaski with his wife and child as frontispiece. Pedigree No. 693. See Iconog. Nos. 143 and 144.]

44. Ekman, Olaus F.: *Descriptionem et Casus aliquot Osteomalaciae sistens*, Dissertatio medica, pp. 5—10. Upsaliae, 1788. [Pedigree No. 781.]

45. Flögel, Karl Friedrich: *Geschichte der Hofnarren*, S. 500—530. Liegnitz und Leipzig, 1789. [Gives an account of the court dwarfs of Imperial Rome and mediaeval Europe.]

46. Soemmering, Samuel Thos.: *Abbildungen und Beschreibungen einiger Missgeburten*, S. 30, Tafel xi. Mainz, 1791. [Gives picture and some measurements of an achondroplasic (?) infant.]

47. Boruwlaski, J.: *Mémoires du célèbre Nain.* Birmingham, 1792. [An edition in French of the Memoirs originally published in 1788. See Bibl. No. 43, Pedigree No. 693.]

48. Guthrie, Matthew: Nains des Anciens et des Russes. *Dissertations sur les Antiquités de Russie*, trans. from the English, p. 153. St Petersburg, 1795. [p. 153 gives the history and description of Prascovia Ivanovna, dwarf of Peter the Great. Her height is not given. She looked like a child of 6 or 8 years of age. She must have been nearly 100 years old, and was still active, with full use of her eyes, legs, and teeth. She complained of no infirmity at date 15 Oct. 1794, after 80 years' sojourn in Russia. Guthrie states there were at that time a number of old dwarfs collected both in the old and new capitals of the Empire, since they had ceased to be fashionable as retainers of great houses.]

49. Ludwig, Ch. F.: *Grundriss der Naturgeschichte der Menschenspecies*, S. 154—155. Leipzig, 1796. [Gives references to various dwarfs. Pedigree No. 724.]

50. Wünsch, Ch. E.: *Unterhaltungen über den Menschen*, Theil i. 2 Ed. Zehnter Unterhaltung. S. 319 and 320. Coloured Plate. Leipzig, 1796. [Refers to Bébé, Boruwlaski and C. H. Stöberin. The plate gives Bébé and C. H. Stöberin with other figures. C. H. Stöberin was almost 3 feet in height and well proportioned: see Bibl. No. 49 and Iconog. No. 138.]

51. Osiander, F. B.: *Historia partus nanae, versionis negotio, a foetu vivo feliciter liberatae.* Plate. Gottingae, 1797. [Pedigree No. 627.]

52. Dumeril, C.: Notice sur un homme mort à l'âge de soixante-deux ans, dont les bras, les avant-bras, les cuisses et les jambes ne s'étoient pas développés. *Bulletins des Sciences, par la Société Philomatique*, N°. iv. 7e année, T. iii, avec une planche xiii. pp. 122—124. Paris, Messidor, An 11 de la République, 1800. [Gives an account of Marc Catozze, called "le petit nain." His parents, brothers and sisters were normal. The trunk and sexual organs were normal. Well-formed hands grew from his shoulders, and the lower limbs consisted of flattened buttocks which supported badly developed but complete feet. He would have had great difficulty in feeding himself but for a peculiar conformation of the lower jaw which enabled him to so protrude and lower it that he could bring it towards the food. He could ride and shoot. There is a plate of the skeleton.]

53. Kirby, R. S.: *Wonderful and Eccentric Museum, or Magazine of remarkable Characters.* London, 1804—1820. [Vol. i. p. 95: Account of the dwarf John Coan and a dwarf from Glamorganshire aged 15, height 30 in., weight 12 lbs. Vol. ii. pp. 145—150: Accounts of Simon Paap, with Plate, height 28 in., weight 27 lbs.; Miss Smith and Mr Leach. Vol. iii. p. 113: Account of George Romondo, with Plate, height 42 in., a clever mimic. pp. 406—413: Accounts of Bébé, Boruwlaski, Peter Dantlow, and Don Jozé Cordero Pereira. Vol. v. pp. 228—229: Accounts of Nannette Stocker and Johann Hauptmann, with Plate. p. 364: Account of the Irish dwarf Owen Farrel, with Plate. See our pp. 363, 370 and Plates II (69), JJ (72) and KK (73) and (74).]

54. Dupuytren: Extrait de la description d'un nain de vingt-six mois présenté à la Société le 24 Juillet 1806. *Bulletin de la Faculté de Médecine de Paris.* No. viii. pp. 146—148. Paris, 1806. [Pedigree No. 756.]

55. Caulfield, James: *Portraits, Memoirs and Characters of Remarkable Persons from the Reign of Edward the Third to the Revolution.* A new edition, completing the 12th Class of Granger's Biographical History of England, with many additional rare portraits, Vol. i. p. 8, Vol. ii. p. 128, and Vol. iii. p. 284. London, 1813. [i. p. 8, portrait of John Jarvis, 3 feet 8 inches in height, who died 1558 or 1560, aged 57. The portrait was taken from a statue carved in oak and coloured to resemble life. He was page of honour to Queen Mary. ii. p. 128, portrait of Jeffrey Hudson. iii. p. 284, portrait of John Wormberg, aged 38, height 31 inches: see Bibl. No. 18b.]

56. Obituary with Anecdotes of remarkable Persons. *The Gentleman's Magazine*, Jan. 1813, Vol. 83, p. 92. London, 1813. [Pedigree No. 799.]

57. Chaussier: Sur les fractures et les luxations observées chez des foetus encore contenus dans la matrice, et faussement attribuées à l'imagination de la mère. *Bulletin de la Faculté de Médecine de Paris*, T. iii. pp. 301—311. Paris, 1813. [Pedigree No. 826.]

58. Home, Sir Everard: *Lectures on Comparative Anatomy*, Vol. i. p. 81. London, 1814. Vol. v. pp. 191—192. London, 1828. [He mentions Boruwlaski, and the reports of a race of pygmies in Madagascar which he thinks fabulous, and gives the history of Caroline Crachami. Pedigree No. 717.]

59. Otto, Adolph Wilhelm: Eine menschliche Missgeburt, mit monströs kurzen obern und untern grossen Gliedern. Plate. *Seltene Beobachtungen zur Anatomie, Physiologie und Pathologie gehörig*, Heft 1. S. 1—9. Breslau, 1816. [Full description of a male foetus. Weight "7 Pfund und 2 Loth." Length "17 Zoll rheinl."]

60. *Dornier: *Description d'une miniature humaine.* Paris, 1817. [Description of Babet Schreier.]

61. Romberg, M.: *De Rhachitide Congenita.* Dissertatio Inauguralis. Berolini, 1817. [A general account of the disease and Pedigrees Nos. 659 and 660.]

62. Béclard: Note sur une naine de l'âge de sept ans ayant à peu près les proportions d'un enfant naissant. *Bulletin de la Faculté de Médecine de Paris*, 1816—1817, T. v. pp. 486—488. Paris, 1818. [This appears to be an account of Babet Schreier. Pedigree No. 703.]

63. Chaussier et Adelon: Article Monstre. *Dictionnaire des Sciences Médicales*, T. xxxiv. pp. 210—213. Paris, 1819. [Gives accounts of several dwarfs, including Babet Schreier. Pedigree No. 703.]

64. Virey, J. J.: Article Nains. *Dictionnaire des Sciences Médicales*, T. xxxv. pp. 145—153. Paris, 1819. Also *Histoire Naturelle du Genre Humain*, T. ii. pp. 263—272. Paris, 1824. [Pedigrees Nos. 698 and 702 (Barbe and Thérèse Souvray).]

65. Caulfield, James: *Portraits, Memoirs and Characters of Remarkable Persons from the Revolution in 1688 to the end of the reign of George II*, Vol. iii. pp. 230—232. London, 1819. [Portrait and account of Owen Farrel, the Irish dwarf, whose skeleton and portrait were said to be preserved in the Hunterian Museum, Glasgow, but the skeleton is not there now. His height was 3 ft. 9 in.]

66. Carus, C. G.: Fünfter Jahresbericht über den Fortgang des königlichen Sächs. Entbindungs- und Hebammen-Instituts zu Dresden im Jahre 1819. *Leipziger Literatur Zeitung*, 1820, No. 93, S. 737—741. Leipzig, 1820. [Pedigree No. 780.]

67. Frank, Luigi: Storia di una intiera famiglia nana esistente in Parma. *Memorie della Reale Accademia delle Scienze di Torino*, T. xxv. pp. xcvi.—xcviii. Torino, 1820. [Pedigree No. 747 (Leporati family).]

68. *Jaeger, G. F.: *Vergleichung einiger durch Fettigkeit oder colossale Bildung ausgezeichneter Kinder und einiger Zwerge.* Stuttgart, 1821.

68b. Wilson, Henry: *Wonderful Characters, comprising Memoirs and Anecdotes of the most remarkable Persons of every Age and Nation.* London, 1821—22. [Vol. i. p. 88: Notice and picture of Jeffrey Hudson standing beside Charles I. p. 216: Notice and picture of Jeffrey Dunstan, known as Sir Jeffrey Dunstan, Mayor of Garrett. He was dwarfish in size and knock-kneed, and his head was disproportioned to his body. In the picture he looks rickety. Vol. ii. p. 375: Notice and picture of Wybrand Lolkes and his wife. Vol. iii. p. 385: Notice and picture of Joseph Boruwlaski, wife and child.]

69. Meckel, J. F.: *Anatomisch-physiologische Beobachtungen und Untersuchungen*, S. 9 u. 45. Halle, 1822. [Pedigree No. 640.]

69b. Smeeton, George: *Biographia Curiosa or Memoirs of remarkable Characters of the reign of George III*, pp. 38, 75, 205, 235. London, 1822. [p. 1 gives picture and notice of Madame Teresia, the Corsican Fairy, born on the mountain of Stata Ota in 1743 and exhibited in London, 1773. Her height was 34 in., weight 26 lbs. She was exceedingly well proportioned. p. 38: Notice and picture of Wybrand Lolkes and his wife. p. 75: Notice and picture of Boruwlaski, wife and child. p. 205: Notice and picture of Jeffrey Dunstan. p. 235: Notice and picture of Simon Paap.]

70. Hone, Wm.: *The Everyday Book*, Vol. i. pp. 1171, 1190, 1194. London, 1826. [p. 1194 mentions Thos. Day, a dwarf 35 inches in height, who said he was aged 35 and who was the reputed father of a dwarf family. They were exhibited at St Bartholomew's fair. No particulars are given of the family, except that there was a boy aged 6, only 27 inches in height. p. 1171 gives a picture of a dwarf, Lydia Walpole, 2 ft. 11 inches in height; p. 1190 gives a picture of a male dwarf 3 feet in height.]

71. Sartorius, Christopher Friedrich: *Rhachitidis congenitae Observationes.* Diss. Inaug. Leipzig, 1826. [Four cases are described: (1) A male, the child of healthy parents, who died some days after birth. The length and weight were almost normal; "pondus quippe erat librarum quinque cum quadrante et longitudo sedecim pollicum," but the long bones of the extremities were fractured. (2) The skeleton of a male infant, "aequat ejus longitudo tredecim pollices ulnae lipsiensis." The long bones of the extremities appeared to have been fractured and to have united again. No family history. (3) The skeleton of a still-born

infant, unsexed. The mother, aged 36, was healthy and had had several previous confinements. The length of the skeleton is not given, its extremities were curved and deformed. (4) The skeleton of a female infant, "quindecim pollices longus," the bones of the extremities were short, thick and curved. Plates of (2), (3) and (4) are given. (2) looks achondroplasic, the other cases appear more doubtful. The rest of the dissertation is a general discussion of the subject.]

72. Roulin: Sur une naine mexicaine. *Le Globe*, T. VII. p. 790. Paris, 1829. Account of a female Mexican dwarf born in the province of Zacatecas of a mother of pure Indian race. She was aged 17. Height $27\frac{1}{2}$ "pouces," with arms, hands and feet well made. Her hips were a little large.

73. Weber, M. J.: Ueber rhachitische Foetus. *Siebold's Journal für Geburtshülfe, Frauenzimmer- und Kinderkrankheiten*, Bd. IX. S. 292—297. Frankfurt-am-Main, 1830. [A description of two achondroplasic (?) foetuses. The mothers were healthy and had several healthy children.]

74. Otto, Adolf Wilhelm: Of Vices relating to Size. *Compendium of Human and Comparative pathological Anatomy*, translated from the German with additional notes and references by John F. South, pp. 21—22. London, 1831. [p. 21, Note, mentions Joseph Hoedle of Endingen, height 30 inches, and Elizabeth Ralph of Devonshire, aged about 21, height 34 in., weight 20 pounds. It also states that among Mr Hunter's papers is a memorandum without name or date of a dwarf woman at Norwich, height 34 inches, who was delivered of a child which measured 22 inches. The woman died 4 hours after delivery. p. 22, Note, mentions Drunken Andrew, who used to sit at Blackfriars Bridge; he was of small make, except for his head.]

75. Geoffroy-Saint-Hilaire, Isidore: Des Anomalies par diminution générale de volume et spécialement les nains. *Histoire générale et particulière des anomalies de l'organisation chez l'homme et chez l'animaux*, T. I. pp. 140—165. Also pp. 220 and 253, and T. II. pp. 208—213. Paris, 1832. [A general account of dwarfs. A note on p. 164 states that M. Underwood told him there was a portrait of a female dwarf 33 inches high (who was exhibited in London, 1784) in the Hunterian Museum, London, and that she died giving birth to a child.]

76. Mansfeld: Beschreibung eines Skeletts mit angeborener Rhachitis und Bemerkungen darüber. *Gräfe und Walther's Journal der Chirurgie und Augen-Heilkunde*, Bd. 19, S. 552—565. Berlin, 1833. [Pedigree No. 770.]

77. Temple, Sir Grenville T.: *Excursions in the Mediterranean, Algiers and Tunis*, Vol. II. Chap. X. p. 180. London, 1835. [Pedigree No. 768 (Dwarf Aboo Zadek).]

78. Busch, R.: Ein Fall von Rachitis congenita. *Neue Zeitschrift für Geburtskunde*, Bd. IV. S. 110—113. Berlin, 1836. [Description of a still-born female achondroplasic (?) child. Length "$12\frac{1}{4}$ Zoll." No family history.]

79. Geoffroy-Saint-Hilaire, Isidore: Nain de Bréda, en Illyrie. *Comptes Rendus de l'Académie des Sciences*, T. III. p. 480. Paris, 1836. [An account of Mathias Gulia, a well-proportioned dwarf aged 22, whose height was not above one metre.]

80. Bancel: Rapport sur la communication des conditions d'existence d'une fille naine à Valognes. *Comptes Rendus de l'Académie des Sciences*, T. V. p. 839. Paris, 1837. [Bancel, a doctor at Valognes (Manche), sent a description of a girl aged $18\frac{1}{2}$ years, height 34 "pouces," not deformed and with no trace of rickets. Serres and Geoffroy-Saint-Hilaire considered the report untrustworthy.]

81. Naegele, Fr. Carl: *Das schräg-verengte Becken*, S. 101—102. Mainz, 1839. [Pedigree No. 726.]

81[b]. Article: Quelques nains célèbres. *Le Magasin Pittoresque*, Septième Année, pp. 332—334. Paris, 1839. [On p. 333 there are pictures of Jeffrey Hudson in a pie, of Wybrand Lolkes and his wife and of Bébé with a dog. Wybrand Lolkes has a large head and very short legs, which appear somewhat curved, but his trunk also looks very short: see Iconog. Nos. 142 and 158.]

82. Dubois, Paul: Accouchement prématuré chez une naine. *Archives générales de Médecine*, 3[e] Série, T. VII. pp. 513—514. Paris, 1840. [Pedigree No. 783.]

82[b]. Liston, Robert: *Elements of Surgery*. London, 1840. [Plate II reproduces an achondroplasic (?) skeleton, but there does not appear to be any description of it, unless the remark on congenital rickets, p. 130, applies to it.]

83. Otto, Adolf Wilhelm: *Monstrorum sexcentorum descriptio anatomica. Museum Anatomico-pathologicum Vratislaviense*, pp. 319—322. Vratislaviae, 1841. [Descriptions of achondroplasic (?) foetuses, human and other. Tafel XXII gives a picture of an achondroplasic (?) infant. No measurements.]

84. Neumann: Ueber eine Familie von Zwergen. *Wochenschrift für die gesammte Heilkunde*, No. 44, S. 705—707. Berlin, 1842. [Pedigree No. 694 (Goerke family).]

85. Schuetze, E. G.: *Symbolae ad ossium recens natorum morbos.* Dissertatio Inauguralis. Plate. Berolini, 1842. [Three cases are described: (1) A still-born female infant, "octo libras gravis et quindecim pollices longa," whose extremities were very thick and short. She was the sixth child of a scrofulous woman aged 33, three of whose children had died young and of the two survivors the elder was rachitic and delicate, and the glands of the younger were affected. A full description and measurements are given. (2) A female infant, who lived six weeks, with large head and short thick extremities. The mother, a prostitute aged 22, was of small stature, born of small parents who had had 18 children, most of whom died young. She herself had had two premature confinements, one at 5 months, the other at 7 months, a still-born male, who was abnormal in many ways. (3) A foetus preserved in spirits with very short extremities. No family history.]

86. Trevisani, Luigi: Alcuni cenni su Antonio Toselli, nano di Pensale. *Rendiconti Accademia Medico-chirurgica di Ferrara.* Seduto, 3 Settembre, 1841. *Bullettino delle scienze mediche della Società Medico-chirurgica di Bologna*, 3 Serie, T. III. pp. 60—63. Bologna, 1843. [Pedigree No. 700 (Antonio Toselli).]

87. Sonntag, Ernst Heinrich: *De rachitide congenita.* Dissertatio Inauguralis. Heidelberg, 1844. [Four cases are described: (1) Pedigree No. 820. (2) A female infant, length from vertex to perineum 8″ 6‴ with short extremities. (3) Female foetus also with short extremities. (4) Skeleton of a rachitic foetus. Then follows a general discussion of the subject.]

88. Hutton, Catherine: A memoir of the celebrated dwarf Joseph Boruwlaski. *Bentley's Miscellany*, Vol. XVII. pp. 240—249. London, 1845. [Title describes subject: see Bibl. Nos. 28, 41 and 43.]

89. Hecker: Der Zwerg Margarethe Leonhard, von Villmar. *Medicinische Jahrbücher für das Herzogthum Nassau*, Heft V. S. 48—54. Wiesbaden, 1846. [Pedigree No. 730.]

90. Article: Highland Dwarfs. *Illustrated London News*, May 30, 1846, p. 357. London, 1846. [Pedigree No. 721.]

91. *De Rensis, Felice: *L'Ateneo.* Napoli, 1846. [?Something as to dwarfs.]

92. Leisinger, J.: *Anatomische Beschreibung eines kindlichen Beckens von einem 25 Jahre alten Mädchen.* Inaug. Diss. Tübingen, 1847. [Account of the abnormally diminutive pelvis, infantile in character, of a woman 25 years old of medium (52 P.″) stature. There was no trace of pathological change or bone disease; while the pelvis was comparable with that of a child, the uterus resembled that of a new-born infant.]

93. Lawrence, Sir Wm.: *Lectures on Comparative Anatomy, Physiology, Zoology, and the Natural History of Man*, 9th edition, p. 296. London, 1848. [He states that the parents, brothers and sisters of C. H. Stöberin were dwarfs and quotes Lavater (Bibl. No. 37) and Ludwig (Bibl. No.49) as authorities. No mention of her family could however be found in Lavater.]

94. Gurlt, Ernestus: *De ossium mutationibus rhachitide effectis.* Dissertatio Inauguralis. Plate. Berolini, 1848. [This paper, which is constantly referred to, deals with rachitis in general. It is divided into two parts, (i) De degeneratione ossium rhachitica in genere, (ii) De deformatione ossium rhachitica in specie.]

95. Barnes, R.: Dwarf exhibited. *Transactions of the Pathological Society*, Vol. II. pp. 126—128. London, 1848—1849. [Mentions two dwarfs, a male dwarf who measured 28 inches when about 9 years old, and whose trunk and limbs in form and proportion were those of a child aged 2 years, and a female dwarf, aged 31, whose skeleton measured 45½ inches.]

96. Schulz, G. R. August: *Ueber Rhachitis Congenita.* Inaug. Diss. Giessen, 1849. [Four foetuses with short extremities are described. (1) A male, length 22 cm. (2) A female, length 36 cm. (3) A female, length 36 cm. (4) A female, length 29 cm. Then follows a more general discussion of the subject. No family history.]

97. Walpole, Horace: *Anecdotes of Painting.* A new edition revised with additional notes by Ralph Wornum Vol. II. p. 533. London, 1849. [This edition has portraits of Richard Gibson, the dwarf painter, and Anne Shepherd his dwarf wife, but they are only miniatures of head and bust. Copied from drawing in Royal Library, Windsor: see Iconography.]

98. Fox, Henry Richard Vassal, Lord Holland: *Foreign Reminiscences*, p. 146. London, 1850. [Note on p. 146 says, "The Duke of Altamira, Marquis of Astorga, was the least man I ever saw in society and smaller than many dwarfs exhibited for money. He was President of the Junta, and drove about with guards like a royal personage. They called him Rey Chico, a name formerly given to a King of Grenada, and it was in allusion to that name that the small club or knot of men I have mentioned gave themselves that of Junta Chica."]

99. Quetelet: Sur un nain belge. *Bulletins de l'Académie Royale des Sciences de Belgique*, T. xvii. 1e Partie, pp. 344—347. Bruxelles, 1850. [Pedigree No. 735.]

100. Depaul, J. A. H.: Foetus rachitique. *Bulletin de l'Académie de Médecine de Paris*, T. xvi. pp. 73—74. Paris, 1851. [Gives a very short account of an 8½ months child with deformed limbs. He said he would make a further communication on the subject when he had mounted the skeleton. See Bibl. No. 165.]

101. Article: The Fairy Queen. *The Illustrated London News*, May 24, 1851, p. 450. London, 1851. [Pedigree No. 720: see also Bibl. No. 138.]

102. Michaelis, Gustav Adolf: *Das enge Becken*, S. 163. Leipzig, 1851. [Pedigree No. 725.]

103. Warren, J. M.: An account of two remarkable Indian dwarfs exhibited in Boston under the name of Aztec children. *American Journal of Medical Science*, Vol. xxi. No. 5, pp. 285—293. Philadelphia and London, 1851. [Title explains subject: see Bibl. No. 113.]

104. Virchow, Rudolf: Das normale Knochenwachsthum und die rachitische Störung desselben. *Virchow's Archiv*, Bd. v. S. 409—507. Berlin, 1853. [p. 490 describes the skeleton of a foetus, (?) achondroplasic.]

105. Charton, Edouard: *Voyageurs Anciens et Modernes*, T. i. p. 160; T. ii. p. 389. Paris, 1854. [p. 160 quotes Ctesias. A curious picture on this page, called "Pygmées asiatiques d'après un dessin de l'Encyclopédie japonaise," represents five pygmies arm-in-arm, Japanese in type, with a large crane with open beak hovering over them. p. 389, quoting Marco Polo, says that in the island of "Java la petite" (Sumatra) they skin small monkeys, take off all the hair except the beard and the hair on the breast and pass them off as small men.]

106. Koenig, Franz: *Beschreibung eines kindlichen Beckens und kindlicher Geschlechtstheile von einem achtzehn Jahre alten Mädchen.* Inaug. Diss. Marburg, 1855. [This dissertation is frequently referred to in articles on dwarf growth. It gives a long description of the infantile pelvis and genital organs of the girl, who was quite incapable of child-bearing; but she was not a dwarf. Superficially she appeared well developed for her age, was over middle height and broad in proportion.]

107. Virchow, Rudolf: Zur Pathologie des Schädels und des Gehirns. *Gesammelte Abhandlungen zur wissenschaftlichen Medicin*, S. 885—1014. Frankfurt a. M. 1856. [This paper is chiefly about cretinism, with some cretin pedigrees. S. 976 gives full description of a new-born infant, 33 cm. in length with flexed thighs. Virchow calls it a cretin, but its extremities appear to be of the achondroplasic type.]

108. Lecadre, Arthur J. *Étude sur le Rachitisme congénital.* Thèse. Paris, 1856. [Pedigree No. 763.]

109. *Lagarde: *Rachitisme intra-utérin.* Thèse. Paris, 1856.

110. Schreier, F.: Die Entbindung einer Zwergin. *Monatsschrift für Geburtskunde und Frauenkrankheiten*, Bd. 8, S. 116—121. Berlin, 1856. [Pedigree No. 707.]

111. Dumenil, L.: Description du squelette d'un foetus rachitique. *Gazette des Hôpitaux*, 3e Série. Année ix. p. 396. Paris, 1857. [Description of the skeleton of a foetus born at term, length 35 cm.]

112. Sänger, W. M. H.: *Beschrijving van eene misvormde menschelijke Vrucht, benevens eenige Opmerkingen omtrent de zoogenaamde aangeboren engelsche Ziekte (Rachitis Congenita).* Inaug. Diss. Leyden, 1857. [This gives a full description of a female child with short curved limbs. Total length 14 "duim," and then follows a general discussion on such deformities. There is a good plate of the child.]

113. Mayer, F. J. C.: Ueber die Azteken Liliputaner. *Verhandlungen d. naturh. Verein. d. preuss. Rheinl. u. Westphal.*, Bd. xiv. S. lxvi.—lxx. Bonn, 1857. [A description of the two Aztec dwarfs, said to be the offspring of an Indian man and negro woman: see also Bibl. No. 103.]

114. Virchow, Rudolf: Knochenwachstum und Schädelformen mit besonderer Rücksicht auf Cretinismus. *Virchow's Archiv*, Bd. xiii. S. 323—357. Berlin, 1858. [On p. 389 there are some remarks on the bones of dwarfs. p. 353 gives a description of a new-born child, (?) achondroplasic. Virchow considers it a cretin: see Bibl. No. 107.]

115. Brown, J. The Black Dwarf's Bones. *Horae subsecivae*, pp. 413—436. Edinburgh, 1858. Reprint, *The World's Classics*, pp. 247—262. London, New York, and Toronto, 1907. [Gives two letters written by Robert Craig, a surgeon, giving an account of David Ritchie, the original of the Black Dwarf. He was 4 ft. in height, his shoulders rather high, but his body otherwise the size of ordinary men. His legs were bent in every direction, but the principal turn they took was from the knee outwards, so that he rested on his inner ankles and the lower part of his

tibias. Brown possessed the femur and tibia and says, "They seem to have been blighted and rickety, the thigh-bone is very short and slight and singularly loose in texture, the leg-bone is dwarfed but dense and stout."]

116. Humphry, Sir George Murray: *A Treatise on the Human Skeleton*, pp. 100—102. Cambridge, 1858. [A few remarks on the proportions of dwarfs.]

116b. *Veratti, Giuseppe: Mostruosità e perfezione. *Il Raccoglitore Medico*, Ser. 2e, T. xvii. p. 474. Fano, 1858. [An early account of the Magri family is given in this paper.]

117. Riedel: Verhandlungen der Gesellschaft für Geburtshülfe in Berlin. Sitzung am 9 Nov. 1858. *Monatsschrift für Geburtskunde und Frauenkrankheiten*, Bd. xiii. S. 11. Berlin, 1859. [Riedel exhibited a deformed foetus, with large head, normal trunk and abdomen, and extraordinarily short extremities.]

118. *Lafont-Marron, H.: *Du rachitisme intra-utérin.* Thèse. Paris, 1859.

118b. Morley, Henry: *Memoirs of Bartholomew Fair.* London, 1859. [There are references to various dwarfs scattered through pp. 321—331. p. 321 gives Pedigree No. 718. p. 460 gives a picture of Keham Whitelamb born at Wisbech. Age 22. Height (?). He is represented standing at the door of a covered case in which he used to be carried about; type of dwarfism doubtful. p. 462 has a notice of Thomas Allen and Lady Morgan. p. 476 gives picture and autograph of Simon Paap: see Pedigree No. 806.]

119. Article: Indian Dwarf. *The Illustrated London News*, May 12, 1860, p. 453. London, 1860. [Picture and account of Mohammed Baux, born in Benares 1839. Height about 37 inches. His parents, brothers, and sisters were all rather above middle height.]

120. Müller, H.: Ueber die sogenannte foetale Rachitis als eigenthümliche Abweichung der Skeletbildung und über ihre Beziehungen zu dem Cretinismus bei Thieren, sowie zu der Bildung von Varietäten. *Würzburger medicinische Zeitschrift*, Bd. i. S. 221—276. Würzburg, 1860. [Chiefly a discussion on abnormal calves, with some reference to human foetuses: see p. 386 *supra*.]

121. Levy: Beretning om Forløsningen af en Dverg med Udmaalinger af Skeletett og Bækenet. *Bibliothek for Laeger*, Femte Raekke, Første Bind, Juli—Oct. 1860, S. 304—316. Kjøbenhavn, 1860. [Pedigree No. 708.]

122. Hink, Wilhelm: Zur paediatrischen Casuistik exquisite Rhachitis congenita, Craniotabes und rhachitische Missbildung der Röhrenknochen der obern und unteren Extremitäten. *Zeitschrift der kaiserlichen königlichen Gesellschaft der Aerzte zu Wien*, S. 107—108. Wien, 1860. [Description of a female infant with short and deformed extremities, the first child of a healthy mother, aged 19. Some measurements are given.]

123. Braun, C.: *Wochenblatt der Zeitschrift der kaiserlichen königlichen Gesellschaft der Aerzte zu Wien*, Jahrgang xvii. S. 223. Wien, 1861. [Prof. Carl Braun showed the body of a new-born child with peculiar S curvature of the four extremities, an anomaly hitherto known as Rachitis congenita.]

123b. His, W.: Zur Casuistik des Cretinismus. *Virchow's Archiv*, Bd. xxii. S. 104—110. Berlin, 1861. [A long description of the corpse of a cretinous male individual aged 58. Constantly referred to as "His's Dwarf." A few scanty thin hairs (similar to those on an elderly woman) were on the chin and upper lip. Total length from vertex to sole 120 cm. Body and limbs were very small, neck extremely short, head very broad. The nose was very broad. The swollen eyes and lip and projecting tongue which are customary in cretins did not exist and there was no goitre. The mouth and teeth showed a peculiar admixture of senile and infantile characteristics. External genitals normally developed. Very full measurements of skull and skeleton are given. There is a plate of the skull.]

124. Sedgwick, Wm.: On sexual limitation in hereditary disease. *British and Foreign Medico-chirurgical Review*, Vol. xxviii. p. 200. London, 1861. [This is merely a note referring to the case of Clauders: see Bibl. No. 14.]

125. Canton, E.: The skull, pelvis and long bones of the extremities of an aged female dwarf. *Transactions of the Pathological Society*, Vol. xii. pp. 173—176. London, 1861. [This dwarf was brought to Charing Cross Hospital. All the different parts were dwarfish but proportionate to one another, with the exception of the head, in which the dimensions of a fully and well-formed cranium obtained. No family history. Hymen intact. Height not given. Humerus, 7½ inches. Femur, 10 inches.]

126. Ecker, Alexander: Vergleichung der Körper-Proportionen zweier Personen von ungewöhnlicher Körpergrosse. Plate. *Berichte über die Verhandlungen der Gesellschaft für Beförderung der Naturwissenschaften zu Freiburg i. B.*, Bd. ii. S. 379—386. Freiburg i. B. 1861. [Full

measurements are given of the dwarf Jacob Fischer, 1·005 m. in height, aged 19¼ years, and of the giant Gottfried Sproll, aged 28, height 2·01 m.]

127. Förster, August: Die Zwergbildung, Mikrosomia, Nanosomia, S. 61—63. Verkrümmung und abnorme Kleinheit aller Extremitäten, S. 64. *Die Missbildungen.* Jena, 1861. [This gives a short general discussion on dwarfs and mentions Bébé, Boruwlaski, and Jeffrey Hudson.]

127b. Valenta, Alois: Weitere Beiträge zur Catheterisatio literi. Künstliche Frühgeburt wegen Zwergwuchses. *Wiener Medizinal Halle*, Bd. II. No. 48, S. 453—454. Wien, 1861. [A woman aged 27, scarcely "51 Wiener Zoll" in height, with limbs perfectly proportioned to her size, healthy and showing no trace of rickets, came to be confined. She had had a previous pregnancy, with premature birth, the child being very small. Premature confinement was brought on and a boy born, weight "4 Pfund," length 17″ 1‴. Mother and son left the hospital in 3 months strong and healthy.]

128. Humphry, Sir George Murray: On the Influence of Paralysis, Disease of the Joints, Disease of Epiphysial Lines, Excision of the Knee, Rickets and some other morbid conditions upon the growth of the Bones. *Medico-Chirurgical Transactions*, Vol. XLV. or N.S. Vol. XXVII. pp. 283—327. London, 1862. [pp. 318—325 give a table of the measurements of several rickety skeletons; the first five he states are cases of dwarfed growth rather than rickets proper.]

129. Welcker, Hermann: *Untersuchungen über Wachsthum und Bau des menschlichen Schädels*, Erster Theil, S. 33. Leipzig, 1862. [Some measurements of three dwarfs are given, (1) Pauline, neighbourhood of Berlin, age 17, stature 933 mm. "Kopfhöhe" defined thus, "vom Scheitel bis zu einer das Kinn betreffenden Horizontalen," 183 mm. (2) Jacob Fischer, Rhenish Palatinate, age 20, stature 1005 mm. "Kopfhöhe" 175 mm. (3) Admiral Piccolomini, Breslau, age 30, stature 1030 mm. "Kopfhöhe" 187 mm.]

130. *Castelain: Nain difforme. *Bulletin médical du Nord*, 2e Série, T. IV. p. 97. Lille, 1863.

131. Hecker, C.: Hochgrädige Beckenverengerung in Folge von Rachitis Congenita complicirt mit Eclampsie und Zwillingen. Kaiserschnitt. Beide Kinder lebend. Mutter todt 40 Stunden nach der Operation. Plate. *Hecker und Buhl's Klinik der Geburtskunde*, Bd. 2, S. 73—92. Leipzig, 1864. [Pedigree No. 823.]

132. Swayne, J. G.: Case of Caesarian Operation. Plate. *Transactions of the Obstetrical Society*, Vol. V. pp. 84—93. London, 1864. [A female achondroplasic (?) dwarf, age 42, height 4 ft. and ½ inch, came to be confined at full term. Caesarian section was performed, the mother died, the child, a male of average size, lived. She had previously had a miscarriage in the 2nd month.]

133. Ender: Kaiserschnitt mit glücklichem Ausgange für Mutter und Kind. *Monatsschrift für Geburtskunde und Frauenkrankheiten*, Bd. XXV. S. 43—50. Berlin, 1865. [Pedigree No. 815.]

133b. Verardini, Ferdinando: Rapporto dell' esame fatto dalla commissione composto dei Soc. Rizzoli, Versari, Brugnoli, Romei, Modonini e Verardini stesso, sui tre fratelli Magri di Pieve di Cento. *Bullettino delle Scienze Mediche della Società Medico-Chirurgica di Bologna*, Serie 4e, Vol. 24, pp. 270—273. Bologna, 1865. [Gives an account with measurements of the three dwarfs in the Magri family and states that Veratti's paper gives other details: see Bibl. No. 116b.]

134. Bidder, Ernst: Eine Osteogenesis imperfecta. Beitrag zur Lehre von den fötalen Knochenerkrankungen. *Monatsschrift für Geburtskunde und Frauenkrankheiten*, Bd. XXVIII. S. 136—153. Berlin, 1866. [Description of a female infant in the Würzburg pathological anatomical Collection with extremely short extremities. Total length of skeleton 27 cm.]

135. Scharlau: Congenitale Rachitis. *Monatsschrift für Geburtskunde und Frauenkrankheiten*, Bd. XXX. S. 63, u. S. 400—414. Berlin, 1867. [Pedigrees Nos. 628 and 646.]

136. Schaaffhausen, H.: Die Sektion eines in Coblenz gestorbenen Zwerges von 61 Jahren. Sitzungsberichte der nieder-rheinischen Gesellschaft für Natur- und Heilkunde zu Bonn, S. 26—27. *Verhandlungen des natur-historischen Vereines der preussischen Rheinlande und Westphalens*, 25 Jahrgang. Bonn, 1868. Also S. 10—11, 39 Jahrgang. Bonn, 1882. [Pedigree No. 705 (Family Lehnen[1]).]

137. Zagorski, Adam. Beiträge zur Kaiserschnitte. *Monatsschrift für Geburtskunde und Frauenkrankheiten*, Bd. XXXI. S. 44—67. Berlin, 1868. [Pedigree No. 723.]

138. Wood, Edward J.: *Giants and Dwarfs*, pp. 236—446. London, 1868. [Short accounts of many dwarfs. Pedigrees Nos. 710, 712 and 716 (Calvin Philips, Robert Skinner, "The Dwarf of the World").]

139. Humphry, Sir George Murray: On the skeleton of a rickety dwarf. *Journal of Anatomy and Physiology*, Vol. II. pp. 42—46. Cambridge and London, 1868. [Description and plate of the skeleton of a female dwarf in Cambridge Anatomical Museum. Said to be the skeleton of

[1] Note the occurrence of the same name as that of the painter Jacob Lehnen: see p. 359 and Iconog. No. 85.

a Paris beggar who died aged 85. Height 32·5 inches. Judging from the plate, it is a case of true rickets and not achondroplasia or ateleiosis.]

140. Down, Langdon: Case of arrested development. *Transactions of the Pathological Society*, Vol. xx. pp. 419—420. London, 1869. [Pedigree No. 835.]

141. von Franqué, Otto: Fünf Fälle von Einleitung der künstlichen Frühgeburt. *Scanzoni's Beiträge zur Geburtskunde und Gynaekologie*, Bd. vi. S. 109—124. Würzburg, 1869. [Pedigree No. 757.]

142. *Roop, F. A.: Lack of Osseous Development. *Philadelphia Medical and Surgical Reporter*, Oct. 16, 1869. Philadelphia, 1869. [An account is given of a female dwarf aged 21, whose height was 33 inches at age of 12 or 15, breasts small but had menstruated regularly for 3 years. There was no dwarfism in the family, but several of her maternal relatives had hare-lip. Original not seen but quoted from *Virchow und Hirsch's Jahresbericht*, Jahrg. iv. Bd. i. S. 178. Berlin, 1870.]

143. Winkler, N. F.: Ein Fall von foetaler Rachitis mit Mikromelie. *Archiv für Gynaekologie*, Bd. ii. S. 101—110. Berlin, 1871. [Pedigree No. 653.]

143b. *Engel: Das rachitische Becken. *Wiener medizinische Wochenschrift*, Jahrgang 1872, S. 40. Wien, 1872.

144. Urtel, H.: *Ueber Rachitis Congenita.* Inaug. Diss. Halle, 1873. [Discusses various cases and gives a description of a case of his own, a foetus with short extremities, length, 44 cm.]

145. Kehrer, F. A.: Zur Entwickelungsgeschichte des rachitischen Becken. *Archiv für Gynaekologie*, Bd. v. S. 55—59. Berlin, 1873. [Pedigree No. 772.]

146. Adams, W.: Foetus with arrested development. *Transactions of the Pathological Society*, Vol. 24, pp. 263—264. London, 1873. [Pedigree No. 818.]

147. *Klebs, E.: Beobachtungen und Versuche über Cretinismus, i. und ii. *Archiv für experimentelle Pathologie und Pharmakologie*, Bd. ii. S. 72. Leipzig, 1874.

148. Schweinfurth, Georg: *Im Herzen von Africa*, Theil ii. Kap. 16, S. 131—135. Leipzig and London, 1874. [Gives an account of the Akkas, a pygmy race in Africa: see p. 356 *supra.*]

149. Biskamp, Albert: *Ein Fall von fötaler Rachitis.* Inaug. Diss. Marburg, 1874. [This dissertation discusses cases described by other authors, more particularly Urtel's Case, and describes a new case, a male infant who died during birth. He was the first-born child of a healthy mother aged 42, weighed 2515 grammes and measured 42 cm. He had short thick extremities. A plate of the child is given.]

150. Telke, Oscar: *Experimentelle Beiträge zur Lehre von Knochenwachsthum.* Inaug. Diss. Greifswald, 1874. [Gives an account of his experiments on rabbits made for the purpose of studying the growth of bones.]

151. Englisch, Josef: Ein Fall von Rachitis foetalis. *Osterreichisches Jahrbuch für Paediatrik*, Jahrgang v. Bd. i. S. 165—176. Wien, 1874. [Description and measurements of a still-born child from a healthy mother, whose parents and siblings were healthy. The extremities were very much curved and short, probably a case of achondroplasia.]

152. Brodowski, P.: Zwei Zwerge. Sitzung der Warsch. ärztlichen Gesellschaft. *Medycyna*, No. 42, quoted in *Virchow und Hirsch's Jahresbericht über die Leistungen und Fortschritte in der Gesammten Medicin*, Jahrgang ix. Bd. i. p. 300. Bericht für das Jahr 1874. Berlin, 1875. [Pedigree No. 822.]

153. Fischer, A.: Ueber einen Fall von Rachitis Congenita. *Archiv für Gynaekologie*, Bd. vii. S. 46—48. Berlin, 1875. [Pedigree No. 809.]

154. Gräfe, Rudolph: Zwei fötal-rachitische Becken. *Archiv für Gynaekologie*, Bd. viii. S. 500—513. Berlin, 1875. [Description of two achondroplasic (?) foetuses, one male and one female, with measurements of head and pelvis. No family history.]

155. Eppinger, Hans: Mittheilungen aus dem pathologisch-anatomischen Institut zu Prag. *Vierteljahrsschrift für die praktische Heilkunde*, Bd. 126, S. 35—38. Prag u. Leipzig, 1875. [Merely a description of Klebs' Case, a female infant with short extremities, length 43·5 cm. See Bibl. No. 147.]

156. Krauss, F.: *Beschreibung des Skelets einer 29 jährigen Zwergin, mit besonderer Berücksichtigung des Beckens.* Inaug. Diss. Freiburg i. B. 1875. [Full measurements of the skeleton of a well-proportioned female dwarf are given. Height 120 cm. Caesarian section was performed and a male infant extracted. Weight 7 "Pfund," length 53 cm. The mother died; it is not stated whether the child lived or not. No family history.]

157. Moreno, Fernandez: Observación curiosa. *El Siglo Medico*, T. xxii. pp. 157—158, 173—174, 204—205. Madrid, 1875. [Pedigree No. 729.]

158. *De Antelo, José: Duos Enanos. *Revista de Antropologia*, 1875, T. II. Madrid, 1875 or 1876. [This is quoted by Taruffi, Bibl. No. 248, and from his description it must be the same case as that described by Moreno, Bibl. No. 157.]

159. Lancereaux, E.: Nanisme et Géantisme. *Traité d'Anatomie pathologique*, pp. 179—180. Paris, 1875—1877. [A short article on dwarfism, containing nothing of special value.]

160. Hoess, Franz: *Ueber Rachitis foetalis.* Inaug. Diss. Marburg, 1876. [A discussion on several well-known cases—Scharlau's, Gurlt's, Winkler's, etc., with description of a new case, a female infant, the first-born child of healthy parents. The heart beat for 10 minutes after birth. Weight 2060 grammes, length 39·5 cm. Other measurements are given. The mother was aged 23.]

161. Parrot, J.: Les lésions osseuses de la syphilis héréditaire et du rachitis. *Archives de Physiologie*, T. VIII. pp. 133—139. Paris, 1876. [On the relations of syphilis to rachitis.]

162. Charpentier: Rétrécissement du bassin chez une femme d'une taille 1·15 m. etc. *Archives de Tocologie des maladies des femmes et des enfants nouveaux-nés*, 3e Année, pp. 45—54. Paris, 1876. [Pedigree No. 647.]

163. Löhlein, Hermann: Zur Lehre vom durchweg zu engen Becken. *Zeitschrift für Geburtshülfe und Frauenkrankheiten*, Bd. I. S. 37—65. Stuttgart, 1876. [Pedigree No. 741.]

164. De Rochas, V.: Article: Nanisme. *Dictionnaire des Sciences Médicales*, 2e Série, T. II. pp. 586—591. Paris, 1876. [On dwarfs in general.]

164b. *Zosi, Ippolito: *Lettera inedita in risposta al Prof. Taruffi*, 4 Novembre, 1877 (? published).

165. Depaul, J. A. H.: Sur une maladie spéciale du système osseux, développée pendant la vie intra-utérine, et qui est généralement décrite, à tort selon moi, sous le nom de rachitisme. *Archives de Tocologie des maladies des femmes et des enfants nouveaux-nés*, 4e Année, pp. 641—650. Paris, 1877. Also 5e Année, pp. 1—8, 321—332, 424—431, 449—457. Paris, 1878. [Pedigree No. 795: see also Bibl. No. 100.]

166. Townsend, E. R.: Case of difficult labour in a dwarf, complicated with convulsions, delivered by the cephalotribe. *The Dublin Journal of Medical Science*, Vol. 64, pp. 90—92. Dublin, 1877. [Pedigree No. 764.]

167. Bornträger, J. B.: *Ueber fötale Rachitis; im Anschluss an einen Fall aus der Königsberger geburtshilflichen Klinik.* Inaug. Diss. Königsberg, 1877. [A general discussion of foetal rachitis with special reference to small pelves and to the position of rachitic children in the womb. Description of a female infant, length 34 cm., with small head and short thick extremities, is given. The body was covered with lanugo. She was the second child of a Polish woman.]

168. *Taruffi, Cesare: Della Microsomia. Nota 5. *Rivista Clinica di Bologna*, 2 s. T. 8. 122 pages. Bologna, 1878. [This paper gives the pedigree of the Magri family, then first described by Taruffi. Pedigree No. 690: see Bibl. Nos. 116b and 133b.]

169. Broca, P. P.: Nain Rachitique. *Bulletins de la Société d'Anthropologie de Paris*, 2e Série, T. XII. Année 1877, p. 448. Paris, 1878. [Broca exhibited a rachitic dwarf, aged 63. The size of his head was above the average. His extremities were short but not curved. Height 114 cm. He was intelligent and enjoyed robust health.]

170. Article: The Marriage of Dwarfs. *Hospital Gazette*, Vol. IV. p. 81. New York, 1878.

171. Müller, Adolf: Rachitic fötus. *Aerztliches Intelligenzblatt*, Jahrgang 25, S. 309. München, 1878. [Description of a still-born rachitic infant, with very short extremities. It was the second child of its mother, the first suffered from ordinary rachitis. No measurements.]

172. Parrot, J.: Sur les malformations achondroplasiques et le Dieu Phtah. *Bulletins de la Société d'Anthropologie de Paris*, 3e Série, T. I. pp. 296—302. Paris, 1878. See also *Recueil de Travaux relatifs à la Philol. et l'Archéol. Egyptiennes et Assyriennes*, Année II. pp. 129—130. Paris, 1880. [Measurements and description of a female achondroplasic dwarf aged 7½, height 93 cm., are given on p. 299.]

173. Eberth, C. J.: *Die foetale Rachitis und ihre Beziehungen zu dem Kretinismus.* Leipzig, 1878. Also *Correspondenz-Blatt für Schweizer Aerzte*, Jahrgang IX. S. 465—466. Basel, 1879. [He divides the subject into four parts. I. Fötale Rachitis bei Menschen. II. Fötale Rachitis bei Thieren. III. Rachitis und fötale Rachitis. IV. Rachitis und Kretinismus.]

174. Rawdon, H. G.: Case of dwarfed growth associated with idiocy and congenital tonic contraction of the spine and limbs. *British Medical Journal*, 1879, Vol. I. p. 386. London, 1879. [Boy, aged 8½, height 28—29 inches; he was a twin; the other twin, parents and other siblings were normal.]

175. *Kassowitz, M.: Die normale Ossification und die Erkrankungen des Knochensystems bei Rachitis und hereditären Syphilis. *Wiener Medicinische Jahrbücher*, S. 145—224 and 293—457, Tafel VIII. XVI. 8. Wien, 1879. [See Bibl. No. 217.]

176. *Kassowitz, M.: Knochenbildung und Knochenresorption. *Wiener Medicinische Blätter*, II. Jahrgang, Nos. 44, 45, 46, 47. Wien, 1879.

177. *Plà, E. F.: Caso de enanismo observando en la raza negra. *Bol. Soc. Antrop. de Cuba*, T. I. pp. 88—91. Habana, 1879—1886.

178. Kingsley, Norman W.: *A Treatise on Oral Deformities*, pp. 18—19. New York, 1880. [Some remarks on the teeth of dwarfs.]

179. Smith, Mary A.: Ueber Rachitis foetalis. 2 Tafeln. *Jahrbuch für Kinderheilkunde*, Bd. XV. S. 79—122. Leipzig, 1880. [Gives some account of the literature on the subject and a case of her own. Pedigree No. 805.]

180. *Smith, Mary A.: *Beiträge zur Lehre der foetalen Rachitis.* Inaug. Diss. Zürich, 1880.

181. Parrot, J.: La syphilis héréditaire et le rachitisme. *Le Progrès Médical*, T. VIII. pp. 623—625, 679—680, 759—762. Paris, 1880. [Chiefly about syphilis, no special cases of rachitis.]

182. Waltiher, Hugo: *Beschreibung eines durchweg zu kleinen weiblichen Skeletes mit besonderer Berücksichtigung des allgemein gleichmässig verengten Beckens.* Inaug. Diss. Freiburg i. B. 1880. [This paper gives some account of the literature on narrow pelves and "pelvis nana," and gives full description and measurements of a well-proportioned female dwarf aged 30, height 128 cm., on whom Caesarian section was performed. The child, a male, weight $4\frac{7}{8}$ "Pfund," lived, the mother died. No family history.]

183. *Guérin, J.: *Recherches sur les difformités congénitales, chez les monstres, le foetus et l'enfant.* Paris, 1880.

184. Topinard, Paul: Géants et Nain à Londres. *Revue d'Anthropologie*, T. III. p. 570. Paris, 1880. [Refers to a notice in the London *Times* (no date given) of the dwarf Chemach, aged 42, height 63 cm. He appears to have been a Chinese.]

185. Larrey: Notes sur un cas de nanisme. *Bulletins de l'Académie de Médecine*, 2e Série, T. X. pp. 1216—1218. Paris, 1881. [This is the same case as Magitot's: see Bibl. No. 186.]

186. Magitot, E.: Étude anthropologique sur un nouveau cas de nanisme. *Gazette hebdomadaire de Médecine et de Chirurgie*, 2e Série, T. XVIII. No. 43, pp. 692—694. Paris, 1881. [Pedigree No. 722.]

187. De Quatrefages de Breau, J. L. A.: Sur Balthazar Zimmermann, dit le prince Balthazar, véritable nain microcéphale. *Bulletins de la Société d'Anthropologie de Paris*, 3e Série, T. IV. pp. 702—708. Paris, 1881. [See Bibl. No. 232, Pedigree No. 765 and Plate JJ (71a).]

188. *De Quatrefages de Breau, J. L. A.: *Pygmées anciens et la science moderne.* Paris, 1881—1882.

189. De Quatrefages de Breau, J. L. A.: Les Pygmées d'Homère, d'Aristote, de Pline d'après les découvertes modernes. *Journal des Savants*, Année 1881, pp. 94—107. Paris, 1881. [This is only "Premier Article," "Les Pygmées d'Homère": he states he will continue it later. Title explains subject. ? Same as No. 188.]

190. Borelli, Diodato: Ueber unvollkommene Entwickelung der Geschlechtsorgane. *Verhandlungen der physikalisch-medicinischen Gesellschaft zu Würzburg*, N. F. Bd. XV. S. 84—92. Würzburg, 1881. [Treats of the effect of malaria on growth and gives the measurements of three boys, two of whom were brothers.]

191. Neumann, Georg: *Ueber fötale Rachitis und ihre Beziehungen zum Cretinismus.* Inaug. Diss. Halle, 1881. [He describes Virchow's Case (*Virchow's Archiv*, Bd. V., 1853) fully and discusses H. Müller's article (Bibl. No. 120) and cases of other writers. Two foetuses examined by himself are described, (1) a female infant prematurely born with short limbs, (2) a male foetus, length 30·5 cm., which he says closely resembled Case (1). No family history.]

192. Barlow, Sir Thos.: A case of so-called foetal Rickets. *Transactions of the Pathological Society*, Vol. XXXII. pp. 364—367. London, 1881. [In this case the limbs were stunted and the neck short and thick. No family history except that a previous child had been born with turned-in feet.]

193. Shattock, Samuel G.: Some Cases of osseous lesions in the Foetus. *Transactions of the Pathological Society*, Vol. XXXII. pp. 369—379. London, 1881. [Two cases, one a female, one unsexed, of which full measurements are given. No family histories.]

194. Spiegelberg, O.: Rachitis, S. 332—334. Zwergbecken, S. 409. *Lehrbuch der Geburtshülfe.* Lahr, 1882. [A general account of rachitic and dwarf pelves.]

195. Schaaffhausen, H. 1882. See No. 136.

196. Virchow, Rudolf: Zwergenkind. Verhandlungen der Berliner Gesellschaft für Anthropologie, Ethnologie, und Urgeschichte. *Zeitschrift für Ethnologie*, Bd. 14, S. 215. Berlin, 1882. [Account of Princess Paulina. Pedigree No. 728: see Bibl. Nos. 129, 214, 215 and 322.]

197. Rumpe, R.: *Ueber foetale Rachitis.* Inaug. Diss. Marburg, 1882. [A discussion of various cases described by other writers, with a description and plate of a still-born male infant, weight 3000 grammes, length 38 cm., with short extremities. He was the third child of a 25-year-old mother.]

198. Bertillon, Jacques: Un Nain Russe. *La Nature*, T. xxii. p. 13. Paris, 1883—1884. [A Russian dwarf, Wassilievitch by name, aged 51, height 1 metre. He was first described by Benzenger (it does not give reference) who presented the photograph which is reproduced, to the Société d'Anthropologie de Paris. He was the youngest of a family of six siblings, all of normal build and healthy. His mother nearly killed him in her efforts to make him grow and keep him warm. He had a wrinkled, beardless face, was quite intelligent although he had had little or no education. He was unmarried. M. Lunier thought his dwarfism might be the result of cretinism, but he had no goitre. The picture represents him standing beside an adult male and apparently he was not achondroplasic: see our Plate KK (76).]

199. Virchow, Rudolf: Fötale Rachitis, Cretinismus, und Zwergwuchs. *Virchow's Archiv*, Bd. xciv. S. 183—184. Berlin, 1883. [A few notes on the different symptoms of rachitis, cretinism, etc.]

200. Virchow, Rudolf: Amerikanischer Zwerg. Verhandlungen der Berliner Gesellschaft für Anthropologie, Ethnologie und Urgeschichte. *Zeitschrift für Ethnologie*, Bd. xv. S. 300. Berlin, 1883. [Account of the dwarf Francis G. Flynn, aged 19, height 80·7 cm. Pedigree No. 740.]

201. Lange: Entbindung einer rachitischen Zwergin. *Berliner klinische Wochenschrift*, 1883, No. 49, xx. Jahrgang, S. 753—755. Berlin, 1883. [Pedigree No. 648.]

202. Guéniot: Rachitisme congénital développé et guéri avant la naissance. *Bulletins et Mémoires de la Société de Chirurgie*, T. ix. pp. 553—556. Paris, 1883. [Description of a new-born infant. No family history or measurements.]

203. Bode, Emil: Ueber sogeuannte fötale Rachitis. *Virchow's Archiv*, Bd. xciii. S. 421—442. Berlin, 1883. [Pedigree No. 769.]

204. *Passerini, A.: Un caso di microsomia con normale sviluppo delle facultà psichiche. *Indipendente*, T. xxxiv. pp. 481—484. Torino, 1883.

205. Garnier, Edouard: *Les Nains et les Géants.* Paris, 1884. [Gives accounts of many historical dwarfs. Pedigree No. 719 (Lucia Zazate).]

206. Fürst, Livius: Exquisite Wachsthumshemmung bei Hydrocephalus Chronicus. *Virchow's Archiv*, Bd. xcvi. S. 363—365. Berlin, 1884. [A female hydrocephalic dwarf aged 13¾ years, height 81 cm. No family history.]

207. Bollinger, Otto: Ueber Zwerg- und Riesenwuchs. *Virchow's und Holtzendorff's Sammlung gemeinverständlicher wissenschaftlicher Vorträge*, Serie xix. Heft 455, S. 1—20. Berlin, 1884. [General observations on dwarf growth.]

208. Bowlby, A. A.: Four cases of sporadic cretinism, with remarks on some points of the pathology of the disease. *Transactions of the Pathological Society*, Vol. xxxv. pp. 450—464. London, 1884. [A description of four foetuses which are in the Museum of St Bartholomew's Hospital, and were classed as cases of foetal rickets; Bowlby considers them to be cases of sporadic cretinism. All four specimens have very short extremities.]

209. Taruffi, Cesare: Storia di un caso di pseudo-rachite fetale. *Memorie della R. Accademia delle Scienze dell' Istituto di Bologna*, Serie iv. T. vi. pp. 661—676. Bologna, 1884. [Pedigree No. 750.]

210. Barlow, Sir Thos.: Limb-bones, skull and brain of a case of so-called foetal rickets (? foetal cretinism). *Transactions of the Pathological Society*, Vol. xxxv. pp. 459—464. London, 1884. [Pedigree No. 807.]

211. Sutton, J. B.: A foetal Cretin. *Transactions of the Pathological Society*, Vol. xxxv. pp. 464—465. London, 1884. [A female foetus, with short limbs. Height 20 inches, length of legs 4½ inches.]

212. Grundler, R.: Zur Cachexia Strumipriva. *von Bruns' Mittheilungen aus der chirurgischen Klinik zu Tübingen*, Bd. i. S. 420—451. Tübingen, 1884. [A paper on the extirpation of goitre and of the thyroid gland.]

213. *Guéniot: Rachitisme développé et guéri avant la naissance. *Revue Mensuelle des Maladies de l'Enfance.* Paris, 1884. [= No. 202.]

214. Bouchard, A.: Du nanisme (à propos de la naine dite Princesse Paulina). *Journal de Médecine de Bordeaux*, Année xiv. pp. 276—279. Bordeaux, 1884—1885. [Account of Pauline Musters, with a letter from Dr L. van der Moolen giving particulars of her birth. Then follows a discussion of the possible causes of dwarfism. Pedigree No. 728: see Bibl. Nos. 129, 196, 215 and 322.]

215. de Mortillet, Adrien: La Princesse Paulina. *Bulletins de la Société d'Anthropologie de Paris*, 3e Série, Tome viii. p. 446. Paris, 1885. [See Bibl. Nos. 129, 196, 214 and 322.]

216. Ranke, H., und Voit, Carl: Ueber den amerikanischen Zwerg Frank Flynn, genannt General Mite, dessen Körper- und Geistesentwickelung und Nahrungsbedarf. *Archiv für Anthropologie*, Bd. xvi. S. 229—239. Braunschweig, 1885. [Pedigree No. 740.]

217. Kassowitz, Max: *Die normale Ossification und die Erkrankungen des Knochensystems bei Rachitis und hereditären Syphilis*, Bd. ii. S. 35—50. Wien, 1885. [A discussion on congenital Rachitis. See also Bibl. 175.]

218. Marchand, F. J.: Ueber die Synostose d. Schädelbasis bei sogenannter foetaler Rachitis. *Tageblatt der* 58. *Naturforschenden Sammlung zu Strassburg*, S. 422—423. Strassburg, 1885. [Discusses Grawitz's Case (Bibl. No. 219) and gives a short description of a child, but no measurements or family history.]

219. Grawitz, P.: Ein Fötus mit cretinistischer Wachsthumsstörung des Schädels und der Skeletsknochen. *Virchow's Archiv*, Bd. c. S. 256—262. Berlin, 1885. [Description of a female achondroplasic(?) foetus. No family history.]

220. Ehrlich, N.: Untersuchungen über die congenitalen Defecte und Hemmungsbilden der Extremitäten. *Virchow's Archiv*, Bd. c. S. 107—138. Berlin, 1885. [S. 116—119 describe a foetus 39 cm. in length with abnormally short limbs.]

221. von Ferro, R.: Ueber einen Fall von Rachitis congenita. *Wiener medizinische Presse*, Jahrgang xxvi. S. 374—375. Wien, 1885. [Case of a boy born at term, either still-born, or died after birth. No measurements but those of the skull given. The parents were healthy and had a healthy child aged 2½ years.]

222. Rohrer, F.: Ein Fall von Zwerghaftigkeit als Beitrag zur Aetiologie der Wachsthumshemmung. *Virchow's Archiv*, Bd. ci. S. 197. Berlin, 1885. [Pedigree No. 827.]

223. Schildowsky, Emil: *Ueber sogenannte fötale Rachitis*. Inaug. Diss. Erlangen, 1884. [A general discussion of the subject with description of a still-born child, length 37 cm., with very short thick curved extremities. It had been described as a "new-born cretin" and was the tenth child of a mother suspected of syphilis all whose children had either been still-born or had died shortly after birth.]

224. Moreau, Paul: *Fous et Bouffons*. Paris, 1885. [pp. 15—27, Rachitisme. pp. 48—50, Crétinisme. pp. 65—145, Nains. pp. 68—72, Nains dans l'antiquité. pp. 72—84, Peuples Nains de l'Afrique. pp. 84—92, Les véritables nains...mention: Gibson, Anne Shepherd, Lolkes, Tom Thumb, Lavinia Warren, Bébé. pp. 93—96, Fabrication des nains. pp. 96—119, Court Dwarfs under various courts. pp. 119—140, Bébé, Boruwlaski, Tom Thumb, General Mite, Millie Edwards, Prince and Princess Colibri, etc. pp. 140—145, Nains en Orient.]

225. Dyes, A.: *Beschreibung eines Falles von Pelvis nana mit kindlichen Habitus bei einer zeugungsfähigen Zwergin*. Inaug. Diss. Freiburg, 1885. [A general account of "Pelvis nana" is given, followed by a description of his case. See Pedigree No. 771.]

226. Brandt, Alexander: Ein extremer Fall rachitischer Verkrüppelung. *Virchow's Archiv*, Bd. civ. S. 540—548. Berlin, 1886. [Description of a female aged from 50 to 60. Length of trunk 57 cm. Length of lower extremities 49 cm. No family history.]

227. Arendes, Adolf: *Ueber Zwergbildung*. Inaug. Diss. Georg-August Universität zu Göttingen. Göttingen, 1886. [Probably rickety dwarfs. Pedigree No. 814.]

228. Fournier, Alfred: *La syphilis héréditaire tardive*, pp. 25—32. Paris, 1886. [Discusses the effect of "hereditary" syphilis on growth: several cases are given. No family history.]

229. Taruffi, Cesare: Storia di un caso di pseudo-rachite fetale. *Bullettino delle scienze mediche della Società Medico-Chirurgica di Bologna*, Anno 57, Serie 6a, T. xvii. pp. 211—212. Bologna, 1886. [Description of a new-born infant which died just after birth with short curved badly formed limbs and many other anomalies. No family history is given, but it is probably the same case as is reported in Bibl. No. 209.]

230. Hutchinson, Sir Jonathan: Congenital absence of hair and mammary glands with atrophic condition of the skin and its appendages in a boy whose mother had been almost wholly bald from alopecia areata from the age of six. *Medico-Chirurgical Transactions*, Vol. lxix. pp. 474—477. London, 1886. [This is same case as is described in Bibl. No. 311.]

231. Daubés, Guyot: Les Nains et les Géants, les variations de la stature humaine. *La Nature*, T. xxviii. pp. 18—22, 193—194, 242—244, 262—263. Paris, 1886—1887. [A series of short papers on variations in the human stature and of causes which influence them. The papers appear to be unimportant and of little scientific value.]

232. Brongiart, Chas.: Une famille de Nains. *La Nature*, T. xxix. pp. 179—182. Paris, 1887. [p. 180 gives a picture of Balthazar Zimmermann described by Quatrefages, Bibl. No. 187, who from the cut does not appear to be achondroplasic[1]: see our Plate JJ (71[a]). p. 181 gives a picture of the dwarf family Kostesky, Pedigree No. 744: see our Plate KK (75).]

232[b]. Klebs, Edwin. Vererbte hereditäre Krankheitsanlagen. *Die Allgemeine Pathologie oder die Lehre von den Ursachen und den Wesen der Krankheitsprocesse*, Theil i. Kap. iii. p. 28. Jena, 1887. [Only a few remarks on the heredity of dwarfism, with a reference to Veratti's and Luigi Frank's dwarf pedigrees: see Bibl. Nos. 67 and 116[b].]

233. *Sabudini: *Alger Médical*, Mai—Juin 1887. [According to Regnault, Bibl. No. 411, Sabudini reported the case of a rachitic dwarf to the Société Médicale d'Algers. She was enceinte and he used the cephalotribe. Regnault reproduces a picture of this dwarf and says she was achondroplasic.]

234. De Quatrefages de Breau, J. L. A.: *Les Pygmées*. Paris, 1887. [On pygmies in general.]

235. Lauro, Vincenzo: Della rachitide nella vita endouterina. *Annali di Ostetricia e Ginecologia*, Anno ix. pp. 385—429. Milano, 1887. [Pedigree No. 617.]

236. Storp, Joh.: *Untersuchungen über fötale Rachitis*. Plate. Inaug. Diss. Königsberg, 1887. [A recapitulation of cases described by other authors followed by measurements, autopsy and microscopical examination of two new cases. (1) A still-born female infant, tenth child of a healthy mother aged 42. Three of the children had died young, six were alive and healthy. The father had suffered from catarrh of the stomach. Length of infant 40·5 cm., weight 2600 grammes. The extremities were remarkably short and thick. (2) An illegitimate female infant, who died half an hour after birth. The mother, aged 24, had been sickly in youth and had had a previous child who died, aged 6 months, of atrophy. The infant weighed 2850 grammes, length 38·5 cm., and closely resembled Case (1) in appearance.]

237. Doutrebente et Manouvrier, L.: Le cerveau, le crâne, etc., d'un nain rachitique et aliéné. *Comptes Rendus de l'Association française pour l'Avancement de Science*, T. xvii. 2[e] P[t], pp. 405—412. Paris, 1888. [This paper is mainly a description of the brain and skull of a dwarf, Mazar by name. He was rachitic, born in Paris, stature 133 cm. In youth he had been a porcelain painter; he died insane aged 71. The left femur was curiously deformed. The neck of the femur not having been able to support the weight of the body, the head of the femur was 4 cm. below the lower edge of the great trochanter. There is no family history except that his father was also insane.]

238. Kirchberg, Adolf: *Ueber einen Fall von sogenannter fötaler Rachitis mit doppelzeitiger Hüftgelenk-Subluxation*. Inaug. Diss. Marburg, 1888. [A long and minute description is given of a new-born female infant with short body and very short thick extremities. The total length was 31·5 cm. The child had a cleft palate and bilateral dislocation of the hip joint. She was the first-born child of healthy parents. A short general discussion of the subject follows.]

239. Quisling, N. A.: Studien über Rachitis. *Archiv für Kinderheilkunde*, Bd. ix. S. 293—355. Stuttgart, 1888. [p. 296 is on congenital rachitis.]

240. Barnum, P. T.: *Life of P. T. Barnum*, pp. 70—79, 213, and 217—228. Buffalo, 1888. [Gives accounts of Mr and Mrs Tom Thumb, and Commodore Nutt.]

241. Caruso, Francesco: Die neuesten Ergebnisse des conservativen Kaiserschnittes mit Uterusnaht (nach Sänger's Methode und anderen Nahtverfahren). *Archiv für Gynaekologie*, Bd. xxxiii. S. 211—269. Berlin, 1888. [p. 219 gives a case of Zweifel's, who allowed Caruso to publish it. Pedigree 840.]

242. Hutchinson, Sir Jonathan: An account of the skeleton of the Norwich Dwarf. Plates. *Transactions of the Pathological Society*, Vol. xl. pp. 229—235. London, 1889. [This dwarf was executed for the murder of his child and attempted murder of his wife. He was aged 35, height 4 feet 2 inches. His legs and arms were short, all the larger bones of his limbs being thick and remarkably short, but not curved in any way. Measurements are given. Pedigree No. 635.]

[1] Regnault (Bibl. No. 411) states on the basis of this picture that Zimmermann was myxoedematous.

243. Kirchberg, Adolf, und Marchand, Felix: Ueber die sogenannte fötale Rachitis (Mikromelia chondromalacica). *Ziegler's Beiträge zur pathologischen Anatomie und zur allgemeinen Pathologie*, Bd. v. S. 183—216. Jena, 1889. [Pedigree No. 629.]

244. Crimail, A.: Opération Césarienne. *Annales de Gynécologie*, T. xxxi. pp. 272—279. Paris, 1889. [Pedigree No. 633.]

245. Stilling, H.: Osteogenesis Imperfecta. Ein Beitrag zur Lehre der sogenannten foetalen Rachitis. *Virchow's Archiv*, Bd. cxv. S. 357—370. Berlin, 1889. [Pedigree No. 766.]

246. Charcot, Jean Martin, et Richer, Paul: Les Nains, les Bouffons et les Idiots. *Les difformes et les malades dans l'art*, pp. 12—51. Paris, 1889. [This gives reproductions of various statues and pictures of dwarfs, which are in the Museums and Picture Galleries of Europe and Egypt, and also information about pictures which are not reproduced.]

247. Porak, C.: De l'achondroplasie. *Nouvelles Archives d'Obstétrique et de Gynécologie*, T. iv. pp. 551—573. Paris, 1889. Also T. v. pp. 19—31, 60—68, 133—141, 223—233, 303—307, 380—387, 421—439. Paris, 1890. [General account with Pedigrees Nos. 650 and 685.]

248. Taruffi, Cesare: Microsomia. *Storia della Teratologia*, T. v. Cap. iii. pp. 432—472. Bologna, 1889. [Gives a bibliography of the literature on dwarfs, with short account of what each paper contains. Pedigrees Nos. 746 and 749.]

249. Schauta, Friedrich: Die Beckenanomalien. *P. Müller's Handbuch der Geburtshülfe*, Bd. ii. S. 221—496. Stuttgart, 1889. [pp. 289—291 give pelvic measurements of Katharina Merglas, a cowherd aged 37. She was a well-formed dwarf 107 cm. in height. Her skeleton is in the Pathological Anatomical Institute at Prague.]

250. Blau, Otto: *Ueber sogenannte fötale Rhachitis.* Inaug. Diss. Berlin, 1889. [There is a short general discussion of the subject and two cases are described. (1) Second illegitimate child of a small strongly built woman. The first child had died of teething convulsions. Her second child, a male, had short curved extremities. Total length 43 cm., weight 2300 grammes. (2) Third child of a healthy mother and weakly father, who as a child had begun to walk late. Their first child had walked later than normal children, and the second, aged 14 months, could not walk alone. The third child, a girl, died at birth. Her length was 38 cm. and weight 2160 grammes. She had very short curved extremities.]

251. Kassowitz, Max: Zur Theorie der Rachitis. *Wiener medicinische Wochenschrift*, 39 Jahrgang, S. 1024—1027, 1080—1084, 1156—1159, 1197—1201, 1305—1308, 1343—1345, 1371—1374, 1401—1405, 1439—1442. Wien, 1889. [On rachitis in general.]

252. *Porak, C.: Le crâne de Nicolas Ferry si connu sous le nom de Bébé, nain du roi de Pologne. *Bulletins et Mémoires de la Société d'Obstétrique et de Gynécologie de Paris*, 1890, p. 77. Paris, 1890.

253. Porak, C.: *De l'achondroplasie.* Clermont, 1890. [This is an offprint of No. 247.]

254. Baldwin, J. F.: A case of Porro-Caesarian operation. *Medical News*, Vol. lvii. pp. 138—141. Philadelphia, 1890. [Pedigree No. 621.]

255. Fayerabend, E.: *Ueber das Vorkommen der Rachitis bei Neugeborenen.* Inaug. Diss. Königsberg, 1890. [Discussion on the frequency and cause of rachitis in new-born children, with tables giving data of 180 children who were examined and some particulars about their mothers.]

256. von Recklinghausen: Ergebnisse der Section eines 18-jahrigen Zwerges. Naturwissenschaftlich-Medicinischer Verein in Strassburg i. E. Sitzung am 13. Juni, 1890. *Deutsche medicinische Wochenschrift*, 16 Jahrgang, S. 1110. Leipzig und Berlin, 1890. [The dwarf was A. Müller, aged 18, height 95 cm., well proportioned in all parts.]

257. Courtois-Suffit: Sur un cas d'arrêt de développement (Infantilisme). *Revue de Médecine*, 10e Année, No. 7, pp. 588—599. Paris, 1890. [Pedigree No. 802.]

258. Le Roux, Hugues, et Garnier, J.: *Acrobats and Mountebanks*, translated from the French by A. P. Morton, pp. 60—70. London, 1890. [Gives short accounts of various dwarfs who have been exhibited.]

259. Stanley, Sir H. M.: *The Great Forest of Central Africa, its Cannibals and its Pygmies*, pp. 20—26. London, 1890. [Gives an account of the pygmies he met with in Africa.]

260. Stanley, Sir H. M.: *In Darkest Africa*, Vol. ii. Chap. 23, pp. 90—101. London, 1890. [Gives an account of the pygmies of Central Africa.]

261. Baginsky, Adolf: Zur Kenntniss der congenitalen Makroglossie und der Beziehungen zwischen Makroglossie, Cretinismus, und congenitaler Rachitis. *Paediatrische Arbeiten, Henoch's Festschrift*, S. 514—531. Berlin, 1890. [Pedigrees Nos. 791, 792 and 793.]

262. Paltauf, Arnold: *Ueber den Zwergwuchs in anatomischer und gerichtsärztlicher Beziehung.* Wien, 1891. [A treatise on dwarf growth. Pedigree No. 727.]

263. Humphry, Sir George Murray: Dwarfs, true dwarfs and dwarfs from rickets. *British Medical Journal*, 1891, Vol. II. pp. 1187—1188. London, 1891. [Gives measurements of the skeleton of a true dwarf which he bought in Paris and mentions another dwarf.]

264. Landau, Max: *Ueber infantilen Habitus, infantile und Zwerg-Becken.* Inaug. Diss. Strassburg i. E. 1891. [He describes the characteristics of infantile and dwarf pelves and gives two cases of dwarfs bearing children. (1) See Pedigree No. 801. (2) A dwarf aged 25, height 33 cm. She said her parents and brothers and sisters were not very small. The labour was so long that clinical assistance was required and a small living child was extracted. A third case is given of a woman with a small pelvis, who had a small child, but apparently she was not a dwarf.]

265. Jacobsen, G. O.: A family of dwarfs. *The Lancet*, 1891, Vol. I. p. 1040. London, 1891. [Pedigree No. 692.]

266. Villa, F.: Un caso d' acondroplasia e del così detto rachitismo micromelico. *Annali di Ostetricia e Ginecologia*, Anno XIII. pp. 653—666. Milano, 1891. [Description of an achondroplasic foetus with some measurements and general remarks on achondroplasia.]

267. *Kundrat, H.: Ueber Wachsthumsstörungen des menschlichen Organismus. II. Des Knochensystems. *Schriften des Vereines zur Verbreitung naturwissenschaftlicher Kenntniss in Wien*, Bd. XXXI. S. 327. Wien, 1891.

268. *Mori, E.: Contributo allo studio anatomo-patologico delle rachitide endo-uterina. *Rivista di Ostetricia e Ginecologia*, T. II. 2 Plates, pp. 513, 561. Torino, 1891.

269. *Sagretti, C.: Nuova patogenesi e cura della rachitide. *Giornale internazionale delle scienze mediche*, N. S., T. XXII. pp. 81—94. Napoli, 1891. And *Atti d. Cong. pediat. ital.* 1890, pp. 337—352. Napoli, 1891.

270. Schmidt, Alexander: Zur Kenntniss des Zwergwuchses. *Archiv für Anthropologie*, Bd. XX. S. 43—81. Braunschweig, 1891—1892. [Pedigrees Nos. 683, 684, 786—790, 830, 831 and 834 (Jacob Hoeppner, Sophie Petersen, — Welsing, Therese Fend, Margaretha Reisberger, Wilhelm Willkowsky, Peter Rose, Heinrich Nisse and Jakob Maier).]

271. *Schantauer: Ein Fall von foetaler Rachitis. *Pester medizinisch-chirurgische Presse. Wochenschrift für die gesammte Heilkunde.* Budapest, 1892.

272. Ornstein, Bernhard: Zwerg in Athen. Plate. Verhandlungen der Berliner Gesellschaft für Anthropologie, Ethnologie und Urgeschichte. *Zeitschrift für Ethnologie*, Bd. XXXIV. S. 541—543. Berlin, 1892. [Pedigree No. 816.]

273. Scholz, Ludwig: *Ueber fötale Rachitis.* Inaug. Diss. Göttingen, 1892. [A general discussion on the subject of foetal rachitis and its connection if any with myxoedema, cretinism, etc. Particulars of three cases are given without family history, all had short extremities. (1) p. 17, a female infant who died soon after birth, length 33 cm. (2) p. 28, a female infant, length 36 cm. (3) p. 51, a female foetus, length 28 cm.]

274. Symington, Johnson, and Thomson, Henry A.: A Case of defective endochondral ossification in a Human Foetus (so-called cretinoid). Plate. *Reports from the Laboratory of the Royal College of Physicians, Edinburgh*, Vol. IV. pp. 237—254. Edinburgh, 1892. Also *Proc. R. Soc. Edinburgh*, Vol. XVIII. pp. 271—286. Edinburgh, 1890—1891. [Pedigree No. 821.]

275. Kaufmann, Eduard: *Untersuchungen über die sogenannte foetale Rachitis* (Chondrodystrophia foetalis). Berlin, 1892. [A general treatise on the subject. Bibliography. Pedigree No. 773.]

276. Lugeol, P.: Achondroplasie. *Mémoires et Bulletins de la Société de Médecine et de Chirurgie de Bordeaux*, 1892, pp. 379—397. Bordeaux, 1893. And **Journal de Médecine de Bordeaux*, T. XXII. p. 461. Bordeaux, 1892. [Two observations: (1) Female infant, still-born at 6 months, the first-born illegitimate child of a well-formed girl aged 22. The father of the child was also well formed. The length of the child was 27 cm. She had a large head and short limbs. (2) A girl of 18, height 120 cm., with large head and short muscular limbs. Measurements and description are given. She was enceinte for the first time. Her parents were well formed and had had 9 children, of whom 7 were alive. The girl was the third child; all the others were well formed except the seventh who had malformation of the skull and only lived 48 hours. Premature confinement at 6 months was brought on, result a well-formed foetus, length 17 cm.]

277. Schwarzwäller, G.: Ueber sogenannte fötale Rachitis. *Zeitschrift für Geburtshülfe und Gynaekologie*, Bd. xxiv. S. 90—99. Stuttgart, 1892. [Describes a male child 42 cm. in length with short extremities. It was the second child of a normal mother and was either still-born or died. He also describes a still-born female child, 44 cm. in length, with short extremities and six fingers on each hand and six toes on each foot.]

277b. Virchow, R.: Vorstellung des Knaben Dobos Janos. *Berliner Klinische Wochenschrift*, 1892, Jahrgang 29, S. 517. Berlin, 1892. [Pedigree No. 734. Cf. Bibl. No. 401.]

278. von Franqué, Otto: Ueber sogenannte foetale Rachitis. *Sitzungsberichte der physikalisch-medicinischen Gesellschaft zu Würzburg*, Jahrgang 1893, S. 80—93. Würzburg, 1893. [Pedigree No. 630.]

279. Kaufmann, Eduard: Die Chondrodystrophia hyperplastica. Ein Beitrag zu den fötalen Skeleterkrankungen. *Ziegler's Beiträge zur pathologischen Anatomie und zur allgemeinen Pathologie*, Bd. xiii. S. 32—62. Jena, 1893. [General account.]

280. Boeckh, G.: Ueber Zwergbecken. *Archiv für Gynaekologie*, Bd. xliii. S. 347—372. Berlin, 1893. [Pedigree No. 620 (Kipke family).]

281. Thomson, J.: Note on three living cases of achondroplasia. *Edinburgh Medical Journal*, Vol. xxxviii. Pt ii. pp. 1109—1113. Edinburgh, 1893. [No history of heredity, the three cases were not related and their families were all well grown.]

282. Müller, Sigfrid: Periostale Aplasie mit Osteopsathyrosis unter dem Bilde der sogenannten foetalen Rachitis. *Münchener med. Abhandlungen*, ii. Reihe, 7 Heft. München, 1893. [See Addenda to Bibliography for account of this paper.]

283. Hutchinson, Sir Jonathan: A short limbed polydactylous dwarf. Plate. *Archives of Surgery*, Vol. iv. pp. 305—306. London, 1893. [The plate of the skeleton is copied from Theodore Kerckring's *Spicilegium Anatomicum*, published in Amsterdam, 1670. The limbs are short. Both hands have seven digits, the right foot has eight and the left nine digits.]

284. Parvin, Theophilus: The Influence of Maternal Impressions upon the Foetus. *International Medical Magazine*, Vol. i. pp. 487—493. Philadelphia, 1893. [p. 488 gives a picture of "The turtle man," evidently achondroplasic. Parvin says the deformity was produced by the mother being frightened by a turtle a few weeks after pregnancy began.]

285. Herman, George Ernest: Cases of Caesarian Section. Remarks. *The Lancet*, 1893, Vol. ii. pp. 1508—1510 and 1565—1568. [p. 1566, Case 6, Pregnant woman aged 24. Height 3 ft. 6 in. Her stepmother said she was deformed from birth. Illustrations show characteristic achondroplasic type. Herman says deformity was partly due to rickets. Measurements are given. The mother recovered, the child was decomposing.]

286. Paal, Hermann: *Ueber sogenannte foetale Rachitis.* Inaug. Diss. Würzburg, 1893. [A general discussion of the views of various writers on the subject followed by a description of a case of his own. Pedigree No. 819.]

287. *Carton: *Du rachitisme intra-utérin.* Thèse. Paris, 1893.

288. Guéniot: Opération césarienne et rachitisme congénital. *Bulletin de l'Académie de Médecine*, T. xxix. pp. 99—100. Paris, 1893. [Pedigree No. 613.]

289. Guéniot: Opérations césariennes multiples. *Bulletin de l'Académie de Médecine*, T. xxxii. pp. 16—18. Paris, 1894. [Pedigrees Nos. 613 and 824.]

290. Kollmann, J.: Pygmäen in Europa. *Verhandlungen der anatomischen Gesellschaft auf der achten Versammlung in Strassburg i. E. vom* 13—16 *Mai*, S. 206—215. Jena, 1894. *Anatomischer Anzeiger. Ergänzungsheft zum Band* ix. Jena, 1894. [On the pygmy races of Europe.]

291. *Neumaier, H.: *Zur Kenntniss des Zwergwuchs nebst Beschreibung eines neuen Falles von Zwergwuchs beim Menschen.* Erlangen, 1894.

292. Mason, R. Osgood: A Case of Congenital Rickets. Plate. *Archives of Pediatrics*, Vol. xi. pp. 670—672. New York, 1894. [Pedigree No. 811.]

293. Townsend, Chas. W.: A Case of Congenital Rachitis. Plate. *Archives of Pediatrics*, Vol. xi. pp. 761—763. New York, 1894. [Pedigree No. 764.]

294. Tyson, Edward: A Philological Essay concerning the Pygmies of the Ancients. *Bibliothèque de Carabas*, Vol. ix. London, 1894. [Reprint of No. 15.]

295. Roth, Joseph Hermann: *Ueber einen Fall von Chondrodystrophia fötalis (sog. fötale Rachitis).* Inaug. Diss. Erlangen, 1894. [He discusses the views of various writers on the subject and gives a case of his own. The third child, a male, of a healthy father and a mother aged 28 whose mental faculties were sub-normal and who was said to be alcoholic. Measurements and a long description are given.]

296. Hirst, Barton Cooke: Two recent additions to the teratologic Collection in the Wistar and Horner Museum of the University of Pennsylvania. Specimens of Acephalus and Micromelus. 2 Plates. *The Medical News*, Vol. lxiv. pp. 184—185. Philadelphia, 1894. [Description of case of rachitis congenita micromelia.]

297. Maass, Karl: Die sogenannte Puppenfee Helene Gabler. Verhandlungen der Berliner Gesellschaft für Anthropologie, Ethnologie und Urgeschichte. *Zeitschrift für Ethnologie*, Bd. xxvi. S. 364. Berlin, 1894. [Pedigree No. 731.]

298. Salvetti, C.: Ueber die sogenannte foetale Rachitis. *Ziegler's Beiträge zur pathologischen Anatomie und zur allgemeinen Pathologie*, Bd. xvi. S. 29—41. Jena, 1894. [General discussion of the subject, with bibliography.]

299. *Porak, C., et Durante, G.: Sur un cas d'ostéogénèse anormale caractérisée par une résorption trop intense des travées osseuses tant d'origine cartilagineuse que périostéale. *Bulletins et Mémoires de la Société d'Obstétrique et de Gynécologie de Paris*, 1894, pp. 177—191. Paris, 1894.

300. Grotthoff, Franz: *Ueber einen Fall von sogenannter fötaler Rachitis.* Inaug. Diss. Berlin, 1894. [The symptoms of rachitis are described at length, followed by description of four cases, three of which he states had been published before the fourth case came under his own observation. (1) In *J. B. der Charité, pro* 1885. The second child of a tall strong woman aged 24. Its length was 36 cm., weight 2110 grammes. The head was very large and the extremities, which were curved and shapeless with elephantiasis, were only small appendages to the large trunk. (2) In *J. B. der Charité*, 1888—9. Second child of a healthy mother aged 26, whose first child though healthy at birth had died, aged 8 weeks, of convulsions. The father was healthy. Length of child 43 cm., weight 2300 grammes. The bones of the skull were so soft that the shape was altered by pressure. The bones of the arms were straight, there was crepitation in the elbow joints, but it could not be decided whether these were fractures or not. The thighs were short, thick and curved. The child lived. (3) Male foetus, length 31·5 cm., with short thick extremities. (4) Male foetus, length 36 cm. and of normal weight, died shortly after birth with abnormally short crooked legs; a very full description is given. The author afterwards discusses the question of the heredity of the disease.]

300[b]. Rieger, K.: Demonstration des sogenannten "Vogelkopf-Knaben" Dobos Janos aus Battonga in Ungarn. *Sitzungsberichte der Würzburger Physikalisch-medicinische Gesellschaft*, Jahrg. 1895, S. 113—128. Würzburg, 1896. [Says Dobos Janos was not rachitic or cretinous, and compares him with a true dwarf Charlotte Uehlein. His height was 107 cm., weight 13·2 kilogs. Cf. Bibl. Nos. 277[b] and 400[b].]

301. Apert, E.: Achondroplasie. *Bulletins de la Société Anatomique de Paris*, T. lxx. pp. 772—775. Paris, 1895. [Account of a female achondroplasic child, who died at birth. Length 31 cm. A first-born child but no family history.]

302. Manouvrier, L.: Observations d'un microcéphale vivant et la cause probable de sa monstruosité. *Bulletins de la Société d'Anthropologie de Paris*, 4[e] Série, T. vi. pp. 227—230. Paris, 1895. [Pedigree No. 785. See Bibl. No. 441[b].]

303. Lampe, Richard: *Ueber zwei Fälle von sogenannter foetaler Rachitis.* Inaug. Diss. Marburg, 1895. [Very full descriptions of two cases are given. (1) Infant unsexed, born in 35th week of pregnancy, died soon after birth, with short extremities, length 46 cm. (2) A male infant, length 45 cm., with extraordinarily short extremities. It either died or was still-born. The mother was an idiot with scoliosis, genu valgum and pes equinus, and had apparently been rendered pregnant by her own father.]

304. Hertoghe, E.: De l'influence des produits thyroïdiens sur la croissance. *Bulletins de l'Académie Royale de Médecine de Belgique*, iv[e] Série, T. ix. pp. 897—935. Bruxelles, 1895. [Pedigree No. 810.]

305. Buday, K.: Beiträge zur Lehre von der Osteogenesis Imperfecta. *Sitzungsberichte der kaiserlichen Akademie der Wissenschaften. Mathematisch-naturwissenschaftliche Classe*, Bd. civ. Abth. 3, S. 61—101. Wien, 1895. [pp. 88—89 describe foetal rickets.]

306. Meige, Henry: L'infantilisme, le féminisme et les hermaphrodites antiques. *L'Anthropologie*, T. vi. pp. 257—275, 414—432, 529—548. Paris, 1895. [Some cases of dwarfed growth.]

307. Margarucci: Du rachitisme foetal. *La Semaine Médicale*, 15e année, 1895, pp. 486—487. Paris, 1895. [Merely a notice of a communication on foetal rickets made the 10th meeting of the Italian Chirurgical Society at Rome, Nov. 1895. No particular case mentioned.]

308. Haliburton, R. G.: Survivals of Dwarf Races in the New World. *Reprint from the Proceedings of the American Association for the Advancement of Science*, 1894, Vol. xliii. pp. 337—344. Salem, 1895. [Short paper on the Dwarf Races of both Old and New Worlds.]

309. Haliburton, R. G.: Dwarf Survivals, and Traditions as to Pygmy Races. *Proceedings of the American Association for the Advancement of Science*, 1895, Vol. xliv. pp. 285—286. Salem, 1896. [Title explains contents.]

310. Ziegler, E.: Durch Entwickelungs- und Wachsthumsstörungen bedingte Knochenveränderungen. *Ziegler's Lehrbuch der speciellen pathologischen Anatomie*, Bd. ii. pp. 163—165. Jena, 1895. [On dwarf growth, micromelia or nanosomia, with plates of two dwarf female skeletons. (1) A woman aged 31 and 118 cm. in height. (2) A woman aged 58 and 117 cm. in height.]

311. Hutchinson, Sir Jonathan: Two cases of dwarfdom with arrested development of skin and appendages. *Archives of Surgery*, Vol. vi. pp. 140—142. London, 1895. [This gives further particulars of case previously published. See Bibl. No. 230. A boy aged 14 in 1894, height 43 inches. He had no hair or eyelashes. His mother was bald from girlhood. The second case is a similar case of Hastings Gilford.]

312. Haliburton, R. G.: Zwergstamme in Sud und Nord Amerika. Verhandlungen der Berliner Gesellschaft für Anthropologie, Ethnologie und Urgeschichte. *Zeitschrift für Ethnologie*, Bd. xxviii. S. 470—472. Berlin, 1896. [Letters from Haliburton to Virchow about pygmies in Guiana and pygmy graves in Tennessee.]

313. Verneau, B.: Nains et Géants. *L'Anthropologie*, T. vii. p. 118. Paris, 1896. [Merely a short note on Auguste Tuaillon and two giants: see Bibl. No. 324.]

314. Feldman, Gustav: Ueber Wachsthumsanomalien der Knochen. *Ziegler's Beiträge zur pathologischen Anatomie und zur allgemeinen Pathologie*, Bd. xix. S. 565—646. Jena, 1896. [pp. 594 *et seq.* discuss various kinds of dwarf growth. There is a long bibliography and tables of measurements.]

315. Hutchinson, Sir Jonathan: A short-limbed dwarf, multiple exostoses. *The Clinical Journal*, Vol. viii. p. 333. London, 1896. [The subject was a man of deficient intellect, aged 38. Height 4½ feet.]

316. Hertoghe, E.: Diagnostic de la possibilité d'une reprise de croissance dans les arrêts ou retards notables dûs au myxoedème, à l'hyperazoturie et au rachitisme. *Bulletins de l'Académie Royale de Médecine de Belgique*, iv^e^ Série, T. x. pp. 564—569. Bruxelles, 1896. [Paper showing the possibility of deciding, by a radiographic examination, whether or not any treatment would increase growth.]

317. Meige, Henry: Les Nains et les Bossus dans l'Art. 3 Plates. *Nouvelle Iconographie de la Salpêtrière*, T. ix. pp. 161—168. Paris, 1896. [Descriptions of pictures of various dwarfs.]

318. Kollmann, J.: Der Mensch. Nuesch's *Schweizerbild. Neue Denkschriften der allgemeinen schweizerischen Gesellschaft für die gesammten Naturwissenschaften*, Bd. xxxv. S. 134—152. Zürich, 1896. [On the bones of an ancient pygmy race found in Switzerland.]

319. Makins, G. H.: A Case of Intra-uterine rickets. *St Thomas's Hospital Reports*, N.S., Vol. xxiii. pp. 121—124. London, 1896. [Description of a child aged 14 days, very small and weighing only 5½ pounds in its clothes and with every deformity common to ordinary rickets. There are no measurements and no statement is made with regard to the length of the limbs, but the bones were curved and thickened. The mother was healthy and had an elder child who had also developed rickets.]

320. Chambrelent: Sur un cas d'achondroplasie, cause de dystocie foetale. **Gazette hebd. des sciences médicales de Bordeaux*, T. xvii. p. 271, Bordeaux, 1896, and **Journal de Médicine de Bordeaux*, T. xxvi. p. 204, Bordeaux, 1896.

321. Tissié: Un cas de double nanisme fraternel. Présentation de malades. *Mémoires et Bulletins de la Société de Médecine et de Chirurgie de Bordeaux*, 1896, pp. 408—415. Bordeaux, 1897. [Pedigree No. 706.]

322. Nagel, J. Darwin: Princess Paulina. *Pediatrics*, Vol. ii. No. 8, pp. 369—373. New York and London, 1896. [Pedigree No. 728: see Bibl. Nos. 129, 196, 214, 215.]

323. Maass, Karl: Birmesischen Zwerge mit einem Salzburger Riesen. Verhandlungen der Berliner Gesellschaft für Anthropologie, Ethnologie und Urgeschichte. *Zeitschrift für Ethnologie*, Bd. xxviii. S. 524—526. Berlin, 1896. Also Bd. xxx. S. 344, Berlin, 1898, and Bd. xxxi. S. 455, Berlin, 1899. [Pedigree No. 709.]

324. Manouvrier, L.: Sur le nain Auguste Tuaillon, et sur le nanisme simple avec ou sans microcéphalie. *Bulletins de la Société d'Anthropologie de Paris*, 4e Série, T. vii. pp. 265—289. Paris, 1896. [Pedigree No. 736. In the discussion on the paper Pedigrees Nos. 738, 739 and 775 are given, also a reference to family in Pedigree No. 744: see Bibl. No. 313.]

325. Neuring, A.: Ueber das Vorkommen von Zwergen neben grossen Leuten in demselben Volke. Verhandlungen der Berliner Gesellschaft für Anthropologie, Ethnologie und Urgeschichte. *Zeitschrift für Ethnologie*, Bd. xxix. S. 91—94. Berlin, 1897. [A notice of Herberstain's *Rerum Muscoviticarum Commentario*, published 1557, about a people who lived between Prussia and Livonia, in the families of which were both very tall children and dwarfs.]

326. *von Geldem-Egmond, Frau Gräfin: *Beitrag zur Casuistik der sogenannten fötalen Rachitis.* Inaug. Diss. Zürich, 1897.

327. *Manouvrier, L.: Le période de croissance d'un nain. *Journal de clinique et de thérapeutique infantiles*, T. v. pp. 1009—1012. Paris, 1897.

328. Haliburton, R. G.: *How a pygmy race was found in North Africa and Spain, and papers on other subjects*, pp. 1—96. Toronto, 1897. [This contains the following papers on dwarfs. (1) Notes on Mt. Atlas and its Traditions. (2) Dwarf Races and Dwarf Worship. (3) The Dwarfs of Mt. Atlas. (4) Some further notes on the existence of Dwarf Tribes south of Mt. Atlas. (5) Racial Dwarfs in the Atlas and Pyrenees, Pts i., ii. and iii. (6) Survivals of Dwarf Races in the New World. (7) Dwarf Survivals and Traditions as to Pygmy Races. (8) The Tiki-Tiki. (9) The dwarf domestic animals of Pygmies.]

329. Klinger, P.: *Ueber einen Fall von Chondrodystrophia hyperplastica und seine Beziehung zur sogenannten foetalen Rachitis.* Inaug. Diss. Freiburg i. B. 1897. [An account of the views of various authors on the subject of rachitis, with particular reference to Kirchberg, Marchand and Kaufmann. A new case is described which was preserved in the Pathological Institute. Length of foetus 27·5 cm.]

329b. Hertoghe, E.: Nouvelles Recherches sur les arrêts de croissance et d'infantilisme. *Bulletin de l'Académie de Médecine de Belgique*, 4e Série, T. xi. pp. 750—760. Bruxelles, 1897. [Chiefly an article on the effect of the thyroid treatment on growth, with examples.]

330. Manouvrier, L.: Observations sur quelques nains. *Bulletins de la Société d'Anthropologie de Paris*, 4e Série, T. viii. pp. 654—664. Paris, 1897. [Remarks on some dwarfs, including "Princesse Blanche." Pedigree No. 737. See also Bibl. Nos. 341 and 345.]

331. Osler, Wm.: Sporadic Cretinism in America. *Transactions of the Congress of American Physicians and Surgeons*, 1897, Vol. iv. pp. 169—206. New Haven, Conn. 1897. [Pedigree No. 639.]

332. Gould, George M., and Pyle, Walter L.: *Anomalies and Curiosities of Medicine*, pp. 333—343. Philadelphia, 1897. [Gives accounts, not always trustworthy, of most of the well-known dwarfs. Pedigree No. 697 (Rossow Brothers).]

333. Hutchinson, Sir Jonathan: A case of hypertrophy of the gums with general dwarfism. *Edinburgh Medical Journal*, N. S., Vol. i. Pt ii. pp. 117—118. Edinburgh, 1897. [Gives account of a youth who was a dwarf in stature, age 25. No measurements and no family history. Mentions two other cases of dwarfism.]

334. Brissaud, E.: De l'infantilisme myxoedémateux. Plates. *Nouvelle Iconographie de la Salpêtrière*, T. x. pp. 240—282. Paris, 1897. [Some of the cases appear to be of dwarfed stature. No family history.]

335. Uhthoff, W.: Ein Beitrag zu den Sehstörungen bei Zwergwuchs und Riesenwuchs resp. Akromegalie. *Berliner klinische Wochenschrift*, xxxiv. Jahrgang, S. 461—464, 501—504, 537—540. Berlin, 1897. [pp. 461—464 are on dwarf growth. Account of a girl aged 14, height 131 cm. Her growth had stopped at age of 9 and eyes became affected. Pedigree No. 808.]

336. Tschistowitsch, Th.: Zur Frage von der angeborenen Rachitis. *Virchow's Archiv*, Bd. cxlviii. S. 140—177, 209—233. Berlin, 1897. [A general discussion on rachitis.]

337. Regnault, Felix: Le Dieu Egyptien Bès était myxoedémateux. *Bulletins de la Société d'Anthropologie de Paris*, 4e Série, T. viii. pp. 434—439. Paris, 1897. [This is a paper to prove that Bes was in type myxoedematous and a cretin.]

338. Jacques, V.: Les nains. *Bulletin de la Société d'Anthropologie de Bruxelles*, T. xvi. pp. 282—302. Bruxelles, 1897. [A paper giving an account of the pygmies in ancient literature and of the pygmy races in Africa.]

339. Meige, Henry, et Allard, Felix: Deux Infantiles. Infantile myxoedémateux et infantile de Lorain. *Nouvelle Iconographie de la Salpêtrière*, T. xi. pp. 105—113. Paris, 1898. [First Case, Pedigree No. 800. Second Case, no family history.]

340. Hitschmann, Richard: Augenuntersuchungen bei Cretinismus, Zwergwuchs und verwandten Zuständen. *Wiener klinische Wochenschrift*, xi. Jahrgang, S. 655—666. Wien, 1898. [He discusses the condition of eyes in cases of dwarf growth. Pedigree No. 732.]

341. Capitan: Photographies anthropologiques. Données physiologiques. *Revue Mensuelle de l'Ecole d'Anthropologie de Paris*, T. viii. pp. 112—113. Paris, 1898. [Account of Blanche B., known as "La Princesse Blanche" or "La Naine de Bazas"—written in conjunction with Manouvrier's paper on same subject. See Bibl. No. 345 and Pedigree No. 737.]

342. Laffargue, Evariste: Sur un cas d'achondroplasie. *La Médecine Moderne*, 8e Année, pp. 364—366. Paris, 1898. [This is the case of a man aged 50, height 98 cm., of Berber-negro race. Full measurements are given but no family history.]

343. Laffargue, Evariste: Quatre nouveaux cas d'achondroplasie. *La Médecine Moderne*, 8e Année, pp. 515—516. Paris, 1898. [Case (1), a man aged about 40, height 121 cm., of Berber-negro race. Case (4), a man aged about 50, height 111 cm., pure negro. Cases (2) and (3) are brothers. Pedigree No. 673.]

344. *Manouvrier, L.: Le nain Boffy. [Abst.] *Journal des connaissances médicales pratiques et de pharmacologie*, 1898, p. 19. Paris, 1898.

345. Manouvrier, L.: Photographies anthropologiques, Mensurations. Naine de Bazas. 5 Plates. *Revue Mensuelle de l'Ecole d'Anthropologie de Paris*, T. viii. p. 111. Paris, 1898. [Measurements of Blanche B., la naine de Bazas or la Princesse Blanche, with three pictures of Blanche and two of her mother. Pedigree No. 737. See also Bibl. Nos. 330 and 341.]

346. Maygrier, Chas.: Présentation d'un foetus achondroplasique. Présentation de photographies, du moulage, d'une radiographie et du squelette. *Bulletin de la Société d'Obstétrique de Paris*, T. i. pp. 248—255. Paris, 1898. [Pedigree No. 761.]

347. Boissard: Opération césarienne faite à la Maternité; mère et enfant vivants. *Bulletin de la Société d'Obstétrique de Paris*, T. i. pp. 33—36. Paris, 1898. [Pedigree No. 762.]

348. John, Rudolf: *Ueber die sogenannte foetale Rachitis.* Inaug. Diss. Berlin, 1898. [He divides rachitic affections into three groups, (1) true post-uterine rachitis, (2) congenital rachitis, (3) so-called foetal rachitis, gives the characteristic symptoms of each group and describes fully a case which appeared on S. 15 *Polikl. Journal*, No. 766, 1897—8. A woman aged 38, who had had seven living children and two miscarriages, came to hospital for her confinement. She stated that she and her husband were healthy and their families were healthy. The skull of the child, a male, was fractured in the endeavour to extract it. The length was about 42 cm. The extremities were short and there was no external division between the upper- and forearm or between the thigh and leg.]

349. *Regnault, Felix: *Des altérations craniennes dans le rachitisme.* Thèse. Paris, 1898.

350. Weiss, Siegfried: Demonstration eines Falles von echten proportionirten Zwergwuchs in Kindesalter. *Wiener klinische Wochenschrift*, xi. Jahrgang, S. 1212. Wien, 1898. [Description of a dwarf boy, 7 years old, 76 cm. in height. No family history.]

351. Johannessen, Axel: Chondrodystrophia foetalis hyperplastica. *Ziegler's Beiträge zur pathologischen Anatomie und zur allgemeinen Pathologie*, Bd. xxiii. S. 351—374. Jena, 1898. [Pedigree No. 759.]

352. Garrod, Archibald: A Case of Achondroplasia. Plate. *Transactions of the Clinical Society*, Vol. xxxi. pp. 294—295. London, 1898. [Description of an achondroplasic girl, aged 6, height 37½ inches. No family history.]

353. Joachimsthal: Fall von sogenannter fötaler Rachitis. *Berliner klinische Wochenschrift*, 1899, Jahrgang 36, S. 245—246. Berlin, 1899. [A girl aged 11, with abnormally short extremities. Her growth had stopped at the age of 3. Height when seen in 1898, 83 cm. The tips of her fingers with arms hanging barely reached the trochanters. She was intelligent. A description is given but no measurements or family history.]

354. Turner, Wm.: On Achondroplasia. *The Practitioner*, Vol. lxiii. pp. 263—277. London and New York, 1899. Also: A case of achondroplasia or chondrodystrophia hyperplastica, aet. 10½ years. *Transactions of the Clinical Society*, Vol. xxxii. pp. 269—271. London, 1899. [Pedigree No. 636.]

355. Flemming, Chas. E. S.: Achondroplasia. *The Bristol Medico-Chirurgical Journal*, Vol. xvii. No. 63, pp. 21—27. Bristol, 1899. [Pedigree No. 832.]

356. Edgeworth, F. H.: Case of Achondroplasia. Plate. *Bristol Medico-Chirurgical Journal*, Vol. xvii. pp. 27—29. Bristol, 1899. [Description of an achondroplasic man aged 59, height 4 ft. 8 inches, one of a numerous family the other members of which were of normal stature. He had no children. Edgeworth states he knew four other cases in Bristol by sight.]

357. Schwenderer, Burkhard: *Untersuchungen über Chondrodystrophia foetalis.* Inaug. Diss. Basel 1899. [Schwenderer adopts Kaufmann's classification. pp. 7—21 describe four cases: (1) length 40 cm., no family history; (2) length 37 cm., offspring of normal parents whose other children were normal; (3) length 36 cm., no family history; (4) Pedigree No. 777. pp. 48—49 describe two more cases: (1) length 28·5 cm., with normal parents, brothers and sisters; (2) a male foetus 21 cm. in length, eleventh child of the mother. These two cases are, he says, types of micromelia, but are free from chondrodystrophia foetalis.]

358. Stoeltzner, Wilhelm: Fötales Myxödem und Chondrodystrophia foetalis hyperplastica. *Jahrbuch für Kinderheilkunde*, Bd. l. S. 106—123. Leipzig, 1899. [A description of the autopsies of two foetuses is given and the author points out that all cases described as foetal rachitis do not belong to the same type of disease.]

359. Schmidt, M. B.: Allgemeine Pathologie und pathologische Anatomie der Knochen. *Lubarsch und Ostertag's Ergebnisse der allgemeinen Pathologie und pathologischen Anatomie des Menschen und der Tiere*, iv. Jahrgang, S. 531—650. Wiesbaden, 1899. [S. 599—612, Chondrodystrophia foetalis. S. 612—617, Osteogenesis Imperfecta. S. 617—626, Kretinismus. S. 626—632, Wahrer Zwergwuchs (Nanosomie). S. 632—650, Die Rachitis. There is a bibliography, but no pedigrees or special cases.]

360. *Spillmann, L.: *Rachitisme.* Thèse. Nancy, 1899—1900.

360[b]. Bernhard, L.: Fall von fötaler Rachitis. *Berliner klinische Wochenschrift*, 1899, Jahrgang 36, S. 245. Berlin, 1899. [Child aged 9. Parents and sisters healthy. It had very short curved extremities. Description given but no measurements. Said to be a case of what Marchand calls "Mikromelia chondromalacia."]

361. *Baginsky, A.: Rachitis foetalis. *Berliner medicinische Gesellschaft*, 15 Feb. 1899. [Not found.]

362. Henoch, Eduard: Die Rachitis. *Vorlesungen über Kinderkrankheiten*, 10th Edition, S. 865—886. Berlin, 1899. [pp. 878—880 on foetal rachitis.]

363. Joachimsthal: Ueber Zwergwuchs und verwandte Wachsthumsstörungen. *Deutsche medicinische Wochenschrift*, xxv. Jahrgang, No. 17, S. 269—271. No. 18, S. 288—290. Berlin, 1899. [In No. 17 some true dwarfs are described. One of them from Königsberg was well built and mentally well developed. At age of 36 his height was 128 cm. He said he had a sister backward in growth. In No. 18 radiographs and descriptions of achondroplasic dwarfs are given. No family history.]

364. Sternberg, Maximilian: Vegetationsstörungen und Systemerkrankungen der Knochen. 1899. *Nothnagel's specielle Pathologie und Therapie*, Bd. vii. 2[te] Hälfte. Wien, 1903. [104 pages on dwarf growth in general with long bibliography. This work is largely used pp. 363—370 above.]

365. Hildebrandt, H.: Ueber Osteogenesis Imperfecta. *Virchow's Archiv*, Bd. clviii. S. 426—444. Berlin, 1899. Also *Münchener medicinische Wochenschrift*, 1899, S. 30. München, 1899. [Description of an infant which died nine hours after birth, with very short limbs. Mother healthy. No family history. And a general discussion on Osteogenesis Imperfecta, Chondrodystrophia foetalis, etc.]

366. Schuchardt, Karl: Intrauterin-erworbene Skeletatrophie (Früher sogenannte fötale Rachitis). Die Krankheiten der Knochen und Gelenke. *von Bruns' Deutsche Chirurgie*, Bd. 28, Kapitel viii. S. 58—64. Stuttgart, 1899. [The contents of the chapter are the following: Fötaler Kretinismus mit Hemmung des Langenwachsthums (chondralen Dysplasie). Fötaler Kretinismus, Victor Horsley. Pseudo-rachitismus, Eberth. Kretinoid oder Kretinoide Dysplasie, Klebs. Chondrodystrophia foetalis, Kaufmann. Micromelia Chondromalacia, Marchand. Achondroplasia, Porak. One description is given of them all.]

367. Thomson, John: Case of a peculiar form of dwarfed growth with notes of the post mortem by Jessie Macgregor. *The Scottish Medical and Surgical Journal*, Vol. vi. No. 3, pp. 209—214. Edinburgh, 1900. [Pedigree No. 779.]

367[b]. Schieb: Ueber Osteogenesis Imperfecta. *von Bruns' Beiträge zur klinischen Chirurgie*, Bd. xxvi. Supplement, S. 93—119. Tübingen, 1900. [Pedigree No. 767.]

368. Article: Les Bouts d'Homme. *Almanach Hachette*, 1900, pp. 356—357. Paris, 1900. [Pictures of the following dwarfs are given: Doubrof, aged 31, height 107 cm. Hop o' my Thumb, aged 12, height 67 cm. Ida Blumenthal, aged 22, height 80 cm. Hippolyte Bureau, an achondroplasic dwarf, died aged 29, height 80 cm. Adrien Esmilaire, aged 16, height 69 cm. Victor Still, died aged 47, an achondroplasic dwarf, height 80 cm. Paul Naf, age not given, height 84 cm. La petite reine Mab, aged 19, height 70 cm.]

368[b]. Spillmann, Louis: Maladie de Barlow. Rachitisme intrautérin. *Le Rachitisme*, Chap. iii. pp. 116—127. Paris, 1900. [A book with an atlas dealing very fully with the subject of rachitis, giving various observations and also experiments on animals. Chap. iii. treats of achondroplasia.]

369. Gilbert, A., et Rathery, F.: Le Nanisme Mitral. *La Presse médicale*, 1900, No. 37, pp. 225—227, No. 38, pp. 231—233. Paris, 1900. [Three cases are given, but from modern standpoint they are not true dwarfs. They are termed cases of "Infantilisme du type Lorain et Faneau de la Cœur." They are (1) a male aged 54, height 142 cm.; (2) a female aged 39, height 150 cm.; (3) a female aged 52, height 140 cm. There is some family history for each case.]

370. Legry, T.: Trois Cas d'Achondroplasie. *La Presse médicale*, 1900, p. 105. [Merely a note about a Séance of the Société Anatomique de Paris stating that Legry had studied two achondroplasic skeletons and one achondroplasic body. The *Bulletin de la Société Anatomique* for 1900 does not contain a description of them.]

371. Marie, P.: L'achondroplasie dans l'adolescence et dans l'âge adulte. *La Presse médicale*, 1900. Deuxième Sém., No. 56, pp. 17—23. Paris, 1900. Also: *La Revue médicale*, 1900, p. 21. [Pedigree No. 674.]

372. Durante, G.: Deux cas d'achondroplasie avec examen histologique des os et du système nerveux. *Bulletins de la Société Anatomique de Paris*, 6[e] Série, T. ii. pp. 785—786. Paris, 1900. [A short description of the cases without family history or measurements. One mother was syphilitic, the other had a renal affection.]

373. Porak, C., et Durante, G.: Deux cas d'Achondroplasie avec examen histologique. *Annales de Gynécologie et d'Obstétrique*, July—August. Paris, 1900. [Seen in offprint.]

374. Colleville: Sur un cas d'achondroplasie chez l'adulte. *Union médicale du Nord-Est*, T. xxiv. pp. 205—210. Reims, 1900. [Account with photographs and radiograph of an achondroplasic dwarf aged 46, named Alexandre D..., normal sister and no family history.]

375. Porak, C., et Durante, G.: Deux cas d'Achondroplasie avec autopsie. *Congrès international des sciences médicales*. Paris, août, 1900. [The same as Bibl. No. 373.]

376. *Pauly et de Teyssier, B.: Un cas d'achondroplasie. *La Province médicale*, 1900, T. xiv. p. 409. Lyon, 1900.

377. Boquel, A.: Bassin rachitique; opération césarienne à terme; mère et enfant vivants. *Bulletin de la Société d'Obstétrique de Paris*, T. iii. pp. 416—418. Paris, 1900. [Pedigree No. 813.]

378. Hergott, Alphonse: Un cas d'achondroplasie. *Comptes Rendus de la Société d'Obstétrique de Gynécologie et de Paediatrie de Paris*, T. ii. pp. 38—57. Plate. Paris, 1900. [Female infant, second child of a tuberculous mother aged about 24, who had lost 12 brothers and sisters, most of them of tuberculosis. She herself had been obliged to remain three years in bed, on account of some illness which would have deformed her legs if they had not been kept straight by boards. She had also been operated on for simple unilateral hare-lip. The child, whose weight was 2200 grammes, died after taking a few breaths. She had a very large head and the limbs, especially the thighs, were very short. There is a short discussion of the various theories on the subject of rachitis.]

379. *Fede e Carace: *La Pediatria*, 1900, No. 2. Napoli, 1900.

380. *De Buck: L'Achondroplasie: *La Belgique Médicale*, 1900, No. 50, p. 737.

381. Kirk, Robt.: Case of Modified Cretinism. *Transactions of the Medico-Chirurgical Society of Glasgow*, Vol. ii. pp. 319—320. Glasgow, 1900. [Description of a boy aged 20½, height 4 ft. and ½ inch. No family history. The boy was bright and intelligent although below average capacity. See also Bibl. No. 390.]

382. Legry, T.: Achondroplasie. *V. Cornil et L. Ranvier's Manuel d'histologie pathologique*, T. I. pp. 799—800. Paris, 1901. [A very short article on the chief characteristics of achondroplasia.]

383. Noble, Wilson: Achondroplasia. *Archives of the Röntgen Ray*, Vol. v. No. 2, pp. 55—56 and Plate CIII. London, 1901. [Radiograph of the arm and ribs of a boy aged 8, with a short note by Mr Holderness for whom the radiograph was taken.]

384. Zimmern, A.: Sur un cas de rachitisme familiale. Plate. *Nouvelle Iconographie de la Salpêtrière*, T. xiv. pp. 299—304. Paris, 1901. [This gives a family history and pedigree of true rachitis: the plate is reproduced in present paper; see Plate Y (33—35).]

385. Cestan, R.: À propos d'un cas d'achondroplasie. *Nouvelle Iconographie de la Salpêtrière*, T xiv. pp. 277—289. Paris, 1901. [Pedigree No. 652.]

386. Apert, E.: Quelques remarques sur l'achondroplasie. Deux observations nouvelles de l'achondroplasie adulte. *Nouvelle Iconographie de la Salpêtrière*, T. xiv. pp. 288—298. Paris, 1901. [No family history of these cases given, but one of them was found later to be father of the girl described by Sevestre, Bibl. No. 492, Pedigree No. 612.]

387. Molin, Henri: *Étude Radiographique et Clinique sur la Dyschondroplasie*, 21 Figs., pp. 60—66. Paris, 1901. [A memoir of 122 pages on Dyschondroplasia. pp. 60—66 are on Achondroplasia. There are sections on Osteogenesis Imperfecta, Osteomalacia, Rachitis and Osteogenic Exostoses.]

387[b]. Richer, Paul: Les Nains, les Bouffons, les Idiots. *L'Art et la Médecine*. Paris, 1901. [Gives account and reproductions of various pictures of dwarfs.]

388. Cestan, R., et Infroit, L.: Étude radiographique d'un cas d'achondroplasie. *Revue neurologique*, T. ix. pp. 437—438. Paris, 1901. [Merely a radiographic examination of the case, without measurements or family history.]

389. Regnault, Felix: *Bulletin de la Société Anatomique de Paris*, 6[e] Série, T. iii. Paris, 1901. [pp. 178—179, Diagnostic de l'achondroplasie par l'examen macroscopique des os foetaux; pp. 179—181, Achondroplasie des os et du tronc; pp. 181—182, Achondroplasie partielle; pp. 182—185, Os d'adulte achondroplasique; pp. 185—187, Squelette d'achondroplase adulte (forme classique); pp. 187—189, Sur un squelette d'achondroplase adulte (type non-classique); pp. 189—192, Sur un squelette d'achondroplase adulte n'offrant pas tous les signes classiques de ce maladie; pp. 386—389, L'achondroplasie chez le chien; pp. 419—421, Des variétés d'achondroplasies foetales; pp. 424—426, Du crâne de l'achondroplase chez le foetus et chez l'adulte; pp. 507—509, Quelques nouveaux cas d'achondroplasie; p. 509, Nanisme vrai chez les adultes; pp. 559—560, Sur un squelette de foetus atteint d'achondroplasie hyperplasique et généralisée.]

389[b]. Apert, E. (1) Sur le traitement thyroïdien dans l'infantilisme. (2) Examen histologique du corps thyroïde et d'autres organes d'un sujet atteint d'infantilisme. *Bulletins de la Société de Pédiatrie de Paris*, mai et juin, 1901, pp. 1—8. Paris, 1901. [Two observations are given, one in which thyroid treatment appeared to improve the child, the other in which the patient, a youth aged 19, height 126 cm., died and the autopsy showed the thyroid body larger than normal. Apert suggests thyroid treatment would have also succeeded in this case.]

389[c]. Apert, E. Traiteuent de l'Infantilisme et de la cryptorchide par les préparations thyroïdiennes. *Le Bulletin Médical*, 20 avril, 1901, pp. 1—35. Paris, 1901. [Three observations are given; the first two are stated to have been cured by thyroid treatment; the third, a youth aged 21, height 115 cm., died of tuberculosis shortly after his entrance to hospital. The father said he had ceased to grow at age of 9. He was the second of seven children, all of whom had died young. The autopsy showed the thyroid body was well developed and healthy, but in an infantile condition.]

390. Kirk, Robert: On serous vaccinia in connexion with cretinism and rickets. *The Lancet*, 1901, Vol. i. pp. 1266—1268. London, 1901. [One of the cases given in the paper appeared to members of the Med. Chir. Soc., Glasgow, not to be cretinism. See Bibl. No. 381.]

391. Patel: Nanisme isolite. *Gazette hebdomadaire de Médecine et de Chirurgie*, 1901, Année xlviii. No. 26, pp. 301—306. Paris, 1901. [Pedigree No. 812.]

392. Meige, Henry: Remarques complémentaires sur les nains dans l'art. Plate. *Nouvelle Iconographie de la Salpêtrière*, T. xiv. pp. 371—372. Paris, 1901. [Gives a list of statues and pictures of dwarfs, stating where they are to be found.]

393. Harbitz, Francis: Ueber Osteogenesis Imperfecta. *Ziegler's Beiträge zur pathologischen Anatomie und zur allgemeinen Pathologie*, Bd. xxx. S. 605—638. Plates. Jena, 1901. [Gives some account of the literature on the subject and an additional case of his own, an achondroplasic (?) illegitimate child of healthy parents, who died soon after birth. Length 37·5 cm.]

394. Collmann, Benno: Beitrag zur Kenntniss der Chondrodystrophia foetalis. *Virchow's Archiv*, Bd. clxvi. S. 1—12. Berlin, 1901. [A long description of a female foetus, length 33 cm., with very short limbs. No family history.]

395. Virchow, Rudolf: Rachitis foetalis, Phocomelie und Chondrodystrophia. *Virchow's Archiv*, Bd. clxvi. S. 192—194. Berlin, 1901. [A criticism of Collmann's paper. Bibl. No. 391.]

396. Nijhoff, G. C.: Baring bij Bekkenvernauwing. *Overdruk uit het Nederlandsch Tijdschrift voor Verloskunde en Gynaecologie*, 1901, Deel xi. pp. 1—64. 1901. [Pedigree Nos. 664 and 817. Two other cases, dwarfs having children, are given.]

397. Klein, Albert: Neuere Arbeiten über "die sogenannte fötale Rachitis." *Centralblatt für allgemeine Pathologie und pathologische Anatomie*, Bd. xii. S. 839—849. Jena, 1901. [A general discussion on foetal rachitis.]

397[b]. *Eserich: Demonstration eines Falles von Chondrodystrophia foetalis. *Sitzungsbericht des Vereins der Aertze in Steiermark*, 1901.

398. *Bossi: Sopra un caso di acondroplasia vivante. *Archivio Italiano de ortopedia*, No. 3, p. 141, 1901. Also *Bollettino dell' Associaz. sanitaria Milanese*, Jan. 1901.

399. *Fochier: Squelette d'achondroplase. *Société de Chirurgie de Lyon*, Dec. 26, 1901.

400. *Simmonds: Untersuchungen von Missbildungen mit Hülfe des Röntgenstrahlen. *Fortschritte auf dem Gebiete der Röntgenstrahlen*, Bd. iv. Heft 4, 1901.

400[b]. Simmonds: Ueber die sogenannte foetale Rachitis. *Münchener Medizinische Wochenschrift*, 1901, Jahrgang 48, S. 1263—1264. München, 1901. [Some remarks on chondrodystrophia hypoplastica and chondrodystrophia hypertrophica. He showed pictures taken by the Röntgen rays and microphotographs. The two cases were published in *Fortschritte auf d. Gebiete d. Röntgenstrahlen*, Bibl. No. 400.]

401. von Hansemann: Echte Nasonomie, mit Demonstration eines Falles. *Berliner klinische Wochenschrift*, 1902, xxxix. Jahrgang, No. 52, S. 1209—1212. Berlin, 1902. [See Bibl. Nos. 277[b], 454 (Dobos Janos). Pedigree No. 734.]

402. Heatley, H. R.: *Life and love-letters of a dwarf, Joseph Boruwlaski.* London, 1902. [This work is apparently compiled from the original memoirs, published 1788. See Bibl. Nos. 43 and 47.]

403. Gilford, Hastings: Ateleiosis; a disease characterised by conspicuous delay of growth and development. Plates. *Medico-Chirurgical Transactions*, Vol. lxxxv. pp. 305—359. London, 1902. [Pedigrees Nos. 713, 714 and 718.]

404. Railton, T. C.: Sporadic Cretinism. *British Medical Journal*, 1902, Vol. i. pp. 694—695. London, 1902. [Pedigree No. 784.]

405. Ballantyne, J. W.: *Manual of Antenatal Pathology and Hygiene.* (The Foetus.) Chap. xix. pp. 338—340. Edinburgh, 1902. [Pedigree No. 638.]

406. Kassowitz, Max: Infantiles, Myxoedem, Mongolismus und Micromelie. Plates. *Wiener medicinische Wochenschrift*, 1902, lii. Jahrgang, S. 1049—1055, 1105—1112, 1155—1159, 1202—1205, 1256—1261, 1301—1306, 1357—1366, 1409—1415, 1452—1456. Wien, 1902. [Gives a large number of non-adult cases, but no family history.]

407. Escher, C.: Zur Frage der angeborenen Rachitis. *Jahrbuch für Kinderheilkunde*, Bd. liv., or 3 F., Bd. vi. pp. 613—638. Berlin, 1902. [A general discussion on congenital rachitis.]

408. Comby, Jules: Rickets and Achondroplasia. *British Medical Journal*, 1902, Vol. ii. pp. 955—956. London, 1902. [Pedigree No. 616: see also Bibl. Nos. 409 and 419.]

409. Comby, Jules: Un cas d'achondroplasie. *Bulletins et Mémoires de la Société médicale des Hôpitaux de Paris*, 3[e] Série, T. xix. pp. 551—552. Paris, 1902. [Pedigree No. 616: see Bibl. Nos. 408 and 419.]

410. Méry, H., et Labbé, R.: Sur un cas d'achondroplasie. *Bulletins et Mémoires de la Société médicale des Hôpitaux de Paris*, 3[e] Série, T. xix. pp. 543—551. Paris, 1902. [Pedigree No. 670.]

411. Regnault, Felix: L'Achondroplasie. Plates. *Archives générales de Médecine*, N. S., T. vii. pp. 232—255. Paris, 1902. [Gives a general account of achondroplasia and also of various pictures and statues of dwarfs.]

411[b]. *Joachimsthal: Die angeborenen Verbildungen der unteren Extremitäten. *Fortschritte auf dem Gebiete der Röntgenstrahlen*, Erg. Bd. viii. 1902.

412. Durante, G.: Contribution à l'étude de l'achondroplasie. *Revue médicale de la Suisse Romande*, T. xxii. pp. 809—826. Genève, 1902. [Pedigree No. 668.]

413. Apert, E.: Le myxoedème et l'achondroplasie sont deux affections totalement différentes. *Comptes Rendus de la Société de Biologie*, T. liv., or 11[e] Série, T. iv. pp. 127—129. Paris, 1902. [Discusses the differences of the two diseases.]

414. Lannois, M.: Deux cas de nanisme achondroplasique chez le frère et la sœur. *Lyon Médical*, T. xcviii. pp. 893—900. Lyon, 1902. [Pedigree No. 618.]

415. Christopher, W. S.: Development the key-note of pediatrics *Archives of Pediatrics*, Vol. xix. pp. 481—488. New York, 1902. [This is merely an address on pediatrics; there is nothing in it about dwarfs or achondroplasia. The reference is however frequently given in bibliographies; possibly it is intended to refer to the discussion, in which Christopher joined, on Morse's paper (Bibl. No. 416) in same volume.]

416. Morse, John Lovett: A case of Chondrodystrophy (*sic!*) Fetalis. *Archives of Pediatrics*, Vol. xix. pp. 561—577. New York, 1902. [In the discussion, Griffith mentions two cases of achondroplasia (p. 575), Holt gives one case (p. 576), and Christopher gives one (p. 577) which he does not think can be cretinism. There is a short bibliography. [Pedigrees Nos. 753 and 825.]

416[b]. Manouvrier, L.: Notes sur quelques prodiges humains exhibés à Paris en 1901. *Revue Mensuelle de l'École d'Anthropologie de Paris*, 12[e] année, pp. 11—19. Paris, 1902. [p. 19 gives an account of two dwarfs—(1) Pierre le Grand, height said to be 58 cm., but Manouvrier suggests he was probably a few centimetres taller and says he was a new edition of Bébé, as his head was in proportion to the size of his body; (2) Queen Mab, who was agreeable and intelligent. No height is given. These dwarfs were in Barnum and Bailey's Circus.]

417. Peloquin, A.: *De l'achondroplasie chez l'homme et les animaux*. Thèse. 78 pages. Lyon, 1902. [He gives Lannois' Case and one of his own, also a bibliography. Pedigree No. 623.]

418. Apert, E.: Pathogénie et traitement pathogénique des enfants retardataires. *Annales de Médecine et Chirurgie Infantiles*, T. vi. pp. 96—100. Paris, 1902.

419. Comby, Jules: Un cas d'achondroplasie. *Archives de Médecine des Enfants*, T. v. pp. 473—477. Paris, 1902. [Typical case in boy aged 5½, 85 cm. in stature, no rachitis. Good photographs; discussion of distinction between riekety and achondroplasic individuals, and rickets complicated with achondroplasia.]

420. Wood, A. Jeffreys, and Hewlett, Herbert M.: Three Cases of Achondroplasia. *Intercolonial Medical Journal of Australasia*, Vol. vii. pp. 385—394. Melbourne, 1902. [Pedigrees Nos. 641, 642 and 643.]

421. Leblanc, P.: Achondroplasie et myxoedème. *Comptes Rendus de la Société de Biologie*, T. liv., or 11[e] Série, T. iv. pp. 88—89. Paris, 1902. [Merely a note drawing attention to the existence of achondroplasia in animals and associating it with myxoedema.]

422. Legry, Th., et Regnault, Felix: Présence de corps thyroïdes normaux chez les achondroplasiques. *Comptes Rendus de la Société de Biologie*, T. liv., or 11[e] Série, T. iv. pp. 567—568. Paris, 1902. [A note stating the thyroid body was found normal in three achondroplasic foetuses.]

423. Regnault, Felix: Différenciation des squelettes de veaux achondroplasiques et natos. *Comptes Rendus de la Société de Biologie*, T. liv., or 11[e] Série, T. iv. pp. 1233—1235. Paris, 1902. [A note stating he had differentiated between calves of the achondroplasic type and bull-dog type, which had hitherto been confounded.]

424. Jaboulay, M.: Achondroplasie chez un adulte. *Lyon Médical*, T. xcviii. pp. 281—282. Lyon, 1902. [Account of an achondroplasic female. No family history. No measurements.]

425. Champetier de Ribes et Daniel, Constantin: Un cas d'achondroplasie. *Bulletins de la Société Anatomique de Paris*, 6[e] Série, T. iv. p. 90. Paris, 1902. [An achondroplasic foetus aged 7½ months. No family history. No measurements.]

426. Méry, H.: Sur un cas d'achondroplasie. Laryngite sous-glottique. *Lucas-Championnière's Journal de Médecine et de Chirurgie pratiques*, T. lxxiii. pp. 90—93. Paris, 1902. [Description of a boy, aged 8, height 96 cm., and brother of five normal children. No further family history.]

427. Ribbert, Hugo: Fötale Rachitis. *Lehrbuch der speciellen Pathologie und der speciellen pathologischen Anatomie*, pp. 684—685. Plate. Leipzig, 1902. [Short account of the features of the disease with plate of foetal skeleton.]

428. *Allaria, G. B.: *Rivista critica di Clinica medica*, No. 5. Firenze, 1902.

429. *Acquaderni, A.: Acondroplasia. *Rivista critica di clinica medica*, p. 327, 5 ap. 1902. Firenze, 1902.

429[b]. Swoboda, Norbert: Ein Fall von chondrodystrophischen Zwergwuchs (Achondroplasie). *Wiener klinische Wochenschrift*, xvi. Jahrgang, S. 669—671. Wien, 1903. [Pedigree No. 666.]

430. *Poncet, A.: *La Province médicale*, Jan. 23, 1902. Lyon, 1902.

431. *Lereboullet, P.: *Les Cirrhoses Biliaires*, p. 76. Paris, 1902.

432. Cantlie, James: On a case of achondroplasia. *The Polyclinic*, Vol. vi. pp. 120—124. London, 1902. [Description of an achondroplasic girl, with no measurements or family history. Photographs are those in Turner's Case, Bibl. No. 354, but there is no reference.]

433. *Vargas, M.: Die Achondroplasie. *Monatsschrift für Kinderheilkunde*, S. 67, Nov. 1902. [Case of achondroplasia or chondrodystrophy hypoplastic, complicated with pes varus; radiography showing absence of ossification of epiphyses; general discussion and bibliography.]

434. Taylor, J.: A case of Achondroplasia. *Reports of the Society for the Study of Disease in Children*, Vol. iii. p. 162. London, 1902—1903. [Pedigree No. 688.]

435. Sutherland, G. A.: Case of Infantilism in a boy aged 10½ years. *Reports of the Society for the Study of Disease in Children*, Vol. iii. pp. 192—194. London, 1902—3. [Pedigree No. 798.]

435b. *Gheorgio, N. Foetus achondroplasique présentant aux mains et aux pieds de la polydactylie: bec-de-lièvre. *Bulletins et mémoires de la Société de Chirurgie de Bucarest*, 1902—3, T. v pp. 13—16. Bucarest, 1902—3.

436. Bayon, P. G.: Beitrag zur Diagnose und Lehre von Cretinismus. *Verhandlungen der physikalisch-medicinischen Gesellschaft zu Würzburg*, Bd. xxxvi. Heft i. S. 46—50. Würzburg, 1903. [Pedigrees Nos. 751 and 782.]

437. Wood, A. Jeffreys, and Hewlett, Herbert M.: Achondroplasia. *Intercolonial Medical Journal of Australasia*, Vol. viii. pp. 17—18. Melbourne, 1903. [Pedigree No. 634.]

438. Variot, G.: Un cas d'achondroplasie anormale, sans dystrophie crânienne, chez une fille de treize ans. *Bulletins et Mémoires de la Société médicale des Hôpitaux de Paris*, 3e Série, T. xx. pp. 268—270. Paris, 1903. [Pedigree No. 637.]

439. *Variot, G.: *La Tribune médicale*, 2ième Série, T. xxxv. p. 229. Paris, 1903. [? = Bibl. No. 438.]

440. Variot, G.: Étude radiographique du squelette d'une fille de 13 ans atteinte d'une variété spéciale d'achondroplasie sans dystrophie crânienne. *Bulletins de la Société de Pédiatrie de Paris*, T. v. pp. 150—158. Paris, 1903. [Comparison of skeleton of girl with that of achondroplasic boy, whose radiographs were lent by Méry: see Bibl. No. 426.]

441. *Molodenkoff, S. S.: Achondroplasia. *J. nevropat. i psikhiat.*, iii. Supplement, Pt ii. pp. 28—31. Korsakova, Moskau, 1903.

441b. Manouvrier, L.: Deuxième examen, à 15 ans, d'un microcéphale observé à 7 ans. *Bulletins et Mémoires de la Société d'Anthropologie de Paris*, 5e Série, T. iv. pp. 591—594. Paris, 1903. [See Bibl. No. 302.]

442. Poncet, A., et Leriche, R.: Les nains d'aujourd'hui et les nains d'autrefois. Nanisme ancestral. Achondroplasie ethnique. *Bulletin de l'Académie de Médecine de Paris*, T. l. pp. 174—188. Paris, 1903. Also *Revue de Chirurgie*, T. xxiii. pp. 657—693. Paris, 1903. Also *Lyon Médical*, T. ci. pp. 609—622. Lyon, 1903. [Pedigree No. 623. Also described by Peloquin, Bibl. No. 417.]

443. Poncet, A., et Leriche, R.: Achondroplasie familiale. *Lyon Médical*, T. c. pp. 202—208, 326—329. Paris, 1903. [Pedigree No. 623: see also Bibl. No. 442.]

444. *Chavigny: Achondroplasie fruste. *Bulletins de la Société de Médecine des Hôpitaux de Lyon*, 1903, pp. 151 and 551. Lyon, 1903. [This reference is quoted by Emerson (Bibl. No. 604). According to him Chavigny had an achondroplasic patient who said he was the tallest of four siblings shaped like himself.]

445. *Heveroch, A.: Kasuistický príspevek. k. achondroplaisii. *Časopis lékarn Českych v. Praze*, xlii. pp. 656—661. Prag, 1903. [Statistical contribution to achondroplasia.]

446. *Gache, S.: Sur un cas d'achondroplasie. *Argentina Medica*, 17 Oct. 1903.

447. Poncet, A.: Sur l'achondroplasie. *Lyon Médical*, T. c. pp. 243—244. Lyon, 1903. [Some remarks on dwarf races.]

448. Herrman, Chas.: A case of Achondroplasia. (Micromelia.) *Archives of Pediatrics*, Vol. xx. pp. 18—25 and 60. New York, 1903. [Pedigree No. 675.]

449. Michel, F.: Osteogenesis Imperfecta. *Virchow's Archiv*, Bd. clxxiii. S. 1—35. Berlin, 1903. [Pedigree No. 758.]

450. *Pelnar, J.: Achondroplasie u 55 leteho muže nález pitevni. [Achondroplasia in a man of 55 years; anatomical findings.] *Časopis lékarn Českych v. Praze*, 1903, xlii. pp. 651—661. And Nový přepad achondroplasie ve věku 56 tet. [A new case of achondroplasia at the age of 56 years.] *Ibid.* pp. 661—663.

451. Foerster, R.: Contribution à la pathologie de la Lecture et de l'Écriture. (Observation chez un achondroplasique.) *Revue Neurologique*, T. xi. pp. 1206—1208. Paris, 1903. [An account of the reading and writing powers of Claudius, the achondroplasic dwarf described by Marie. See Bibl. No. 371.]

451b. Kienböck, Robert: Zur radiographischen Anatomie und Klinik der chondralen Dysplasie der Knochen mit multiplen cartilaginären Exostosen. *Wiener Medizinische Wochenschrift*, 1903, Jahrgang 53, pp. 2202—2206, 2274—2279, 2316—2322, 2369—2377, 2411—2423, 2455—2463. Wien, 1903. [Several observations are given. p. 2316 gives the pedigree of a family, several members of which had exostoses, and three members appear to have been so deformed by the disease as to be practically dwarfs.]

452. Silberstein, Adolf: Ein Beitrag zur Lehre von den foetalen Knochenerkrankungen. *Langenbeck's Archiv für klinische Chirurgie*, Bd. lxx. S. 863—875. Berlin, 1903. [He divides foetal bone disease into four divisions: (1) True rachitis. (2) Syphilitic bone disease. (3) Osteogenesis imperfecta. (4) Chondrodystrophia.]

453. West, J. Park, and Piper, W. O. S.: A case of Chondrodystrophy (*sic!*) Foetalis. *Archives of Pediatrics*, Vol. xx. pp. 730—734. New York, 1903. [Pedigree No. 778.]

454. Lardennois: Étude d'un type de nain véritable. *Union médicale du Nord-Est*, T. xxvii. pp. 121—123. Reims, 1903. [Account of a Hungarian ateleiotic dwarf, Dobos Janos: see Pedigree No. 734: see also Bibl. Nos. 277b, 401.]

455. *Devay: Un cas d'achondroplasie. *Soc. Nat. de Méd. de Lyon*, fév. 9, 1903.

456. Daniel, Constantin: De l'achondroplasie chez le foetus. *Annales de Gynécologie et d'Obstétrique*, T. lix. pp. 25—49. Paris, 1904. [Pedigree No. 662.]

457. Comby, Jules: Présentation de radiographies d'achondroplasiques. *Bulletins de la Société de Pédiatrie de Paris*, mai, 1903, pp. 173—174. Paris, 1903. [Account of the radiographs of two achondroplasic boys, one aged $5\frac{1}{2}$ years and the other 14 months. See Bibl. No. 419.]

458. Bramwell, Byrom: Case of Infantilism. *Clinical Studies*, Vol. i. Pt ii. pp. 157—163. Edinburgh, 1903 (printed 1902). [Account of effect of the administration of pancreatic extract on a boy aged $18\frac{10}{12}$ years, with chronic diarrhoea of 9 years' standing and complete arrest of bodily development.]

459. *Pauly: Main en Trident. *Société médicale de Lyon*, 9 Mar. 1903.

459b. Dunlop, G. H. Melville: Case of Achondroplasia. *The Lancet*, Vol. ii. p. 32. London, 1903. Also *Transactions of the Medico-Chirurgical Society of Edinburgh*, N.S. Vol. 22, pp. 286—287. Edinburgh, 1903. [A girl aged 12, height 46 inches, who manifested the main and most striking characteristics of achondroplasia, but the arms were not typical, they were not shortened.]

460. *Morlat: *Infantilisme et insuffisance surrénale.* Thèse. Paris, 1904.

461. *Zander, R.: Riesen und Zwerge. *Naturw. Wochenschrift*, N. F. Bd. iii. S. 385—390. Jena, 1903—1904.

462. Article: Une Naine: Mort d'un phénomène. *Le Progrès médical*, 3e Série, T. xix. p. 63. Paris, 1904. [A notice quoted from *Le Petit Var*, 16 Jan. 1904, of Maria Schumann, who died, aged 28, at Stockmar in Bavaria. She had passed her whole life in the cradle of her infancy, and till death had the appearance and figure of a child of a few months old. Her intelligence had developed normally, and she conversed with vivacity and esprit like an adult. On p. 79, under the heading "Un Nain," is a notice quoted from *La République des Pyrenées-Orientales* du 24 janvier of a conscript 126 cm. in height who weighed 26 kilos in his clothes.]

463. Bourneville et Lemaire: De quelques formes de nanisme et de leur traitement par la glande thyroïde. *Le Progrès médical*, 3e Série, T. xix. pp. 385—388, 403—405, also T. xx. pp. 402—408. Paris, 1904. [Gives an account of the effect of thyroid treatment on several dwarfs. Some family details are given in three cases, two of which however seem rather too tall to be considered dwarfs. Pedigrees Nos. 803 and 804.]

464. Galliard, L., et Levy, F.: Micromélie avec malformation symétrique des radius. *Bulletins de la Société médicale des Hôpitaux de Paris*, 3e Série, T. xxi. pp. 1103—1106. Paris, 1904. [Account of a woman aged 28, height 143 cm., with very short limbs; her hands were not trident-shaped. It was doubtful whether it was a case of achondroplasia or not. No family history.]

465. Balme, Harold, and Reid, Archibald D.: Notes on achondroplasia (Chondrodystrophia foetalis) with particulars and skiagrams of a case of the disease. *The Practitioner*, Vol. lxxiii. pp. 780—793. London, 1904. Also *Med. Electrol. and Radiol.* Vol. vi. pp. 3—15. London, 1905. [Pedigree No. 626.]

466. Meynier, Emilio: Contributo allo studio dell' acondroplasia. *Archivio per le scienze mediche*, T. xxviii. Fasc. iv. pp. 461—497. Torino, 1904. [Pedigrees Nos. 631 and 654.]

467. Dide, Maurice, et Leborgne: Nouveau cas d'achondroplasie. *Nouvelle Iconographie de la Salpêtrière*, T. xvii. pp. 200—202. Paris, 1904. [Pedigree No. 645.]

468. Cooke, Joseph Brown: Chondrodystrophia foetalis. Plates. *The American Journal of Obstetrics*, Vol. l. pp. 808—813. New York, 1904. [Case of a male infant, the first child of healthy parents whose family history was negative.]

469. Leriche, R.: De l'achondroplasie chez l'adulte. Historique. Symptomatologie. Étude anatomique. Pathogénie. *Gazette des Hôpitaux*, 1904, 77^{e} année, pp. 195—201, 227—232. Paris, 1904. [He gives a bibliography.]

469^{b}. Apert, E. Myxoedème fruste, croissance tardive, diabète. *Nouvelle Iconographie de la Salpêtrière*, T. xvii. pp. 1—8. Paris, 1904. Plate. [A man aged 66, height 145 cm., who did not stop growing till he was aged 30. He was obese and had cryptorchism. His limbs were short and hand trident-shaped, but he was not achondroplasic. He was the last of five children; he thought his parents, brother and sisters were all normal. His intelligence was normal.]

470. Pernet, George: The antiquity of Achondroplasia. *British Journal of the Diseases of Children*, Vol. i. pp. 7—10. London, 1904. [This paper is chiefly about the small Egyptian glazed earthenware statuettes in the British Museum.]

470^{b}. Seligmann, C. G.: Congenital Cretinism in Calves (with plates of bones, skulls, etc.). *Transactions, Pathological Society of London*, Vol. 55, pp. 1—20. London, 1904.

471. Treub, H.: Un cas d'achondroplasie. Présentation de photographies et de radiographies. *Bulletin de la Société d'Obstétrique de Paris*, T. vii. pp. 58—66. Paris, 1904. [Pedigree No. 661.]

471^{b}. *Bruneau de Laborie: *Du nanisme mitral.* Paris, 1904.

472. Lequeux, P.: Un foetus achondroplasique. Présentation de la pièce anatomique, de radiographies et de coupes histologiques. *Bulletin de la Société d'Obstétrique de Paris*, T. vii. pp. 150—153. Paris, 1904. [Pedigree No. 672.]

472^{b}. *von Mettheimer, A.: Mikromelie bei einem 7-jährigen Mädchen. iii. *Versammlung der Vereinigung süd-deutscher Kinderärzte*, 11 Dez. 1904. Frankfurt am M. 1904.

473. *Muggia, A.: Su un caso di acondroplasia. *La Pediatria*, 2. Ser., T. ii. pp. 260—276. Napoli, 1904. [Girl of 12 years, height 103 cm., micromelia, *mains en trident*, parents healthy, thyroid treatment no effect; normal intelligence.]

474. Lepage, G.: Opération césarienne chez une primipare achondroplasique. Enfant vivant présentant des déformations achondroplasiques. Plates. *Comptes Rendus de la Société d'Obstétrique, de Gynécologie et de Pédiatrie*, T. vi. pp. 270—278. Paris, 1904. [Pedigree No. 614. In the discussion which followed Lepage's paper Potocki mentioned Guéniot's Case (Bibl. No. 289), at which he had assisted, and gave some later details of the family. Pedigree No. 613.]

475. Nathan, P. W.: Chondrodystrophia foetalis. Plate. *American Journal of Medical Sciences*, Vol. cxxvii. pp. 690—702. Philadelphia and New York, 1904. [A general paper on the subject read before the Orthopaedic Section of the New York Academy of Medicine, Oct. 16, 1903.]

475^{b}. Comby, J.: Nouveaux cas d'achondroplasie. *Archives de Médecine des Enfants*, T. vii. pp. 541—547. Paris, 1904. [The first of the three cases is that of Bibl. No. 419; the second of a female child of 14 months, with good photograph and excellent radiograph of hands (*mains en trident* and absence of cartilaginous ossification); the third a boy of $4\frac{1}{2}$ years, height 84 cm., parents and two siblings normal and well proportioned. The author draws attention to the two classes of achondroplasia of Kaufmann, *forme hypoplastique*, for those cases which indicate an arrest of cartilaginous ossification, and *forme hyperplastique* for those which show a too precipitated ossification: see above, p. 372.]

476. Matsuoka, M.: Beitrag zur Lehre von der fötalen Knochenerkrankung. Plate. *Deutsche Zeitschrift für Chirurgie*, Bd. lxxii. S. 428—444. Leipzig, 1904. [A general discussion of the subject.]

476^{b}. *Breus und Kolisko: Die pathologischen Beckenformen. Wien und Leipzig, 1904.

477. Bayon, P. G.: Ueber angebliche verfrühte Synostose bei Kretinen und die hypothetischen Beziehungen der Chondrodystrophia Foetalis zur Athyreosis. *Ziegler's Beiträge zur pathologischen Anatomie, und zur allgemeinen Pathologie*, Bd. xxxvi. S. 119—130. Jena, 1904. [A discussion on the differences between cretinism and chondrodystrophia foetalis, criticizing Virchow's paper (Bibl. No. 114).]

478. Leriche, R.: Nanisme simple ou essentiel. *Gazette des Hôpitaux*, 1904, 77e année, pp. 1041—1046. Paris, 1904. [Account of a true dwarf, a male aged 21, height 135 cm. The measurements are given but no family history.]

479. *Bouchacourt, L.: Radiographies de 5 foetus achondroplasiques. *Bulletin de la Société d'Obstétrique de Paris*, T. vii. p. 58. Paris, 1904.

479b. Weigandt, W.: Der heutige Stand der Lehre von Kretinismus. *Sammlung zwangloser Abhandlungen aus dem Gebiete der Nerven und Geistes Krankheiten*, Bd. iv. Heft 6—7. Halle, 1904. [Discussion on cretinism and infantile myxoedema, with exception of S. 65, photograph and account of a female rachitic dwarf, height 101 cm., and S. 66, photograph and account of an achondroplasic male dwarf, height 123·2 cm. No family history. Photographs of cretins.]

480. *Lévi, L., et Bouchacourt, L.: Radiographies de foetus achondroplases. *Revue d'hygiène et de médecine infantile*, T. iii. pp. 514—528. Paris, 1904.

480b. Thomson, John: Two Cases of Infantilism. *Transactions of the Medico-Chirurgical Society of Edinburgh*, N.S. Vol. 23, pp. 165—166. Edinburgh, 1904. [These cases were diagnosed as cases of pancreatic infantilism. (1) Youth aged $24\frac{8}{12}$ years, height $51\frac{1}{2}$ in. He was very intelligent. The genital organs were infantile. He was first seen by Thomson in 1894 and under treatment he grew $2\frac{1}{2}$ in. between 15 and $17\frac{1}{2}$ years of age, 3 in. in the next 5 years and since then had not grown $\frac{1}{4}$ in. (2) Youth aged $17\frac{9}{12}$, height $49\frac{1}{2}$ in., with infantile genitalia; he had just been placed under Byrom Bramwell for treatment.]

481. Bramwell, Byrom: Pancreatic Infantilism; remarkable improvement (growth of body and sexual development) as the result of administration of pancreatic extract. *Clinical Studies*, Vol. ii. pp. 346—348. Edinburgh, 1904. *Scottish Medical and Surgical Journal*, Vol. xiv. pp. 321—324. Edinburgh, 1904. *Clinical Studies*, N. S. Vol. iii. pp. 172—174, 1905.

481b. Bramwell, Byrom: Achondroplasia. *Clinical Studies*, Vol. ii. pp. 346—348. Edinburgh, 1904. [An account of Thomson's Case, Bibl. No. 367, with some measurements.]

482. Tollemer: Nanisme pseudo-myxoedème. *Le Progrès médical*, 3e Série, T. xxi. p. 433. Paris, 1905. [A dwarf child, aged 7, with normal brothers and sisters. Its intellectual faculties were normal. No thyroid gland could be felt. No family antecedents.]

483. Bramwell, Byrom: Achondroplasia. *Clinical Studies*, N. S. Vol. iii. Pt ii. pp. 174—175. Edinburgh, 1905. [Pedigree No. 680.]

484. Miller, D. J. M.: Chondrodystrophy (*sic!*) foetalis (Achondroplasia). *American Journal of Medical Sciences*, Vol. v. 130, N. S., pp. 30—36. Philadelphia—New York, 1905. [Pedigree No. 644.]

485. Marconi, Egidio: Acondroplasia fetale e speciali alterazioni placentari. *Annali di Ostetriccia e Ginecologia*, T. xxvii. pp. 634—640. Milano, 1905. [Pedigree No. 651.]

486. Horand, Réné: Chondrodystrophie ou achondroplasie et nanisme dysthroïdien myxoedémateux. *Lyon Médical*, T. civ. pp. 926—933. Lyon, 1905. [Pedigrees Nos. 655 and 838.]

487. Heiman, Henry: A case of achondroplasia. *Archives of Pediatrics*, Vol. xxii. pp. 842—846. New York, 1905. [Pedigree No. 754.]

488. Parhon, C., Shunda, Atil., et Zalplachta, Z.: Sur deux cas d'achondroplasie. *Nouvelle Iconographie de la Salpêtrière*, T. xviii. pp. 539—559. Paris, 1905. [Pedigree No. 656.]

489. *Porak, C., et Durante, G.: Les dystrophies osseuses congénitales. *Rapport de la Séance annuelle de la Société Obstétricale de France*, avril, 1905. [See No. 491.]

490. Durante, G.: Nains achondroplasiques et nains rachitiques. *Académie de Médecine*, 1905; *La Presse médicale*, 13 année, p. 280. Paris, 1905. [Short account of Durante's paper at Académie de Médecine, mai 2, 1905, on difference of two kinds of dwarfs. See Bibl. No. 491.]

491. Porak, C., et Durante, G.: Les micromélies congénitales. Achondroplasie vraie et dystrophie périostale. Plates. *Nouvelle Iconographie de la Salpêtrière*, T. xviii. pp. 481—539. Paris, 1905. [A general account of achondroplasia with bibliography. See also Bibl. No. 490.]

491b. Porak, C. Présentation de deux squelettes de naines provenant de la Maternité. Société obstétricale de France, 27—29 avril, 1905. *La Presse médicale*, 1905, 13e année, p. 279. Paris, 1905. [A notice of two dwarf skeletons presented at the séance of the Société Obstétricale de France. The one was achondroplasic, the other rachitic.]

491c. Durante, G.: Nouveaux faits d'achondroplasie. *La Presse médicale*, 1905, 13e année, p. 279. Paris, 1905. [A short note on a paper read before the Société obstétricale de France. Attention is drawn to the fact that many different affections are classed under achondroplasia merely because of the shortness of the extremities.]

492. Sevestre: Sur un cas de l'achondroplasie. *Bulletins de l'Académie de Médecine*, 3e Série, T. liii. pp. 574—577. Paris, 1905. Also *La Presse médicale*, 1905, 13e année, p. 360. Paris, 1905. [He examined another member of the family in Pedigree No. 612.]

493. Launois, P. E., et Apert, E.: Achondroplasie héréditaire. *Bulletins et Mémoires de la Société médicale des Hôpitaux de Paris*, 3e Série, T. xxii. pp. 606—613. Paris, 1905. [Pedigree No. 615.]

493b. *Launois, P. E., et Apert, E.: L'hérédité de l'achondroplasie chez l'homme et chez les animaux. *Bulletins de la Société de Pathologie comparée*, T. xii. p. 19. Paris, 1905. [This gives an account of case described in Bibl. No. 493.]

494. Lepage, G.: Un cas d'achondroplasie chez un foetus extrait par opération césarienne chez une femme achondroplasique. *Revue d'Orthopédie*, 2e Série, T. vi. pp. 109—118. Paris, 1905. [Pedigree No. 614: see Bibl. No. 474.]

495. *Michael, May: A case of achondroplasia. *Women's Medical Journal*, Vol. xv. p. 75. Toledo, U.S.A., 1905.

496. Nobécourt et Paisseau: Un cas d'achondroplasie fruste. *Annales de Médecine et Chirurgie infantiles*, T. ix. pp. 413—418. Paris, 1905. [Gives an account of a girl aged 12, height 1288 mm. The cranial portion of her head was large, the face small. The peculiarity of the patient consisted in the fact that the arms were normal in dimensions while the legs were short, and, though not curved, exhibited an appreciable degree of genu valgum.]

497. *Parache: Un caso de acondroplasia. *Revista de medicina y cirurgia practicas (nacional y extrangera)*, T. lxvi. pp. 281—283. Madrid, 1905.

497b. *Lugano: Sul cretinismo sporadico. *Rivista di Pat. nerv. e ment.*, 1905.

498. Thibault, A.: Note sur un cas d'achondroplasie. *Angers Médical*, T. xii. pp. 6—10. Anjou, 1905. [Eugenie R., aged 40 years, normal parents, 10 siblings all dead; mother attributed dwarfism to maternal impression, having frequently seen, during her training as *sage femme* at Angers, the picture of a dwarf in the lecture-room. Height 132 cm., trunk average size. E. R. at 32 became pregnant, and child delivered by Caesarian operation died at four days.]

499. *Variot, G.: Achondroplasie, son traitement. *Journal de Médecine interne*, T. ix. p. 109. Paris, 1905.

500. *Variot, G.: L'achondroplasie. *Revue générale de clinique et de thérapeutique*, T. xix. p. 487. Paris, 1905.

500b. *Bernstein, S. L.: Achondroplasia. *Cleveland Medical Journal*, Vol. vii. pp. 12—18. Cleveland, U.S.A., 1905.

501. Joseph, H. M.: Chondrodystrophia foetalis or Achondroplasia. *The Lancet*, 1905, Vol. ii. p. 217. London, 1905. [Measurements and description of a girl, aged 3 years and 11 months, height 75 cm. No family history.]

502. Chavigny: Achondroplasie partielle. *Lyon Médical*, T. civ. pp. 1252—1253. Lyon, 1905. Also **Bulletins de la Société Médicale des Hôpitaux de Lyon*, T. iv. p. 228. Lyon, 1905. [Description of a soldier with hands of the achondroplasic type.]

503. Salomon, P.: Description d'un foetus achondroplasique. *Bulletins et Mémoires de la Société d'Anthropologie de Paris*, 5e Série, T. vi. pp. 303—308. Paris, 1905. [The subject of this observation was a still-born male child preserved since 1864 in the Dareste Collection, and known as "Le Phocomèle."]

504. Sinnetamby, M.: A case of Achondroplasia in which Caesarian section was successfully performed. *Journal of the Ceylon Branch of the British Medical Association*, Vol. ii. Pt ii. pp. 72—75. Colombo, 1905. [Pedigree No. 679.]

505. Nau: Le rachitisme congénital. **Rapport de la Séance annuelle de la Société obstétricale de France. La Presse médicale*, 1905, 13e année, p. 279. Paris, 1905. [Notice of Nau's paper. He stated that the study of intra-uterine rachitis was based on those cases which are not classed under achondroplasia.]

506. Verneau, B.: Les pygmées et les nains achondroplasiques. **Rapport de la Séance annuelle de la Société obstétricale de France*, 1905. *La Presse médicale*, 1905, 13e année, p. 280. Paris, 1905. [Notice of Verneau's paper in which he discusses whether the pygmy races are achondroplasic or not and denies the heredity of achondroplasia.]

507. *Porak, C.: Pseudo-achondroplasie: opération de Gigli; mère et enfant bien portants. Plate. *Bulletin de la Société d'Obstétrique de Paris*, T. viii. pp. 113—115. Paris, 1905.

507b. *Rondeau: Les rapports du rachitisme congénital et de l'achondroplasie. Thèse. Paris, 1905.

508. Fuchs, Emil: Vier Fälle von Myxödem nebst Beitragen zur skiagraphischen Differential-diagnose der verschiedenen Formen verzogerten Langenwachstums. *Archiv für Kinderheilkunde*, Bd. xli. S. 60—81. Stuttgart, 1905. [Title indicates subject.]

509. Smith, G. Elliot: Notes on African Pygmies. *The Lancet*, 1905, Vol. ii. pp. 425—431. London, 1905. [An account with measurements (some clearly erroneous) of six pygmies brought to Cairo by Colonel Harrison from the Ituri Forest.]

510. Veron: Un nouveau-né pseudo-achondroplasique rachitique (avec examen histologique). **Obstétrique*, T. x. pp. 235—242. Paris, 1905. *La Presse médicale*, 1905, 13e année, p. 279. Paris, 1905. [A notice of Veron's paper. His case was a child whose mother had slight traces of rachitis. Length 43 cm. Weight 2500 grammes. It had short thick-set limbs, and the radiographs showed short thick incurved bones.]

511. Fuchs, Emil: Ein Beitrag zur Kasuistik der Mikromelie mit drei Abbildungen. *Archiv für Kinderheilkunde*, Bd. xli. S. 380—383. Stuttgart, 1905. [Description of a man, aged 26, height 127 cm., with very short limbs.]

512. Durante, G.: Achondroplasie et rachitisme. *La Semaine médicale*, 1905, 25e Année, p. 213. Paris, 1905. [A short notice of a paper by Durante on achondroplasia and rachitis. See Bibl. No. 490.]

513. Calwell, Wm.: Observations on Dwarfism and Infantilism. *British Medical Journal*, 1905, Vol. i. pp. 1376—1378. London, 1905. [He showed two cases and a skeleton. (1) Male aged 21, height 4 ft. 4½ in., weight 7 st. 7½ lbs. He had no hair or only slight traces of it on face, axillae or pubis. Hair of head was turning grey. The relative length of the limbs to the body was about normal. Calwell calls it a case of infantilism, ateleiosis or arrested development with signs of progeria. (2) Female aged 26, height 3 ft. 8 in., weight 3 st. 10 lbs. She had excessive scoliosis with some lordosis. The upper limbs were as long as those of a woman of normal height, the lower limbs much bent. She was a dwarf from deformity. (3) Skeleton of the achondroplasic type. Illustrations are given.]

513b. Hektoen, Ludwig: Body of a dwarf with short limbs. *Transactions of the Chicago Pathological Society*, 9 April, 1906, p. 443. Reference in *Revue Neurologique*, T. 14, p. 949. Paris, 1906. [Morphological description of the corpse of an achondroplasic actor and singer who died of pneumonia.]

514. Thomson, John: Achondroplasia. *Green's Encyclopaedia and Dictionary of Medicine and Surgery*, Vol. i. pp. 38—40. London, 1906. [Article on the subject.]

515. Decroly, M. O.: Cas d'achondroplasie héréditaire et familiale. *Société Royale des Sciences Médicales et Naturelles de Bruxelles. Bulletin des Séances*, 64e Année, pp. 2—27. Bruxelles, 1906. [Pedigree No. 622.]

516. Herrgott, Alphonse: Du nanisme au point de vue obstétrical. Achondroplasie familiale, opérations césariennes. *Annales de Gynécologie et d'Obstétrique*, 2e Série, T. iii. pp. 1—18. Paris, 1906. [Pedigree No. 625.]

517. Auché: Achondroplasie chez un enfant de trois ans. *Gazette hebdomadaire des Sciences médicales de Bordeaux*, T. xxvii. pp. 116—117. Bordeaux, 1906. Also *Journal de Médecine de Bordeaux*, 36e année, p. 67. Bordeaux, 1906. [Pedigrees Nos. 649 and 755.]

517b. Curtis, M., et Salmon, J.: Un nouveau cas de Phokomélie avec étude histologique du système osseux. *Comptes Rendus de la Société de Biologie*, T. lx. année 1906, 1er Semestre, pp. 1058—60. [Title describes subject.] Paris, 1906.

518. Rankin, Guthrie, and Mackay, Ernest C.: Achondroplasia. *Medico-Chirurgical Transactions*, Vol. 89, pp. 395—418. London, 1906. *Pediatrics*, Vol. 19, pp. 77—88. New York, 1907. *British Medical Journal*, 1906, Vol. i. pp. 1518—1522. London, 1906. [Pedigree No. 658.]

519. Dufour, Henri: Achondroplasie partielle, forme atypique. *Nouvelle Iconographie de la Salpêtrière*, T. xix. pp. 133—135. Paris, 1906. [Pedigree No. 752.]

520. Haushalter: Un cas de nanisme achondroplasique. *Comptes Rendus de la Société de Biologie*, T. lx. p. 1079. Paris, 1906. [A short description of an achondroplasic girl, aged 19 months. No measurements and no family history.]

521. Emanuel: Achondroplasia. *British Medical Journal*, 1906, Vol. ii. p. 1305. London, 1906. [Description of a boy, aged 8, without measurements or family history.]

522. *Poujol, J.: Sur un cas de nanisme (rachitisme et achondroplasie) chez un Musulman algérien. *Bulletin médical de l'Algérie*, T. xvii. pp. 37—41. Alger, 1906.

522b. *Bergrath: Ueber Chondrodystrophia foetalis. Inaug. Diss. Bonn, 1906.

523. Bircher: Zwei Fälle von Chondrodystrophie. *Correspondenz-Blatt für Schweizer Aerzte*, Bd. xxxvi. S. 467. Basel, 1906. [Description of a male aged 16, and a female aged 24, without measurements or family history.]

524. Le Lorier: Un foetus achondroplasique [Rapport de P. Rudaux]. *Comptes Rendus de la Société d'Obstétrique, de Gynécologie et de Pédiatrie de Paris*, T. viii. pp. 127—128. Paris, 1906. After reporting Le Lorier's Case Rudaux gave a case of his own. [Pedigrees Nos. 671 and 676.]

525. Bonnet-Laborderie et Gorisse: Note sur un cas d'achondroplasie observé chez un nouveau-né. *Journal des Sciences Médicales de Lille*, 1906, T. i. pp. 25—32 and p. 278. Lille, 1906. [A female infant, second child of an apparently healthy mother aged 39; the father was aged 60. The mother insisted that she had been greatly frightened by a drunken man in the 4th month of her pregnancy. The child was a typical achondroplasic with micromelia of the rhyzomelic type. The total length was 44 cm. She had clubfoot (varus equinus). The authors discuss the probable causes of achondroplasia. p. 278 gives a note by M. Duret on the above case. He thinks that the age of the parents in this case played a part in the etiology of the disease.]

526. Keyser, C. R.: Achondroplasia; its occurrence in men and animals. *The Lancet*, 1906, Vol. i. pp. 1598—1602. London, 1906. [Gives some particulars of 34 collected cases of achondroplasia. In one of his own cases the father was only 4 ft. 8 in. tall, the mother normal. Pedigree No. 667.]

527. Poynton, F. J.: Achondroplasia. *Transactions of the Medical Society*, Vol. xxix. pp. 431—432. London, 1906. [Pedigree No. 663.]

528. Parhon, C., und Marbe, S.: Die Achondroplasie (mit zwei neueren Beobachtungen von Achondroplasie beim Erwachsenen. **Revista Stüntelor medicale*, No. 7, 1906. Reported in *Münchener medizinische Wochenschrift*, 1906. Jahrgang liii. No. 31, p. 1540. München, 1906. [A notice of the article in the above Roumanian journal in which the authors contend that dwarf growth depends on a disturbance of the functions of the glands associated with internal secretions.]

529. Dieterle, Theophil: Die Athyreosis unter besonderer Berücksichtigung der dabei auftretenden Skelettveränderungen, sowie der differential-diagnostisch vornehmlich in Betracht kommenden Störungen des Knochenwachstums. Untersuchungen über Thyreoaplasie, Chondrodystrophia foetalis und Osteogenesis Imperfecta. *Virchow's Archiv*, Bd. 184, S. 56—122. Berlin, 1906. 5 Textfiguren und Tafeln ii, iii, iv. [In two chapters. I. Die Athyreosis (kongenitales Myxoedem). II. Die foetalen Skeletterkrankungen. This chapter is on Chondrodystrophia foetalis and Osteogenesis Imperfecta. He concludes that no form of diseases of the skeleton can be attributed (kann zurückgefuhrt werden) to disturbance of the functions of the thyroid body.]

530. Hay, Kenneth R.: A Case of Achondroplasia. *Reports of the Society for the Study of Disease in Children*, Vol. vi. pp. 197—198. London, 1906. [A boy aged 2, his body was long relatively to the limbs. The humerus and ulna each measured $3\frac{3}{4}$ inches, the femur measured $4\frac{3}{8}$ inches. The father was phthisical and subject to fits, the mother was not robust. There was a younger child aged 4 months who was apparently normal.]

531. *Cerletti: Effetti delle iniezioni del succo d' ipofisi sull' accrescimento somatico. *Reale Accademia dei Lincei Roma*: 15 luglio e 5 agosto. Roma, 1906.

532. Porter, J. Houston: Achondroplasia. Notes of three Cases. *British Medical Journal*, 1907, Vol. i. pp. 12—14. London, 1907. [Pedigree No. 619.]

533. Litchfield, W. F.: A case of Achondroplasia. *Australasian Medical Gazette*, 1907, Vol. xxvi. pp. 624—625. Sydney, 1907. [Pedigree No. 624.]

534. Poynton, F. J.: Achondroplasia. *Allbutt and Rolleston's System of Medicine*, 3rd edition, Vol. iii. pp. 117—123. London, 1907. [An article on achoudroplasia with bibliography.]

535. Rankin, G., Mackay, E. C., Lunn, J. R., and Cranke, J.: Achondroplasia, with notes of cases. Plates. *British Medical Journal*, 1907, Vol. i. pp. 11—12. London, 1907. [Gives descriptions of three cases; one described under Cranke's name, one under Rankin and Mackay's, previously described (Bibl. No. 518), and the third Lunn's Case. A man aged 53, height not given, no family history. Probably this last is same case as in Bibl. No. 536. Pedigree No. 681.]

536. Lunn, John R.: Achondroplasia. *Transactions of the Clinical Society*, Vol. xl. pp. 252—253. London, 1907. [Pedigree No. 665.]

537 Macewen, J. C.: A case of Achondroplasia. *British Medical Journal*, 1907, Vol. ii. pp. 1646—1647. London, 1907. [Pedigree No. 657.]

538. Schmolck: Mehrfacher Zwergwuchs in verwandten Familien eines Hochgebirgtales. *Virchow's Archiv*, Bd. clxxxvii. S. 105—110. Berlin, 1907. [Pedigree No. 689.]

539. Langenbach, E.: Ein Fall von Chondrodystrophia foetalis mit Asymmetrie des Schädels. Plates. *Virchow's Archiv*, Bd. clxxxix. S. 12—17. Berlin, 1907. [Description of a skeleton, length 47 cm. No family history.]

540. Variot, G.: Analogie des troubles de l'ossification dans le myxoedème et dans l'achondroplasie. *Bulletins et Mémoires de la Société médicale des Hôpitaux de Paris*, 3e Série, T. xxiv. pp. 59—66. Paris, 1907. [See also No. 542.]

541. Brissaud, E., et Bauer: Un cas d'infantilisme "réversif" avec autopsie. *Bulletins et Mémoires de la Société médicale des Hôpitaux de Paris*, 3e Série, T. xxiv. pp. 39—41. Paris, 1907. [Case of a female, who reached adult age normally, and some time after pregnancy signs of infantilism became pronounced. The autopsy showed a small thyroid body and small genital organs. Nothing is stated as to stature.]

542. Variot, G.: Note sur les troubles de l'ossification dans l'achondroplasie étudiés par la radiographie. *Bulletins et Mémoires de la Société médicale des Hôpitaux de Paris*, 3e Série, T. xxiv. pp. 128—129. Paris, 1907. [See also No. 540.]

543. Voisin, Jules, et Voisin, Roger: Troubles de l'ossification dans le myxoedème et l'achondroplasie. *Bulletins et Mémoires de la Société médicale des Hôpitaux de Paris*, 3e Série, T. xxiv. pp. 73—76. Paris, 1907. [Title describes subject.]

544. Broca, A., et Debat-Ponsan, J.: Un cas d'achondroplasie. Plate. *Bulletins de la Société de Pédiatrie de Paris*, T. ix. pp. 91—94. Paris, 1907. [Girl aged 8, parents and sister normal, height 90·6 cm. She appeared normal at birth, growth stopped at age of 3, then from Jan. 1906 to Feb. 1907, she grew 10 cm. She was a typical case of achondroplasia.]

545. Reyher, P.: Zur Kenntniss der Chondrodystrophia foetalis mit 4 Abbildungen. *Charité Annalen*, Jahrgang xxxi. S. 129—145. Berlin, 1907. Also *Berliner klinische Wochenschrift*, Jahrgang xliv. S. 1423. Berlin, 1907. [Pedigrees Nos. 774 and 776.]

546. Freiberg, Albert H.: Defect of both femoral heads in a chondrodystrophic dwarf. *The American Journal of Orthopedic Surgery*, Vol. iv. pp. 184—189. Philadelphia, 1906—1907. [A girl aged 15, first child of her mother, height 48 inches. The trunk appeared disproportionately large for the arms and legs. Skiagrams showed the absence of the femoral head. There is a full account of the deformity and a discussion as to whether the case is one of chondrodystrophy or not.]

547. Meige, Henry, et Feindel, E.: Achondroplasie de l'adulte. *Pratique Médico-Chirurgicale*, T. i. pp. 52—55. Paris, 1907. [A short article on the main features of achondroplasia.]

548. Bouffe de Sainte-Blaise: Nouveau-né (Pathologie). Rachitisme intra-utérin. Achondroplasie ou rachitisme foetal. *Pratique Médico-Chirurgicale*, T. iv. p. 604. Paris, 1907. [A short article on intra-uterine rachitis.]

549. Meige, Henry: Nanisme. *Pratique Médico-Chirurgicale*, T. iv. pp. 457—458. Paris, 1907. [A short article on the different kinds of dwarfism.]

550. Feindel, E.: Infantilisme. *Pratique Médico-Chirurgicale*, T. iii. pp. 869—876. Paris, 1907. [Article on the different kinds of Infantilism.]

551. Spicer, Scanes: A Case of Achondroplasia in a child aged three years. *Journal of Laryngology, Rhinology and Otology*, N.S., Vol. xxii. pp. 57—59. London, 1907. [Pedigree No. 687.]

552. Hobhouse, E.: A Case of (?) Achondroplasia. *British Medical Journal*, 1907, Vol. ii. pp. 85—86. London, 1907. [A girl aged $5\frac{1}{2}$, family history nil. She had some rickety stigmata, but showed in addition peculiar features. The femora were bowed and very short. Length of femur $7\frac{3}{4}$ inches, length of tibia $6\frac{1}{4}$ inches. The arms were short as compared with the body.]

553. Clarke, H. H.: Chondrodystrophia foetalis. *The Liverpool Medico-Chirurgical Journal*, Vol. xxvii. No. 52, pp. 219—228. London, 1907. 4 Plates. [A short general account of chondrodystrophia foetalis is given with a detailed description of his own case. The child was born alive but died 20 minutes later. No family history.]

554. Gundlach, J.: Achondroplasia. *St Bartholomew's Hospital Reports*, Vol. 42, 1906, p. 187. London, 1907. [A still-born female achondroplasic foetus was presented to the Museum by Gundlach; extremities characteristically stunted. The mother, aged 25, was a primipara.]

555. Geist, Emil S.: Chondrodystrophia Foetalis. *The American Journal of Orthopedic Surgery*, Vol. v. pp. 240—248. Philadelphia, 1907—8. [A general discussion on achondroplasia with a case of his own, a female infant aged 14 months, height 61 cm. It was the first child of well-proportioned Roumanian parents. Full measurements are given.]

556. *Hegar: Entwickelungsstörungen, Fötalismus und Infantilismus. *Münchener medizinische Wochenschrift*, 1907, S. 737. München, 1907. [Reference wrong, paper not found: see, however, Hegar's *Beiträge zur Geburtshilfe*, Bd. x. Heft 2, 1906, and Bd. xii. Heft 1, 1907. Leipzig.]

557. Poisson : Un cas très curieux de nanisme. *Gazette médicale de Nantes*, 2e Série, T. xxv. pp. 210—212. Nantes, 1907. [A case of ateleiosis. A male dwarf, height 134 cm., admirably proportioned and without any deformity. He was very intelligent, belonging to a profession which required full possession of the mental faculties. But the sexual organs and sexual instinct were atrophied. It states that M. Sourdille had seen a similar case, in which the generative functions were normal.]

558. Comby, Jules: Nouveau cas d'achondroplasie. *Archives de Médecine des Enfants*, T. x. pp. 349—352. Paris, 1907. [A girl aged 16 months, only child of normal parents ; the father was aged 28, the mother 29. She was an 8 months' child. Height 55 cm. The limbs were very short, with the upper and lower segments of equal length. She died of hypothermia. It is stated that the diagnosis was very difficult, but that rachitis, mongolian idiocy and congenital myxoedema were excluded. Skiagrams are given.]

559. Chaumier et Taty : Confusion mentale chez un achondroplase, glycosurie, acetonurie. xviie Congrès des Médecins Aliénistes et Neurologistes de France et des Pays de langue française, Geneva-Lausanne, 1—7 août, 1907. *Le Bulletin médical*, T. xxi. Pt 2, p. 713. Paris, 1907. [An achondroplasic man, aged 28, suffered from polydipsia and took to drink. He became mentally affected and attempted suicide but when the glycosuria and acetonuria were cured under treatment, he recovered. The authors state that his hereditary antecedents support the theory that tuberculosis plays an important part in the genesis of achondroplasia, but no details are given in this report of the paper.]

560. Papillon : Achondroplasie ou rachitisme. *Le Bulletin médical*, T. xxi. p. 1121. Paris, 1907. [Three children were shown at the Société de Pédiatrie 17 déc. 1907, two of whom were achondroplasic. The eldest, a girl aged 8, was radiographed. The third child was a less typical case as the skull was normal.]

561. Mills, Albert: Sur un cas de nanisme generalisé, aplasie partielle, disséminée. *Clinique*, T. xxi. pp. 161—164. Bruxelles, 1907. [A child, aged 2 days, whose length and weight were much below the normal. It appeared well proportioned, all parts of its body exhibited arrested development in varying but symmetrical degrees. The sexual organs showed not only arrested development but were also abnormal. Superficially it appeared to be of the female sex. The right hand had a supplementary finger, with syndactyly. It had clubfoot (talus valgus) on both sides. It died a few hours after examination and an autopsy could not be performed. The father was healthy and had healthy children by his first marriage. The mother had always miscarried previously.]

562. Guérin-Valmale: Bassin rachitique, opération césarienne. *Société des Sciences Médicales de Montpellier*, 14 déc. 1906. Rapporté dans *La Gazette des Hôpitaux de Toulouse*, 1907. [A primipara aged 32, height 120 cm. She was very deformed. Caesarian section was performed and a living child extracted. Both mother and child lived.]

563. Charon, Degouy et Tissot : Un cas d'Achondroplasie. *Nouvelle Iconographie de la Salpêtrière*, T. xx. pp. 390—395. Paris, 1907. 4 Plates. [Pedigree No. 678.]

564. Cavazzini, A.: Sur la Pathogénie de l'Achondroplasie. *La Pédiatrie Pratique*, 1907, ve année, pp. 125—130. Lille, 1907. Also **La Pediatria*, 2a Serie, T. v. pp. 168—178. Napoli, 1907. [A boy aged 6. Parents and two sisters normal. He had very short limbs, the upper segments being shorter than the lower. At age of 3 his height was about 66 cm. He was electrically treated, massaged and his limbs stretched by weights; in 4 months he had grown 9 cm. When seen in 1906 his height was 90 cm. The special point of interest in his case was that his mother before and during her pregnancy with this child had consumed a large quantity of thyroid tablets to make herself thin.]

565. Apert, E. : La dysthyroïdie bénigne chronique. *Bulletins et Mémoires de la Société médicale des Hôpitaux de Paris*, 24e année, pp. 528—538. Paris, 1907. [This paper is on infantilism and myxoedema and the effects of thyroid treatment on growth ; some cases are described.]

566. Apert, E.: L'Achondroplasie, maladie hérédo-familiale et les autres dysostoses familiales. *Maladies familiales et maladies congénitales*, Chap. vi. pp. 95—126. Paris, 1907. [A general discussion on achondroplasia in human beings and animals, its origin and the affections which must be distinguished from achondroplasia.]

567. *Schirmer, K. H. : Achondroplasie (Chondrodystrophia foetalis). Mikromelie. *Centralblatt f. d. Grenzgeb. d. Medicin und Chirurgie*, Bd. x. S. 641 and 689. Jena, 1907.

568. *Pelizzi : Dell' Infantilismo. *Annali di Freniatria*, 1907.

569. *Berghino : Sopra un caso di Achondroplasia. vio *Congresso italiano di Pediatria*, 2—5. Oct. 1907.

570. Bowlby, Anthony A.: Achondroplasia. *Surgical Pathology and Morbid Anatomy*, 5th edition, pp. 320—322. London, 1907. [A few remarks on achondroplasia, with an illustration taken from Thomson's paper in the *Edinburgh Medical Journal*, Bibl. 281.]

571. Fruhinsholz, A., et Michel, Gaston: Opération de Porro chez une femme achondroplasique, césarotomisée pour la quatrième fois. *Annales de Gynécologie et d'Obstétrique*, 2e Série, T. iv. pp. 23—28. Paris, 1907. [A woman aged 31 had had six pregnancies and the paper gives an account of the seventh. The first and second accouchements were terminated by basiotripsia, the third, fourth and fifth by caesarian section, the sixth by a miscarriage, the seventh by Porro's operation, a girl weighing 2935 grammes being extracted. In this last operation the uterus was also removed. Only a few uterine measurements are given.]

572. *Boulenger: Idiotie et Achondroplasie. *Journal de Neurologie*, No. 13, 1907. [Account of an achondroplasic idiot.]

573. *Wiencke: Chondrodystrophie als Ursache der Phokomelie. *Münchener medizinische Abhandlungen*, Erste Reihe, Heft 31. München, 1907. (Arbeiten aus d. Path. Institut.)

574. Mackenzie, Hector: Cretinism: Synonyms—Cretinoid Idiocy, Infantile Myxoedema, Cretinismus, Crétinisme, Pachydermic Cretinoids, Cachexia Pachydermica. *Allbutt and Rolleston's System of Medicine*, Vol. iv. Pt i. pp. 333—344. London, 1908. [A short article on cretinism, pp. 337—339, gives average stature and general appearance of cretins. There is a bibliography.]

575. Drysdale, J. H., and Herringham, W. P.: An undescribed form of dwarfism associated with a spatulate condition of the hands. *Quarterly Journal of Medicine*, 1908, Vol. i. No. 2, pp. 193—197. Oxford and London, 1908. [Pedigree No. 704.]

576. Thomson, John: Achondroplasia (Chondrodystrophia foetalis). *Guide to the Clinical Examination and Treatment of Sick Children*, pp. 488—492. Edinburgh and London, 1908. [On the clinical features, etc., of achondroplasia.]

577. Thomson, John: Infantilism. *Allbutt and Rolleston's System of Medicine*, Vol. iv. Pt i. pp. 486—492. London, 1908. [An article on infantilism and its possible causes.]

578. Regnault, Felix: Enfoncement de la base du crâne (platybasie) chez une achondroplase. *Bulletins de la Société anatomique de Paris*, 6e Série, T. ix. pp. 439—440. Paris, 1908. [Description of the skull of an achondroplasic female with platybasia. He says this malformation though often found in rickets had not been found before in achondroplasia.]

579. Regnault, Felix: Anomalies des plis de la main dans l'achondroplasie et la dysostose. *Bulletins de la Société anatomique de Paris*, 6e Série, T. ix. pp. 439—440. Paris, 1908. [Title describes subject.]

580. Planchu: Foetus pseudo-achondroplasique (dysplasie périostale de Porak et Durante). Réunion obstétricale de Lyon, 19 fév. 1908. *Bulletins de la Société d'Obstétrique de Paris*, T. xi. pp. 106—110. Paris, 1908. *L'Obstétrique*, 1908, p. 387. Paris, 1908. *La Presse médicale*, 1908, p. 141. Paris, 1908. [Presentation of the skeleton of a foetus, length 38 cm. The trunk was normal, head nearly normal, the limbs short with the long bones fractured. The mother, aged 32, had at 18 undergone partial thyroidectomy. She had had three normal children who died in infancy.]

581. Trillat: Achondroplasie foetale, difficultés du palper pendant la grossesse. Réunion obstétricale de Lyon, 16 avril, 1908. *Bulletins de la Société d'Obstétrique de Paris*, T. xi. pp. 183—185. Paris, 1908. Also *L'Obstétrique*, 1908, No. 4, p. 392. Paris, 1908. [A similar case to that of Planchu, Bibl. No. 580, a female foetus, firstborn child, length 36 cm. weight 1850 grammes. It showed all the characteristics of pseudo-achondroplasia, with arrested development of the limbs and absence of ossification of the bones of the vault of the skull. The mother, aged 30, was normal.]

582. Gonnet: Un cas de pseudo-achondroplasie, type dysplasie périostale. *Réunion obstétricale de Lyon*, 16 avril, 1908. *Bulletins de la Société d'Obstétrique de Paris*, T. xi. p. 185. Paris, 1908. Also *L'Obstétrique*, 1908, No. 4, p. 392. Paris, 1908. [This infant, which lived for a day, had several abnormalities. In appearance it resembled a basset-hound. Total length 43 cm. The thorax and abdomen were nearly normal, but there was pronounced micromelia of the limbs, and the long bones were fractured.]

583. Gilles et Dargein: Foetus abortif achondroplasique. Société d'Obstétrique de Toulouse. *Comptes Rendus de la Société d'Obstétrique, de Gynécologie et de Pédiatrie de Paris*, T. x. pp. 252—255. Paris, 1908. [The mother showed signs of Basedow's disease and had an enormous uterine

tumour, very probably a dermoid cyst. It was a 5 months' foetus, length 30·5 cm., and exhibited the typical characteristics of achondroplasia. Family history negative. A long description is given.]

584. Guinon, L. Lobligeois, et Apert, E.: Présentation de radiographies d'achondroplasiques. *Bulletins de la Société de Pédiatrie de Paris*, 1908, pp. 199—204. Paris, 1908. [Description of the radiographs of the achondroplasic girl described in Bibl. No. 495.]

585. Méry et Parturier: Un cas de rachitisme congénital. *Bulletins de la Société de Pédiatrie*, 1908, pp. 233—240. Paris, 1908. [A long description is given of a male infant aged about 6 weeks, with thick, short, incurved limbs. Length of right arm 19 cm., of left 16 cm. Length of right leg 14·5 cm., of left 12·5. The father was aged 36, the mother 31, both healthy; they had an elder child aged $7\frac{1}{2}$ years who was healthy. The authors say it is a case of congenital rachitis, not achondroplasia.]

586. Marfan, A. B.: Sur le rachitisme congénital. *Bulletins de la Société de Pédiatrie*, 1908, pp. 241—247. Paris, 1908. [The author states there are two kinds of congenital rachitis, one type which he calls pure and one type associated with achondroplasia.]

587. Schrumpf, P.: Ueber das klinische Bild der Achondroplasie [Chondrodystrophie] beim Erwachsenen und eine, ihr sehr ähnliche bisher noch nicht beschriebene Form von mikromelen Zwergwuchs bei einer 56-jährigen Frau. *Berliner klinische Wochenschrift*, 1908, Jahrgang xlv. S. 2137—2142. Berlin und Leipzig, 1908. [Discussion of the characteristics of achondroplasia, with account of a woman aged 56, height 119 cm. She had normal head and trunk and short limbs. Her brothers and sisters were normal. She appeared normal at birth and developed normally till the age of 7 when she had a feverish affection. Her arms and legs became paralysed and she had to remain lying till the age of 15, when she began to walk and use her hands again. Her trunk and head continued to develop during this period but her limbs ceased to grow.]

588. Levi, Ettore: Contribution à l'étude de l'infantilisme du type Lorain. *Nouvelle Iconographie de la Salpêtrière*, T. xxi. pp. 297—324 and pp. 421—471. Plates. Paris, 1908. [There is a long and useful bibliography of infantilism. Pedigree No. 829.]

589. *Plummer, W. E.: Achondroplasia. *China Medical Journal*, Vol. xxii. p. 360. Plate. Shanghai, 1908.

590. *Hochsinger, K.: Diagnostische Betrachtungen über einen Fall von Chondrodystrophia foetalis in Säulingsalter. *Zentralblatt für Kinderheilkunde*, Bd. xiv. S. 43—48. Leipzig, 1908.

591. *Paugay et Gallinger: Foetus achondroplasique. *Écho Méd. du Nord*, T. xii. p. 34. Lille, 1908.

592. *Speidel, E.: Report of an obstetrical Case. Achondroplasia. *Kentucky Medical Journal*, Vol. vii. p. 500. Bowling Green, U.S.A., 1908—9.

593. *Brudzinski: Myxoedème infantile, Mongolisme et Achondroplasie. *Archives de Médecine des Enfants*, 1908, No. 8. Paris, 1908.

594. Labbé, Marcel, Rosenthal, G., et Marcorelles: Rétrécissement mitral pur et le nanisme. *Bulletins et Mémoires de la Société médicale des Hôpitaux de Paris*, T. 25, 3e Série, pp. 636—640. Paris, 1908. Also *La Presse médicale*, 1908, pp. 307 and 497—499. Paris, 1908. [A man, Leon H., aged 27, height 1·51 metres. He had mitral contraction (rétrécissement mitral) and showed signs of hereditary syphilis and arrested pulmonary tuberculosis. Some details of his family are given, but he seems too tall to be considered a dwarf.]

595. Raymond, Felix, et Claude, J.: Sur une forme de dyschondroplasie avec arthropathies et micromélie (Pseudo-achondroplasie rhumatismale). *Comptes Rendus de la Société de Biologie*, T. 64, pp. 263—265. Paris, 1908. Also *La Presse médicale*, 1908, p. 118. Paris, 1908. [Case of a girl aged 20 who, according to her parents, was absolutely normal till the age of 7, and then had acute articular rheumatism followed by stiffness of the joints and complete ankylosis of some of them so that the bones ceased to develop in length. She was under observation for 4 years: the face and trunk developed normally. Compare Schrumpf's Case, Bibl. No. 587.]

596. *Busi: Tre casi di nanismo da mixedema. *Archivio di Ortopedia*, 1908, Fasc. 2.

597. Neusser: Zur Klinik der chronischen Polyserositis. Morbus Bamberger (Rhumatismus und Infantilismus). *Wiener klinische Wochenschrift*, No. 14, S. 489, 1908. Wien, 1908.

598. Bullard, Wm., and George, Arial W.: Achondroplasia. *Boston Medical and Surgical Journal*, 1908, Vol. clviii. pp. 969—971. Boston, 1908. [The chief characteristics of achondroplasia are given, and one case, a boy, first seen in July, 1894, then aged 10 months, is described. The parents were healthy, the father aged 54, the mother aged 30. She had had five pregnancies. (1) Boy aged 8, well. (2) Girl aged 7, well. (3) Miscarriage at the 4th month. (4) Boy

aged 4½, well. (5) Patient. Picture and description of the boy when aged 14 are given, and show the typical achondroplasic type. He had slight rosary, rachitic curvature of spine and double coxa vera. No measurements are given. He was undergoing thyroid treatment.]

599. *Cerletti: Nuove ricerche circa gli effetti delle iniezione del succo d'ipofisi e di altri succhi organici nell' accrescimento somatico. *Reale Accademia dei Lincei Roma*, 26 aprile e 3 maggio, 1908. Roma, 1908.

600. *Porot: Le Nanisme à la cour des Beys (quelques cas). Reference in *Revue neurologique*, 1908, p. 896. Paris, 1908.

601. *Sandri: Contributo all' anatomia et alla fisiologia dell' ipofisi. *Rivista di Pat. nerv. e mentale*, 1908, p. 518.

602. *Wiesermann: *Ueber Chondrodystrophie foetalis mit besonderer Berücksichtigung ihrer Entstehung durch mechanischen Ursachen.* Inaug. Diss. Marburg, 1908.

603. *Knoop: Chondrodystrophia foetalis. *Versammlung deutscher Naturforscher und Aerzte.* Köln, 1908.

604. Emerson, C. P.: Achondroplasia. *Osler and Macrae's Modern Medicine, its Theory and Practice*, 1st edition, Vol. vi. pp. 683—703. London, 1909. [Gives an account of the disease and the literature on the subject.]

605. Moir, Gordon: Achondroplasia occurring in a Chinaman. *British Medical Journal*, 1909, Vol. ii. p. 516. London, 1909. [Pedigree No. 677 and Plate R ((11)—(13)).]

606. Dixon, A. Francis: The skeleton in Achondroplasia. *British Medical Journal*, 1909, Vol. ii. pp. 672—673. London, 1909. [On the peculiarities of the achondroplasic skeleton.]

607. Charles, J. R.: A Case of Foetal Rickets with comments. *The British Journal of Children's Diseases*, Vol. v. pp. 293—299. London, 1909. [The parents were alive and healthy. The maternal grandmother and one uncle died from cancer. The child was the third of a family of five; another child, aged 9, was said to have been born with rickets, but there was no great amount of evidence in support of this statement.]

608. Eckstein, H.: Ein eigenartiger Fall von Achondroplasie (Chondrodystrophia foetalis). *Berliner klinische Wochenschrift*, 1909, Jahrgang xlvi. S. 1072—1073. Berlin und Leipzig, 1909. [Pedigree No. 686.]

609. Hempstead, Helen: Achondroplasia. Report of a case with pathological Report. *The Cleveland Medical Journal*, Vol. 8, No. 11, pp. 675—682. Cleveland, U.S.A., 1909. [A short discussion of the subject with one case, a boy aged 1 year, height 70 cm. Distance from crown to umbilicus 38 cm., from umbilicus to soles 32 cm. Height of father 187 cm., height of mother 154 cm. There were two sisters aged 5, and two who were very healthy (it is doubtful whether this means four sisters or two sisters), and one brother aged 3, whose legs showed extreme rachitic deformities. Full measurements are given.]

610. Apert, E.: Une famille d'achondroplasiques (présentation de malades). *Bulletins de la Société de Pédiatrie de Paris*, No. 2, fév. 1909, pp. 35—37. Paris, 1909. [This gives further particulars of the case described by Launois and Apert in 1905, Bibl. No. 495.]

611. Fussell, M. H., McCombe, Robt. S., de Schweinitz, George L., Pancoast, Henry K.: Achondroplasia. *The Journal of the American Medical Association*, Vol. 53, pp. 1614—1617 and 1617—1619. Chicago, 1909. Also **Pennsylvania Medical Journal*, Athens, Penn., 1909—10, Vol. xiii. pp. 751—756. [pp. 1614—1617, a short paper discussing the main features of achondroplasia with pictures. pp. 1617—1619, characteristic radiographic features of achondroplasia, cretinism and rickets, by Henry K. Pancoast.]

612. Bauer, A.: Infantilisme et Chétivisme. *La Presse médicale*, 1909, 17e année, pp. 870—872. Paris, 1909. [A discussion on the different kinds of infantilism. He proposes to use the word "Chétivisme" to designate "Infantilisme du type Lorain."]

613. Marie, A.: Nano-infantilisme et Folie. *Bulletins et Mémoires de la Société d'Anthropologie de Paris*, 5e Série, T. x. Fasc. 2, pp. 101—113. Paris, 1909. [p. 101 gives a picture of four male dwarfs whom Marie says he observed in Germany. No details are given: see our Plate JJ (72). He divides dwarfism into three classes. (1) Pure dwarfism with relative proportions perfect but reduced. (2) Dwarfism and infantilism with deformities of the skeleton. This class has three sub-divisions. (3) Dwarfism and infantilism caused by dystrophies. This class is divided into two: Total—with five sub-divisions, and Local—with nine sub-divisions specified. He discusses the views of various authors.]

614. *Dziembowski: Przyegynek de nanki o zboczeniach ivzrostn chrznstek (Chondrodystrophia foetalis). *Now. lek. Poznai*, xxi. pp. 586—590. 1909.

615. *Beneke: Chondrodystrophia foetalis. *Sitzungsbericht der Gesellschaft zur Beforderung der gesammten Wissenschaften zu Marburg*, 1908, S. 38—41. Marburg, 1909.

616. *Markeloff, G. T.: (A typical case of Achondroplasia). *Russk. Vrach.* viii. pp. 824—826. St Petersburg, 1909.

617. *Voisin, J., et Voisin, R.: Un cas d'achondroplasie. *L'Encéphale*, 1909, T. ii. pp. 221—227. Paris, 1909. Two Plates.

618. Sainton, Paul: Les Nains. *La Tribune médicale*, 1909, 42e année, pp. 293—294. Paris, 1909. [A short article on dwarfs in general. He divides them into the following classes: (1) myxoedematous, (2) achondroplasic, (3) rachitic, (4) "nains pottiques" (see ft. p. 369 *supra*), (5) anangioplasic, (6) pygmies, (7) dwarfs of surrenal origin. He refers to an achondroplasic brother and sister, aged 37 and 32 years respectively, with five normal siblings and normal parents, the family P—— from Bordeaux.]

619. Launois, P. E.: Essai biologique sur les nains. *Le Bulletin médical*, 23e année, pp. 957—962. Paris, 1909. [A lecture on dwarfs illustrated by poor reproductions of some very good photographs of a troupe (largely same as London Olympia troupe: see p. 406 *supra*) exhibited in the Jardin d'Acclimatation, Paris, 1909. Among these the Magri family, a mulatto dwarf, and a Russian achondroplasic dwarf shown with a slightly taller dwarf son at Earl's Court Exhibition in August, 1911.]

620. Levi, Ettore: Sur un nouveau cas d'achondroplasie chez l'adulte. *Nouvelle Iconographie de la Salpêtrière*, T. xxii. pp. 133—152. Paris, 1909. Plates. [Account of Luigi Lasti, aged 25, height 115 cm. Father normal, died aged 45 of an acute infectious disorder. Mother normal, alive and healthy and intelligent. Two sisters and a brother alive and healthy, three sisters died of acute disease, all normal.]

621. Regnault, Felix: Plusieurs cas de dysplasie périostale montrant les divers degrés d'intensité de cette maladie. *Bulletins et Mémoires de la Société anatomique de Paris*, 6e Série, T. xi. pp. 429—432. Paris, 1909. [A description of some specimens in the Musée Dupuytren, two of which were achondroplasic. Regnault shows how periostal dysplasia and achondroplasia have been confounded.] *Ibid.* pp. 433—434, Micromélie segmentaire et symétrique. [Description of a skeleton in the Musée de Toulon which showed limited achondroplasia or micromelia limited to the two humeri.] See also *La Presse médicale*, 1909, pp. 503—504. Paris, 1909.

622. Dustin: Sur le nanisme, présentation de nains myxoedémateux, rachitique et achondroplasique. Société Clinique des Hôpitaux de Bruxelles, 12 Juin, 1909. *La Presse médicale Belge*, 61e Année, 1909, pp. 628—629. Bruxelles, 1909. [This appears to be a paper or a series of papers on the different kinds of dwarfism of which only the first instalment is given in the above reference.] See also **Policlinique*, T. 18, pp. 198—200. Bruxelles, 1909. **Journal médical de Bruxelles*, T. 14, p. 375. Bruxelles, 1909. **Clinique*, T. 23, pp. 452—454. Bruxelles, 1909.

623. *Giuffrida, Ruggeri: I caratteri pseudo-infantile. *Archivio per l'Antropologia e l'Etnografia*, 1909, Nos. 1—2.

624. *Birnbaum: Klinik der Missbildungen und kongenitalen Erkrankungen des Fötus. Berlin, 1909.

625. Weinzierl, Hans: Ein Beitrag zur Casuistik der Chondrodystrophia foetalis, mit 7 Abbildungen. *Archiv für Kinderheilkunde*, Bd. 51, S. 138—150. Stuttgart, 1909. [A few remarks on Chondrodystrophia foetalis with description of a case. A boy aged 17 months, parents healthy, height 65·5 cm. He had short arms and legs, and genu valgum. The upper extremities were longer than the lower. There is a long description and measurements. No family history.]

626. Article: Tiny Town. *British Medical Journal*, Vol. ii. p. 1768. London, 1909. [On the dwarfs exhibited at Olympia, London. Says there were some striking examples of achondroplasia; two of exceptional interest inasmuch as they were instances of achondroplasia in parent and offspring. According to the mother in this case there were two other deformed children, who died in infancy. The reference is probably to the Kipke family: see Pedigree No. 608.]

627. Bloch, Adolphe: Présentation de radiographies des mains d'un nain et d'un achondroplase. *Bulletin de la Société de l'Internat des Hôpitaux de Paris*, 6e année, pp. 324—328. Paris, 1909. [In addition to the radiographs of the hands, photographs are given of the ateliotic dwarf aged about 21, height 98 cm. in his shoes, and of the achondroplasic dwarf aged 28, height 127·8 cm.]

628. Molodenkoff, S. S.: Un cas d'achondroplasie chez un Chinois. *Nouvelle Iconographie de la Salpêtrière*, T. xxiii. pp. 43—46. Paris, 1910. [Account of an achondroplasic Chinaman. No family history.]

629. James, C. H.: Three varieties of dwarfs. Plates. Reprint from *The Indian Medical Gazette*, Vol. xlv. No. 11, Nov. 1910. [Pedigrees Nos. 796 and 797 and our Plates LL and MM.]

630. Batty-Shaw, H.: Two Cases of Symptomatic Infantilism. *Clinical Section. Proc. Royal Society of Medicine*, Nov. 11, 1910; also *The Lancet*, Vol. II. 1910, p. 1487. London, 1910. [Two cases of the symptomatic infantilism described by Hastings Gilford were shown. (1) A female, aged 18, height 4 ft. 4½ inches, weight 4 stones 4½ pounds, belonging to a family of normal height and weight. The delay in growth was first noticed in the third year of life. (2) A female, aged 27, height 4 ft. 6 inches, who also belonged to a family of average height and weight. She was a 6 months' child, and at the age of 1 year was smaller than her new-born sister. A case of asexual ateleiosis (sub-group of essential or cryptogenetic infantilism of Hastings Gilford, Bibl. No. 403, p. 316). The patient was a male, aged 36, height 3 ft. 8¾ inches, weight 4 stones 11 lbs. 1 oz.]

631. Pritchard, Eric: A Case of Achondroplasia. *Section for the Study of Disease in Children. Proc. Royal Society of Medicine*, Vol. IV. No. 1, pp. 1—3. London, 1911. Plate. Also *British Medical Journal*, 1910, Vol. II. p. 1442, and *The Lancet*, 1910, Vol. II. p. 1344. London, 1910. [A girl aged 5, height 30 in. Length of arm 10 in., length of leg 12½ in. The skiagrams showed defective ossification of the epiphyses of the long bones, with overgrowth of the cartilages. The patellae were either absent or rudimentary. Parents and five other normal children were healthy. A full description is given in first reference.]

632. Jubb, A. A.: A Case of Dwarfism. *British Medical Journal*, Dec. 31, 1910, p. 2026. [H. W., a boy of 9, height 36½", with photographs; said not to be a case of achondroplasia but of infantilism and rickets. Father, mother, and four siblings said to be normal.]

633. Article: Dwarfs. *Encyclopaedia Britannica*, 11th Edition, Vol. VIII. pp. 739—740. Cambridge, 1910. [A short article giving an account of some well-known dwarfs, such as John Jarvis, Jeffrey Hudson, etc. The information is apparently taken chiefly from Wood; see Bibl. No. 138.]

634. Forssell, O. H.: Ett fall af Chondrodystrophia foetalis. *Hygeia*, 2 F. Bd. X. pp. 550—553. Stockholm, 1910. [A foetus asphyxiated at birth, length 40 cm., weight 3200 grammes. It is said to have exactly resembled the case described by Salvetti, Bibl. No. 298. The extremities were curved. A description was given, but no measurements. The mother, aged about 30, had had two normal children, one 12 years, the other 4 years, previously. The father was said to be alcoholic; nothing could be ascertained with regard to syphilis.]

635. Jeannin, C., et Surun: Foetus achondroplasique. *Bulletins de la Société d'Obstétrique de Paris*, T. XIII. pp. 181—184. Paris, 1910. [A male infant, length 36 cm., weight 2120 grammes, died one hour after birth. The thorax was normal, the limbs short and thick with enlarged epiphyses. The mother had had a previous child in 1907, who died, aged 7 months, of broncho-pneumonia.]

636. Cafferata, Juan F.: Un cas d'achondroplasie. *Archives de Médecine des Enfants*, T. XIII. pp. 275—276. Paris, 1910. [A girl aged 2 years and 7 months. The family history was unimportant. The parents were healthy, the father aged 40 and the mother aged 30. There were two elder children, boys, both healthy. Her height was hardly 71 cm. She had a large head and short limbs, the upper and fore-arm being of same length. Radiographs are given.]

637. Variot, G., et Pironneau: Nanisme avec dystrophie osseuse et cutanée spéciales (*sic!*). Soupçon d'agénesie des capsules surrénales. *Bulletins de la Société de Pédiatrie de Paris*, 1910, pp. 307—314. Paris, 1910. Also *Ibid.* 15 Nov. 1910 and *La Presse médicale*, 1910, pp. 494 and 894. Paris, 1910. [A girl aged 15, height 102 cm. and weight 11·650 kilos. Trunk and limbs were proportionately reduced. She had only a few scattered hairs on her head, and her expression was extraordinarily aged. A long description is given. She was an eight months' child, breast-fed, was weaned at 15 months and ceased to grow regularly from this time. The father and mother were healthy and had two normal sons, a third had died of meningitis. The second paper gives three observations sent to them by Gilford.]

638. Apert, E.: Achondroplasie. *Bulletins de la Société de Pédiatrie*, 1910, pp. 213—215. Paris, 1910. [Description of a boy aged 16⅓ years, height 122 cm. The mother was of abnormal height and weight. The father and three other children, two boys and a girl, were normal.]

639. Zozin, P.: Un cas d'achondroplasie. *Nouvelle Iconographie de la Salpêtrière*, T. XXIII. pp. 31—42. Paris, 1910. [A general discussion on achondroplasia with a new case; a boy aged 21, height 118 cm., weight 36 kilos. His parents were healthy and he had four brothers. There is a long description with measurements.]

640. Levi, Ettore: Contribution à la connaissance de la mierosomie essentielle, hérédo-familiale. Distinction de cette forme clinique d'avec les nanismes, les infantilismes et les formes mixtes de ces différentes dystrophies. *Nouvelle Iconographie de la Salpêtrière*, T. XXIII. pp. 522—561 and 600—684. Paris, 1910. Plates. [A discussion on the different forms of dwarfism. Pedigrees Nos. 695, 742.]

641. Levi, Ettore: Encore sur la question des infantilismes à propos d'une note de M. A. Bauer sur ce sujet. *Nouvelle Iconographie de la Salpêtrière*, T. xxiii. pp. 20—24. Paris, 1910. [Title describes subject: see Bibl. No. 612.]

642. Bauer, A.: Sur le Chétivisme [Réponse à M. Ettore Levi]. *Nouvelle Iconographie de la Salpêtrière*, T. xxiii. pp. 25—30. Paris, 1910. [Title explains subject: see Bibl. No. 641.]

643. *Apert, E.: Achondroplasie. *Annales de Médecine et Chirurgie infantiles*, T. xiv. pp. 391—393. Paris, 1910.

644. Franchini, Giuseppe, et Zamasi, Mauro: L'achondroplasie, est-elle héréditaire? Quatre cas d'achondroplasie chez des adultes. Étude clinique et radiographique. *Nouvelle Iconographie de la Salpêtrière*, T. xxiii. pp. 244—275. Paris, 1910. Plates. [A discussion on the heredity of achondroplasia, with four interesting cases. (1) Andrea Bernabé, a man aged 59, height 120 cm. His parents and two sisters were normal. No other details with regard to the family could be ascertained. A long description and measurements are given. (2) Auguste C.: see Pedigree No. 682. (3) Otto B., aged 23, height 113 cm. His parents, two sisters and three brothers were normal. Nothing remarkable in ancestors or collaterals. Full description and measurements given. (4) Anna G.: see also Pedigree No. 682.]

645. Milne, Robt.: Two Cases of Achondroplasia. *Clinical Section. Proc. Royal Society of Medicine*, Vol. iii. Pt i. p. 55. London, 1910. [Woman aged 22, height 45 inches, and boy aged 15, height 45 inches, not related, both typical cases of achondroplasia.]

646. Weber, F. Parkes: Ateleiosis in a man aged 42. Physical development said to have been arrested at about the age of 9 years. *Section for the Study of Disease in Children. Proc. Royal Society of Medicine*, Vol. iii. Pt i. pp. 143—146. London, 1910. [A man aged 42, unmarried, height 47·7 inches, weight 4 stone 13 lbs. His head was rather large for his body. Description and skiagrams are given. No history of dwarfism in the family.]

647. Hutchinson, R.: Achondroplasia in a twin. *Section for the Study of Disease in Children. Proc. Royal Society of Medicine*, Vol. iii. Pt i. p. 41. London, 1910. [A girl aged 1½ years, a case of well-marked achondroplasia. The twin was healthy. The mother had had two miscarriages. See our Plate P ((2)—(5)).]

648. Gilford, Hastings: Case of asexual Ateleiosis. *Clinical Section. Proc. Royal Society of Medicine*, Vol. iv. No. 2, pp. 34—35. London, 1910. [T. L S., a man aged 28, height 113·5 cm., weight 30·38 kilos. This is the same case as is described in Bibl. No. 403, p. 305.]

649. Chevalier-Lavaure et Voivenel: Nanisme mitral, sclérodermie et glandes à sécrétion interne. Congrès des Médecins Aliénistes et Neurologistes. *La Presse médicale*, 1910, p. 621. Paris, 1910. [After examining the various etiologies of mitral dwarfism, the authors considered the action of the glands concerned with internal secretions, which histological dissection showed were injured in a male dwarf aged 44, height 123 cm., weight 23 kilos. This dwarf had Maurice Raynaud's paralysis, followed by sclerodermia, which showed that these two pathological processes resulted from the same cause.]

650. Perrin, Maurice, et Richon, Louis: Le Nanisme toxique. *La Presse médicale*, 1910, pp. 339—340. Paris, 1910. [A paper on the effect of poisons, especially tobacco, on the growth of animals, with accounts of experiments on rabbits inoculated with infusions of tobacco. References are given to other papers on the same subject.]

651. Bloch, Adolphe: Renseignements fournis par la radiographie dans le nanisme et l'achondroplasie. *La Presse médicale*, 1910, 18e année, p. 45. Paris, 1910. [Shows how the uniting cartilages differ in true dwarfism and achondroplasia.]

652. Keppel et Chabrol, E.: Le nanisme mitral myxoedémateux. *Revue de Médecine*, T. xxx. pp. 153—161. Paris, 1910. [Account of a girl aged 16 who looked 12, height 145 cm. She was of the classic infantile type, with superior intelligence. Her father had died, aged 45, of an affection of the liver. There was said to be an hereditary alcoholic trait in the family. Her mother, aged 50, was healthy and she had three vigorous, well-built brothers, aged 27, 22 and 19. She died, and the autopsy showed that there existed "rétrécissement mitral et une hypoplasie du système artériel."]

653. *Minet, J., et Vendeau, M.: Cryptorchidie bilatérale, absence du verge, infantilisme chez un homme de 67 ans. *Écho Méd. du Nord*, T. 14, p. 51. Lille, 1910.

654. *Cramer: Zwei Fälle von Micromelie. *Archiv f. Orthop. etc.* Bd. viii. S. 258—269. Wiesbaden, 1910.

655. *Shabad, T. O.: Dwarf child (from trauma of the head). *Med. Obozr.* lxxiii. pp. 960—968. Moscow, 1910.

656. *Sante de Santos: Infantilismo e mentalia infantile. *Rivista Italiana di Neuropat. Psich. ed Elettroteropia*, 1910, p. 58.

657. *Viola: L'abito tisico et l'abito apopletico nei rapporti con l'infantilismo, la precocita, il gigantismo et la acromegalia. *La Clinica Medica Italiana*, aprile, 1910.

658. Sumita, Masao: Beiträge zur Lehre von der Chondrodystrophia foetalis (Kaufmann) und Osteogenesis imperfecta [Vrolik] mit besonderer Berücksichtigung der anatomischen und klinischen Differentialdiagnose, mit 9 Abbildungen. *Deutsche Zeitschrift für Chirurgie*, Bd. 107, S. 1—110. Leipzig, 1910. [A long paper with description of one case of Chondrodystrophia foetalis malacica, three of Chondrodystrophia foetalis hypoplastica and three of Osteogenesis imperfecta with elaborate tables of measurements. There is a discussion of the subjects mentioned in the title with a bibliography of 254 references to articles on myxoedema, cretinism, chondrodystrophia foetalis, achondroplasia, etc.]

659. *Mouchet et Sequinot: Soc. de Pédiatrie, 18 Jan. 1910, reported in *Archives de Médecine des Enfants*, T. xiii. p. 238. Paris, 1910. [They showed a girl aged 14, suffering from an abnormal form of achondroplasia, with multiple congenital malformations. Hexadactyly of both hands and absence of all the incisors.]

660. Symes-Thompson, H. E.: Case of Infantilism. *Clinical Section. Proc. Royal Society of Medicine*, Vol. iv. No. 3, pp. 45—46. London, 1911. [A woman aged 34, single. height 3 ft. $2\frac{1}{2}$ in., weight 3 stone. She ceased to grow at 3 or 4 years of age. There was nothing of note in the family history. The thyroid body could not be felt. Skiagrams showed a backward state of ossification. The author says this case, according to Hastings Gilford's classification, was one of primary infantilism or ateleiosis.]

661. Kellie, Kenneth: Two cases of Infantilism. *Section for the Study of Disease in Children. Proc. Royal Society of Medicine*, Vol. iv. No. 4, p. 59. London, 1911. [Case (1). A girl aged 4, weight 17 lbs. 2 oz., height $29\frac{1}{4}$ inches. The parents and two other children were healthy, one child died of wasting (?), one child was born dead. Case (2). A boy aged $3\frac{1}{4}$, a twin; the other died. Weight 16 lbs., height 28 inches. Two elder children and one younger were healthy.]

662. Apert, E: *Médications générales de la croissance*, pp. 418—419. Paris, 1911. [On the various kinds of retardation in growth and the best method of treating them. pp. 418—419 treat of "Nanisme dit essentiel," "Nanisme achondroplasique" and "Nanisme micromélique."]

663. Fletcher, H. Morley: Case of Infantilism with Polyuria and Chronic Renal Disease, and a case of Infantilism with Thyroid Inadequacy. *Section for the Study of Disease in Children. Proc. Royal Society of Medicine*, Vol. iv. No. 6, pp. 95—96. London, 1911. [(1) Boy aged 6, said not to have grown since the end of his first year. Height 2 ft. 7 inches, weight 21 lbs. He was the eldest child and had a healthy sister aged 4. The mother was phthisical and had had one miscarriage. Administration of thyroid had no effect. This case was considered one of infantilism associated with or due to chronic renal disease dating from intrauterine existence. (2) Girl aged 8. Height 33 inches, weight $24\frac{1}{2}$ lbs. She was the seventh of nine children, all alive except the third. Thyroid treatment did not affect growth.]

664. Gilford, Hastings: *The Disorders of Post-Natal Growth and Development*. London, 1911. [Deals with infantilism, ateleiosis, and to a less extent with achondroplasia, myxoedema, and cretinism, largely from an individual standpoint.]

ADDENDUM.

282. Müller, Sigfrid. [His paper of 1893 gives an exhaustive account of Anna Harlander born 17 Jan. 1892, daughter of a tram-driver; she died aged 5 weeks. She was a twin and the seventh child of apparently healthy parents. No syphilis in parents. Birth 5 weeks before term. She was born with curved (krummen) limbs. Length 33·5 cm. The first child died aged 1 year from convulsions due to teething ("an Zahnfreisen"). The second was a miscarriage at 4 months, the third a premature birth at 6 months, it died 1 hour after birth. The fourth, aged 4, was scrofulous. The fifth, aged 3, and the sixth, aged $1\frac{1}{2}$, were healthy. The second twin was perfectly healthy and well developed.]

ALPHABETICAL LIST OF WRITERS REFERRED TO IN BIBLIOGRAPHY.

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The Index to names of historic dwarfs and published names of dwarf families follows the family histories.

	Artist	Dates of Artist	Subject	Place	Description of Dwarf
1	Aretino, Spinello.	1333—1410 (circa)	St Benedict discovers the guile of Totila.	Fresco. Sacristy of San Miniato, Florence.	Two attendants on left of picture of short stature said to be dwarfs. No special characters which would identify type.
2	,, ,,	,, ,,	St Benedict leaves the paternal home.	Fresco. Sacristy of San Miniato, Florence.	Attendant said to be dwarf on left of picture, but no characteristic features.
3	Fra Angelico, or Angelico da Fiesole.	?—1455	The abbot St Romuald reproaching the Emperor Otho III with the murder of Crescentius, a Roman Senator.	Museum. Antwerp.	A dwarf almost certainly achondroplasic holds the Emperor's sword.
4	Lippi, Filippino.	1458—1504	Adoration of the Magi (ascribed to Lippi, but has been said to be a masterpiece of Botticelli freely retouched).	National Gallery, London.	A bearded dwarf, almost certainly achondroplasic, to left of picture.
5	Fogolino, Marcello.	1470—1550	Adoration of the Magi.	Museo Civico, Vicenza.	A dwarf with adult face and thick legs, probably achondroplasic, to right centre of picture.
6	Mantegna, Andrea.	1431—1506	The Triumph of Julius Caesar.	Original cartoons at Hampton Court. Copies in Vienna.	Dwarf to right of picture. By shortness of lower limbs achondroplasia is suggested, and the muscularity and some features of head support this view. The shapes of arms and hands do not confirm it; there is marked macrocheilia more suggestive of myxoedema, and the ears are of a form which does not occur in the scale above the lower apes. The figure is probably a composite one.
7	,, ,,	,, ,,	The Family of Ludovico II, with a female dwarf in the service of Barbara von Brandeburg, wife of Ludovico.	Painted on the walls of the "Camera dei sposi" in the old Palazzo, Mantua.	To the right centre a female dwarf, probably a case of infantile myxoedema.
8	Ghirlandaio, Domenico.	1449—1498	The Feast of Herod.	Fresco on the right wall of the Choir Chapel in Santa Maria Novella, Florence.	A dwarf with his back to spectator to left centre of top compartment of picture, type uncertain, probably not achondroplasic.
9	Carpaccio, Vittorio.	1450—1522 (circa)	The ambassadors to the King of the Moors.	Accademia di Belle Arti, Venice.	A dwarf with bowed legs to left centre on quay at back of picture.
10	Carpaccio, Vittorio.	1450—1522 (circa)	Two Courtesans on a Balcony.	Museo Civico, Venice.	Dwarf to left back of picture under balustrade, infantile myxoedema (?).
11	Ansuino da Forli.	Middle of 15th century	St Christopher before the King.	Chiesa degli Eremitari, Padua.	Achondroplasic (?) dwarf to left, outside entrance to audience chamber.
12	Bosch, Hieronymus (properly Van Aken)	1460—1516 (circa)	Das Steinschneiden.	Rijksmuseum, Amsterdam.	Small dwarfish figure with big head and slender limbs in operating chair. Dwarf-like figure or gnome to left bottom on frame of picture.

[1] This iconography makes no claim to completeness, especially in the sections of prints, book illustrations, and in recent times, newspaper illustrations; many of the latter will be found referred to in the text or bibliography. Those marked with * have *not* been seen in original, in photograph or other reproduction by the iconographer.

SECTION I. *continued*

	Artist	Dates of Artist	Subject	Place	Description of Dwarf
13*	Cossa, Francesco.	Worked 1460—1486	?	?	Richer, *L'Art et la Médecine*, Paris, 1901, p. 208 mentions dwarf in a picture of Cossa's and describes him as a little man with features covered with fat and sausage-shaped limbs. He might pass for a myxoedematous type. His short tunic shows the genital organs atrophied. (Not reproduced.)
14	Cranach, Lukas.	1472—1533	The Sleeping Giant (or Hercules) and the Pygmies.	Königliche Galerie, Dresden.	Incident referred to in ftn. on p. 355. Hercules is attacked by gnomes or dwarfs of folklore type—as far as they are true to nature they are achondroplasic in type.
15	,, ,,	,, ,,	The Awakened Giant (or Hercules) and the Pygmies.	,, ,, ,,	
16	Giorgione or Barbarelli, Giorgio.	1478?—1511	Moses saved from the Waters.	Galleria Pitti, Florence.	To right of picture a bow-legged (?) dwarf, with hair on face, offers a goblet to female musicians.
17	Badila, Antonio.	1480?—1560	The presentation of Jesus to Simon.	R. Pinacoteca, Turin.	To extreme right (and (?) to extreme left also) a dwarf figure.
18	Ferrari, Gaudenzio.	1484—1549	Adoration of the Magi.	R. Pinacoteca, Palazzo Brera, Milan.	To right of central panel an achondroplasic dwarf.
19	,, ,,	,, ,,	,, ,,	Chiesa di San Giuliano, Vercelli.	Left side only of a dwarf figure to right of picture. Adult bearded face but hardly achondroplasic limbs.
20	Florentine School.	15th century	Episode in the life of the young Tobias.	Museum, Berlin.	In the centre is a dwarf dressed as a buffoon with violin on his shoulder. He has thick short legs, crooked nose and oldish head. (Richer, *L'Art et la Médecine*, Paris, 1901, p. 207.)
21	Romain, Jules (pupil of Raffaele).	1492—1546	Apparition of the Cross.	Fresco, Vatican, Rome.	? Achondroplasic dwarf figure to right, the figure might be rickety.
22	Gozzoli, Benozzo.	1420—1478	Tower of Babel.	Fragment of a fresco in the Campo Santo di Pisa.	An achondroplasic dwarf of remarkably small size on a rather damaged part of fresco. Cf. E. Levi, Un document medico-artistique sur l'achondroplasie. *Nouvelle Iconographie de Salpêtrière*, T. XXII. p. 228, Paris, 1909.
23	Bonifacio.	1491—1553	Moses saved from the Nile.	R. Pinacoteca, Palazzo Brera, Milan.	Achondroplasic dwarf to right centre indicated especially by hand.
24	Moretta da Brescia.	1498—1555 (circa)	Christ in the House of the Pharisee.	Chiesa della Pieta, Venice.	Achondroplasic dwarf by left-hand column.
25	Bassano, J.	1510—1592	The Preaching of the Baptist.	S. Giacoma dell' Orio, Venice.	Dwarf (? achondroplasic) to left-hand with dog.
26	Mor, Antonio (van Dashorst).	1512—1578?	The dwarf Corneille, favourite of Charles V.	Musée du Louvre, Paris.	Dwarf (? myxoedematous) with dog.
27	Veronese, Paolo.	1528—1588	Marriage of Cana.	,, ,, ,,	An adult achondroplasic dwarf with a parrot on his left shoulder stands table.

29	,, ,,	,, ,,	Moses saved from the Waters.	copy in Hermitage, St Petersburg.	of picture.
30	,, ,,	,, ,,	Discovery of Moses.	Königliche Galerie, Dresden.	? Same dwarf as in 28 to left of picture.
31*	,, ,,	,, ,,	Moses saved from the Waters.	A picture of this name by Veronese was in the cabinet of Louis XV in 1742, according to Richer, *L'Art et la Médecine*, Paris, 1901. (Is this the one now in the Hermitage, St Petersburg?)	Not seen.
32	,, ,,	,, ,,	Adoration of the Magi.	R. Pinacoteca, Palazzo Brera, Milan.	A dwarf, probably achondroplasic, holding a dog to right of picture.
33	,, ,,	,, ,,	The family of Darius before Alexander.	National Gallery, London.	Bow-legged (? rickety) dwarf to extreme left of picture.
34	,, ,,	,, ,,	Jesus in the House of Levi.	R. Accademia, Venice.	Dwarf to left centre, type not determinable.
35*	Strada, Giovanni, or Jan van der Straet.	1536?—1605	Dwarfs in several pictures.	Engraved by Galle (see Nos. 136—7 and p. 470).	We have seen several references to these dwarfs, but the original pictures have not been located
36	Liano, Felipe de.	1556—1625	Infanta Isabella with dwarf.	El Prado, Madrid.	Female dwarf of indeterminable type.
37	Van der Venne, Adriaen.	1589—1662	De Zielenvisscherij (1614).	Amsterdam.	Achondroplasic dwarf to right.
38*	Caracci, Annibale.	1560—1609	Satirical Composition.	National Museum, Naples.	At the left of the picture is a dwarf with bowed legs leaning on a dog; he has a parrot on his wrist. Described but not reproduced in Richer's *L'Art et la Médecine*, Paris, 1901.
39	Argenti, Jacopo.	2nd half of 16th century	Charles Emmanuel as a youth with his hand on the head of a dwarf.	Galleria Reale, Turin.	Achondroplasic dwarf.
40	Dutch School.	16th century	Ein Leiermann	Gemäldegalerie, Vienna.	Infantile myxoedema.
41	Rubens, Peter Paul.	1577—1640	Count Thomas Arundel with his wife, dwarf and falcon, fool and dog.	Alte Pinakothek, Munich. See No. 182[bis] below.	Dwarf to right. Type not sufficiently defined, but appears to be ateleiotic.
42	Domenichino.	1581—1641	Meeting of St Nilo with the Emperor Otho at Gaeta.	Grotta Ferrata, Greek Monastery of Basilians.	Head and left shoulder of a probably achondroplasic dwarf with shield and sword of Emperor to left centre of picture.
43[a]	Mytens, Daniel.	1590—1658?	Jeffrey Hudson, with Charles I and Henrietta Maria, horses, etc. He is holding dogs in a leash, c. 1627.	Buckingham Palace, London.	Ateleiotic dwarf. Once the Earl of Dunmore's; purchased for Queen Victoria at Christie's.
43[b]	,, ,,	,, ,,	,, ,, ,,	Lord Derby at Knowsley.	? Original of 43[a] and 43[c]. Background less detail, said to have been presented by Charles I. to 7th Lord Derby.
43[c]	,, ,,	1590—1660	Jeffrey Hudson, with Charles I and Henrietta Maria, horses, etc., Hudson is holding dogs in left-hand corner of picture.	In the possession of Viscount Galway, Drawing Room, Serlby Hall, Bawtry, Yorks.	The picture "belonged to Queen Anne, was given by her to Addison, then passed to Lady Warwick, his wife, at Holland House and so to Serlby" (Letter of Lord Galway, August 7, 1911).
44[a]	,, ,,	,, ,,	Jeffrey Hudson, in a red dress (? 1630).	Hampton Court.	Probably the best illustration of J. H.'s ateleiosis.
44[b]	,, ,,	,, ,,	Jeffrey Hudson, with a dog, under trees.	This is said in works of the 18th century to be in St James' Palace, London.	It is, perhaps, the picture now at Hampton Court as there is no such picture at St James' Palace (Letter from the Comptroller, August 8, 1911).

SECTION I. *continued*

	Artist	Dates of Artist	Subject	Place	Description of Dwarf
45	Mytens, Daniel.	1590—1660	Jeffrey Hudson with a dog, 1637.	National Portrait Gallery, London.	Once in possession of Sir Ralph Woodford, the picture reproduced by G. P. Harding and engraved by Snow for the *Biographical Mirror*: see No. 150, and our Plate HH (66).
46*	?	„ „	Queen Henrietta Maria and Jeffrey Hudson	In the collection of Earl Fitzwilliam.	No reproduction known. A picture, said to be in the collection of "Lord Milton" by writers of last century, no doubt refers to Earl Fitzwilliam's picture. See *Corrigendum*, p. 470.
	[N.B. A picture of Hudson and the King and three pictures of Hudson and the Queen, still unlocated, were painted by Mytens: see C. Stopes: "Daniel Mytens in England," *The Burlington Magazine*, Vol. XVII., pp. 160—3, London, 1910.]				
47	Callot, Jacques.	1592—1635	Le Pont Neuf de Paris.	Accademia di Belle Arti, Venice.	Achondroplasic dwarf to left centre of picture.
48	„ „	„ „	La Foire de l'Imprunette.	Accademia di Belle Arti, Venice.	Several dwarf-like figures, but dimensions of photograph we have been able to examine do not permit us to assert their actual dwarfism, much less suggest its type.
49	Vandyck, Sir Anthony.	1599—1641	Queen Henrietta Maria and Jeffrey Hudson, c. 1632.	Northbrook Collection.	Jeffrey Hudson is represented with a monkey on his shoulder looking up at the Queen, who has her hand on the monkey. At Petworth is a copy of this by Chas. Jervas (1675—1739).
50	„ „	„ „	Mary, Duchess of Richmond, and Mistress Gibson.	Pembroke Collection.	Mrs Gibson is handing a pair of gloves to the Duchess. She looks like a child and her long dress completely conceals her figure. She appears ateleiotic.
51*	„ „	„ „	Portrait of Richard Gibson.	Said to have been once in the possession of Sir W. Hamilton.	Ateleiotic dwarf. Further Gibson portraits are referred to in *Dict. Nat. Biog.*, but are not traceable.
52	Velasquez.	1599—1660	Un nano (El Primo).	El Prado, Madrid.	An achondroplasic dwarf: see Plate VV.
53	„	„ „	Un nano (Sebastiano de Morra).	„ „	„ „ „ „
54	„	„ „	Un nano (Antonio l'Inglese).	„ „	„ „ „ „
55	„	„ „	Il Bimbo de Vallecas.	„ „	? Myxoedematous dwarf.
56	„	„ „	Las Meninas.	„ „	To right of picture an achondroplasic female dwarf, Maria Barbola, and an ateleiotic male dwarf, Nicolasino Pertuseno: see Plate VV.
57	„	„ „	Il Bobo de Coria.	El Prado, Madrid.	? Achondroplasic. As far as the face is concerned this dwarf looks like an older stage of No. 54.
58*	„	„ „	The dwarf Barbola.	Musée d'Auch.	Garnier, *Les Nains et les Géants*, Paris, 1884, describes this picture as follows: "La naine Barbola y est représentée de face de grandeur naturelle, et tenant un petit chien sur son bras droit. Ses cheveux tombent dans toute leur longueur.... Elle est affreusement laide." (Not reproduced.) Cf. No. 55.
59	„	„ „	Portrait of a Spanish dwarf. [illegible]	[illegible]	

60	Velasquez	1599—1660	Don Balthazar Carlos and his dwarf	Castle Howard, Seat of the Earl of Carlisle.	Attended.
61	Molinaer, Jan Miense.	1610—1668	L'Atelier du Maitre (1631).	Berlin.	Typical achondroplasic dwarf dancing with a dog in centre of picture.
62	Van Ostade, A.	1610—1685	A Village Dentist.	Vienna.	An achondroplasic (? infantile myxoedematous) dwarf holds basin to left of picture.
63	Teniers, David.	1610—1690	The Temptation of St Anthony.	El Prado, Madrid.	Dwarf-like figures, type indefinite.
64	Carreno di Miranda, Juan.	1614—1685	Ragazza gigantesca.	,, ,,	See Bibl. 14[a] for contemporary description. The type appears to be one not yet classified, but comparable with that of Carrie Akers (see our p. 361) and of Barbino. See Bibl. No. 14[a] and Icon. No. 118[b].
64 *	,, ,, ,,	,, ,,	La monstreuse nue.	Royal Palace, Madrid.	A painting in the nude of the female dwarf recorded in 64[a] mentioned by Widdrington in *Spain and the Spaniards in* 1843, Vol. II. p. 20, 1844, as the portrait of a nude female dwarf represented as Silenus and wrongly attributed by him to Velasquez.
65	,, ,, ,,	,, ,,	La Regina Maria Teresa.	Cook Gallery, Richmond.	Dwarf in background, type uncertain.
66*	Lely, Sir Peter.	1618—1680	The dwarf Artist Rich[d] Gibson and Anne Shepherd his wife, hand-in-hand.	?Originally in the possession of the Earl of Pembroke.	Both probably ateleiotic. Not traceable. Lely painted other portraits of both dwarfs, also not traceable.
67*	,, ,,	,, ,,	Gibson and his master Francisco Clein as archers.	Originally in possession of Mr Rose, Gibson's son-in-law.	Gibson was probably ateleiotic. Not traceable.
68	Verwilt, Franz.	1623—1691	Portrait of an Admiral's son.	Amsterdam.	The nature of the dwarfism is not clear. It might be ateleiosis or a mild form of infantile myxoedema.
69	Fyt, Jan.	1625—1671	Dog, boy and dwarf.	Gemäldegalerie, Dresden.	Dwarf probably of infantile myxoedematous type.
70*	Giardano, Luca.	1632—1705	Dwarf.	Fresco, Escurial, Madrid.	See *Charcot et Richet*, p. 44.
71	Steen, Jan.	1626—1679	La Ménagerie.	Hague Museum.	A dwarf, possibly rickety, to left.
72*	?	?	The dwarf Michele Magnan.	Museo Civico, Bologna in 1642.	Ateleiotic dwarf, see Bibl. No. 8.
73	Bocchi, Fausto.	1659—1742 (circa)	Fight of Dwarfs with a chicken.	Palazzo Martinengo, Brescia.	Large number of "fancy" dwarfs. The artist used largely the achondroplasic type as his models.
74	,, ,,	,, ,,	Village Fête of Dwarf-folk.	,, ,, ,,	
75	Tiepolo, Giovanni Battista.	1696—1770	Banquet of Antony and Cleopatra. Tiepolo called it Aeneas and Dido.	Fresco in the Palazzo Labbia, Venice.	A dwarf of quite uncertain type ascends steps in centre of picture.
76	,, ,, ,,	,, ,,	The Feast of Cleopatra.	Hermitage, St Petersburg.	A dwarf, possibly of achondroplasic type, to right of picture.
77	,, ,, ,,	,, ,,	The Ceremonious Reception.	Königliche Galerie, Berlin.	A dwarf, type uncertain, to right of picture reclining and holding a dog.
78	,, ,, ,,	,, ,,	Scene from the story of Iphigenia.	Pinakothek, Munich.	Dwarf stooping, holding dogs to right of picture.
79	,, ,, ,,	,, ,,	Marriage of the Emperor Frederick.	National Gallery, London.	Dwarf kneeling to centre of picture. Nos. 75—79 look as if they had had the same dwarf for model, but in no case is it possible to determine the nature of the dwarfism clearly.
80	School of Veronese.	?	Il Doge Ziani e il Papa chiamano gli Ambasciatori per inviarli di Barbarossa.	Pal. Ducale, Venice.	Dwarf with dog to extreme right of picture, type uncertain.

SECTION I. *continued*

	Artist	Dates of Artist	Subject	Place	Description of Dwarf
81	van Kellen, Jan.	?	Two dwarfs leading a large dog.	Collection Racrynski, Berlin.	The elder dwarf is aged 22, the younger 18. Meige (*Nouv. Icon. de la Salp.* T. IX. p. 176, 1896) says the elder dwarf has rachitic curvature. He classes them as cases of myxoedematous infantilism.
82	?	?	Bébé (Nicholas Ferry) with a large dog.	Musée de Nancy, Pastel Portrait. Reproduced by Garnier, *Les Nains et les Géants*, p. 157.	Bébé is said to have shown signs of congenital syphilis combined with microcephaly and infantilism (Porak) on the authority of Richer, *L'Art et la Médecine*, p. 205. A portrait vaguely said to be 'from a' memorial card is given by Hastings Gilford, *The Disorders of Post Natal Growth and Development*, London, 1911, p. 639 (? related to Versailles portrait).
83*	Gobert.	?	Bébé (Nicholas Ferry).	Versailles Gallery.	
84	Deveria, Eugen.	1805—1865	The birth of Henry II.	Musée du Louvre, Paris.	Dwarf to centre of picture with dog, probably achondroplasic.
85	Painted by Garnier (?).	1802—1847 (circa)	Jakob Lehnen, Painter.	Reproduced in Garnier, *Les Nains et les Géants*, p. 239, Paris, 1884.	An ateleiotic dwarf.
86	Painted by Reckers; drawn by Garnier.	?	Simon Jan Paap.	Reproduced in Garnier, *Les Nains et les Géants*, p. 189, Paris, 1884.	Ateleiotic dwarf.
87	R. Horne (?).	?	Owen Farrel.	Royal College of Surgeons, London.	Achondroplasic (?) dwarf.
87b	Hulett after Gravelot.	1742	,, ,,	Hunterian Museum, Glasgow.	,, ,,
88	Painted by Jeanne; drawn by Garnier.	?	Tom Thumb in costume of Frederick II.	Reproduced in Garnier, *Les Nains et les Géants*, p. 217, Paris, 1884.	Ateleiotic dwarf.

Tapestry.

89	Queen Matilda (?).	11th century	Bayeux Tapestry with the dwarf commonly called Turold in that part of the Tapestry entitled "Ubi Nuntii Willelmi Ducis venerunt ad Widone."	Musée de Bayeux.	This dwarf does not look achondroplasic, he is represented as holding the horses of the ambassadors. The name Turold is worked above him, but probably applies to Turold, Constable of Bayeux, who is said to have been one of the ambassadors, see Comte Jules, *La Tapisserie de Bayeux*, Paris, 1878, Pl. XI.

90	Bronzino, Angiolo	1502—1572	Feast of Pharaoh in the history of Joseph.	A 16th century Florentine tapestry [illegible] in Florence. In the Museum of Industrial Art, Milan.	The dwarf, [illegible] Charcot says he looks athletic but has no deformity except a repulsive face (Charcot and Richer's *Les Difformes et les Malades dans l'Art*). He may be achondroplasic.
91	?	Middle of 17th century	Election of Cosimo di Medici I.	Villa Reale della Petraia, neighbourhood of Florence.	A macrocephalic dwarf, not of achondroplasic type, seated at the very centre of the picture.
91bis *	Flemish.	Early 16th century	Procession of women holding dwarfs in their arms and riding giraffes.	Robinson Tapestry, No. 375, 1906. South Kensington Museum.	At present (1911) under repair and not visible.

Section II. *Plastic Representations of Dwarfs.*

Egyptian[1].

92	?		Ptah-Seker-Osiris with head of Anubis.	22930 British Museum.	Achondroplasic dwarf-like figure.
93	,,		Polytheistic figure uniting Khnounkis, Anubis, Osiris, Horus, and the Soul.	1419 ,, ,,	,, ,, ,,
94	,,		Jug in the form of a woman.	30459 ,, ,,	,, ,, ,,
95	,,		Composite deity combining Ptah-Seker, Osiris, Amen, Thoth and Horus.	36453 ,, ,,	,, ,, ,,
96	,,		Ptah-Seker-Àsàr.	11157 ,, ,,	,, ,, ,,
97	,,		,,	1211 ,, ,,	,, ,, ,,
98	,,		Ptah-Seker-Àsàr and Bes.	26316 ,, ,,	,, ,, ,,
99	,,		Bes.	15291 ,, ,,	Some achondroplasic features.
100	,,		,,	22610 ,, ,,	,, ,, ,,
101	,,		Steatopygous woman forming a jug.	Museum at Cairo.	Achondroplasic type: see our Plate QQ (95).
102	,,		,, ,, ,, ,,	29935 British Museum.	Achondroplasic type: see our Plate QQ (94).
103	,,		Woman, said to be jug or vase.	E 2427 Ashmolean Museum, Oxford.	Some achondroplasic features: see our Plate QQ (96).
104	,,		Khnoumhotpu.	Boulak Museum, Cairo.	Achondroplasic looking dwarf with bathrocephalic head, see p. 35.
105	,,	B.C. 1516—1481	Queen of Punt.	Bas-relief at Deir-el-Bahari.	Must be associated with the above figures: see our Plate QQ (93a—b).
106	,,		Bronze figure of Bes.	Musée du Louvre, Paris.	See our Plate S (15), Fig. V.
107	,,		Ptah.	,, ,,	Many figures of this god as well as of Bes showing achondroplasic features are in the Musée du Louvre. See also Charcot et Richer, p. 15.
108	?		Egyptian pygmies.	Musée du Louvre, Paris.	See our Plate VV.
109	,,		Egyptian dwarfs.	Reproduced in H. Rossolini, *Monumenti dell' Egitto e della Nubia*, T. II. Pl. 93, Pisa, 1832—44.	? Type, they are deformed.

[1] Naturally this section of the Iconography is illustrative, not exhaustive.

Classical — SECTION II. *continued*

	Artist	Dates of Artist	Subject	Place	Description of Dwarf
110	Antique bronze statuettes.	?	Negro pygmies.	Collection Thiers, Louvre.	Racial dwarfs. See our Plate UU.
111	Statuette.		Roman Emperor Caracalla in caricature.		See our Plate S (15) Fig. X.
112[a]	Antique bronze statuette.	?	Dwarf.	Musée du Louvre, Paris.	Archaic bearded achondroplasic figure.
112[b]	,, ,, ,,	?	Dwarfs.	,, ,, ,,	Most lifelike figures with adult faces and more rickety than achondroplasic looking limbs. See our Plate UU.
113	,, ,, ,,	?	A Dwarf fighting. No. 131.	Collection Oppermann, Bibliothèque Nationale, Paris.	Both achondroplasic dwarfs. See our Plate UU.
114	,, ,, ,,	?	Punishment in the cangue (a kind of pillory used in China). No. 260.		
115	,, ,, ,,	Circa 300 B.C.?	Aesop as Dwarf.	South Kensington Museum.	? Rickety dwarf with marked lordosis.

Medieval

116	Laurana, Francesco.	Latter half of 15th century	Triboulet. A Medal. Only the head is given with cap of buffoon.	Reproduced in Garnier's *Les Nains et les Géants*, p. 97.	Triboulet is stated to have been a dwarf with a very small head, who lived in 1466 at the Court of Réné of Anjou, King of Sicily.
117[a]	Meit, Konrad?	1510—1530?	Painted clay bust of female dwarf (?).	Royal Collection, Windsor Castle.	Suggested as dwarf, by Bode, *Jahrbuch d. k. preuss. Kunstsammlungen*, Bd. XXIII. Heft. 4, S. IV.—XV. Berlin, 1901.
117[b]	?	Before 1560	John Jarvis, painted wooden statue, dwarf page of honour to Queen Mary.	Once in possession of George Walter, Esq. Now? Reproduced in Caulfield's *Portraits*. See No. 149.	Ateleiotic dwarf. Not traceable.
118[a]	Bologna, Giovanni da.	1524—1608	Equestrian statue of Cosmo I. One of the bas-reliefs has a dwarf in it to right centre.	Florence.	There can be no doubt that Nos. 118[a]—118[c] are the same myxoedematous dwarf. Meige (*Nouv. Icon. de la Salp.* T. IX. p. 176, Paris, 1896) says No. 118[b] is a case of myxoedematous infantilism. He has a prominent forehead, flat nose, thick lips, pendant cheeks covered with a short beard. Body a mass of fat. Cf. Carrie Akers (p. 361) and the Ragazza Gigantesca, No. 64[a]: see our Plate TT.
118[b]	Cioli, Valerio.	1529—1599	Dwarf Barbino on a bronze fountain.	Museo Nazionale, Florence.	
118[c]	Cioli, Valerio?	,, ,,	Bronze Dwarf Morgante, upright, blowing horn in left hand and with a stick in right.	South Kensington Museum. Another in Berlin, and several at Florence (Bargello).	
119	? (Small sculpture).	?	Stone effigy of Jeffrey Hudson, c. 1640.	Formerly in Newgate Street over entrance to Bagnio Court, accompanied by W. Evans the gigantic porter to Charles I.	Ateleiotic dwarf. See Pennant's *Account of London, Westminster and Southwark*, London, 1793, 3rd Edn., p. 244.
120[a]	Paduan master.	circa 1500	Two statuettes of the same nude dwarf.	K. K. Hofmuseum, Vienna.	Extremely stout, short arms and legs, latter not bowed. Genital organs not fully developed. Probably myxoedematous. Cf. Bode and Marks, *The Italian Bronze Statuettes of the Renaissance*, Vol. I. Plate LXIII.,
120[b]				Museo Sforzesco, Milan.	

121	Jeannet (a surgeon).	1764	Nicolas Petit. Bust.		dressed in his clothes with wig of his hair. See our Plate RR (98).
121bis	Chelsea Porcelain.	circa 1765	Male and female dwarfs.	Schreiber Collection, No. 167, South Kensington Museum.	Grotesque male dwarf, uncertain type; (?) ateleiotic female, expression cretinous.
122	Bonomi, Joseph.	1796—1878	The dwarf Joseph Boruwlaski.	University of Durham (1837).	Ateleiotic dwarf: see our Plate II (67) and (68).
123	Richer, Paul.	Last quarter 19th century	Statuette of a boy aged 19. 103 cm.	Modelled about 1898.	Meige (*Nouv. Icon. de la Salp.* Vol. XI. p. 136, Paris, 1898) says this is a case of myxoedematous infantilism.

Section III. *Prints and Books.*

124	J. Wierix.	2nd half 16th century	Feast of Dives, with Dwarf.	Jan Wierix's *Bible*, 1594.	The dwarf is teasing a monkey and looks rather achondroplasic, the arms seem short but the curvature of the legs may be due to his position.
125	De Malery, C.	,,	Prodigal Son, with Dwarf.	Jan Wierix's *Bible*, 1594. The dwarfs in these engravings are reproduced in Hone's *Year Book*, London, 1838, pp. 16—19, and p. 66.	The dwarf is represented with fool's cap and bells mocking the Prodigal Son. He appears well proportioned and looks like a boy—probably from an ateleiotic model.
126	Lycosthenes.	?	Giant and dwarf of Nicephorus Callistus.	Reproduced in Garnier, *Les Nains et les Géants*, 1884, p. 168.	
127	?	?	Miniatures of Rich[d] Gibson and his wife.	? Reproduced in Walpole's *Anecdotes of Painting*, 1849, Vol. II. p. 533.	Ateleiotic dwarf. See Fig. 699, text p. 360 and No. 185 below.
128	?	?	Mute and Dwarf standing side by side.	See Paul Rycaut's *The Present State of the Ottoman Empire*, London, 1668, p. 24.	Dwarf does not appear achondroplasic but has a large head.
129	Le Clerc, Sebastian.	1637—1714	Muets et Nains du Grand Seigneur.	Rycaut's *Histoire de l'état présent de l'Empire Ottoman*, 1670, trans. from the English.	This picture is not a copy of the English original and is inferior to the English. Another version appears in the Amsterdam French Edn. of 1678.
130	Gole, J.	1660—1737	"John Worrenberg."	Mezzotint Engraving, 1689, Print Room, British Museum. Short inscriptions in French and Dutch.	Portrait of achondroplasic (?) dwarf and normal man.
131	Oliver, John.	1688	"The true Portraiture of John Wormbergh."[1]	Engraved about 1688, Print Room, British Museum.	Dwarf aged 38, carries a sword and stick and is elaborately dressed. Long inscription in English, which among other matters states he was 2′ 7″ high.
132	Schenk, P.	1645—1715	"Hans Worremberg."	Whole length mezzo-tinto portrait, Print Room, British Museum.	This dwarf is still more elaborately dressed than in No. 131. In the pictures this dwarf looks clumsily made and appears to have no neck. He looks something like Wybrand Lolkes. His height is small for achondroplasia and type hard to define.

[1] An apparent variant of this simply entitled: "J. Worrenburg, The Swiss Dwarf" is in Eugenics Laboratory.

SECTION III *continued*

	Artist	Dates of Artist	Subject	Place	Description of Dwarf
133*	Drapentier, J. or Drapentière, J.	c. 1674	Portrait of Wormberg.	England? With eight lines of Dutch and four of English. Mentioned by Wood, *Giants and Dwarfs*, London, 1868, p. 304.	Type? No copy seen.
134*	?	About middle of 17th century	Gomme Lapon.	In possession of E. J. Wood in 1868.	E. J. Wood in *Giants and Dwarfs*, 1868, pp. 285, 286 says "An ancient and very rare foreign engraving in the possession of the author represents the full-length figure of a dwarf standing on a chequered floor. He has a large head, which is bald, a wide open forehead, a small moustache and a long beard. He wears a short braided tunic, baggy breeches, stockings and buckled shoes. His hands are clasped in front of him and he has the appearance of a man in thought. A superscription in French tells us that his name was Gomme Lapon and that he 'est habitant des frontières des sauvages': his age was about 110 years (!) and his height 2 ft. 4 inches: he was very well shaped and proportioned 'joli de figure' and his white beard was more than a foot long. This engraving is undated but we conjecture that it was issued about the middle of the 17th century."
135*	Alessandri.	1740—?	French engraving of the dwarf Ackenheil. It is undated.	In the possession of E. J. Wood in 1868. A copy is in the *Collection Hennin* according to Garnier.	Wood, *Giants and Dwarfs*, p. 379. "Engraving represents a neat and pretty male dwarf dressed in military costume and wig, holding in his left hand a plumed hat and standing on a terrace beside a flag and drum. In the foreground lie a sword, bayonet and knapsack. The superscription in French tells us that his name was 'Akeneil,' and that he was born in Germany in the Black Forest. He was 15 years of age, 30 inches in height and had not grown taller since he was 5 years old. He was very beautiful, gay, lively and active and he learned easily and retained his knowledge. About May 1788, this dwarf was exhibited in Paris."

	(the Elder).		cession at the Coronation of Cosimo di Medici.	*Nouv. Icon. de la Salp.* 1901, T. XIV. p. 372.	dress. One who is partly hidden looks well proportioned, the other is called an achondroplasic dwarf by Meige, who describes him as "trapu, les jambes fort arqués mais surtout on remarque l'exiguité de ses bras." Latin inscription under engraving.
137	Philip Galle, after Jan van der Straet.	1537—1612	Coronation of Cosimo di Medici by Pope. Dwarf to centre of picture.	Print (1583) with Latin inscription. South Kensington Museum (95. G. 36).	Achondroplasic dwarf.
138	Nilson, J. E.	1721—1788	The dwarf Catherina Helena Stöberin.	Portrait done in 1775. South Kensington Museum (G.6a).	Ateleiotic dwarf. 2 ft. 4 ins. at 16·5 years.
139	Le Bas, M. O.	1783—1843	The dwarf Mons. La Grandeur.	Engraving reproduced in Garnier, *Les Nains et les Géants*, 1884, p. 193.	
140			"Madame" Bébé and her sister.	Lithograph in *Le Théâtre Comte.* Reproduced in Garnier, p. 67.	Ateleiotic dwarf.
141	?	?	Thérese Souvray, betrothed to Bébé.	*Dict. des Sci. Méd.* T. XXXV. 1819.	Ateleiotic dwarf: see our Plate II (70).
142	R. Page?	?	Dwarf Bertholde of Berganoma.	Engraved from original picture (?). In Eugenics Laboratory.	Achondroplasic Dwarf: see Wilson's *Wonderful Characters*, Vol. I. p. 453.
143[a]	C. F. Fritsch.	?	Dutch engraving, Wybrand Lolkes.	Print Room, British Museum.	Achondroplasic dwarf.
143[b]*	Wilkes. Published C. Johnson.	Last half of 18th century	Wybrand Lolkes and his wife.	? Copied in *Le Magazin Pittoresque*, Paris, 1839, p. 333; Smeeton's *Biographica Curiosa*, London, 1822, p. 38 and Wilson's *Wonderful Characters*, London, 1821—22, Vol. II. p. 375.	Achondroplasic dwarf. See Wood, *Giants and Dwarfs*, p. 383.
144[a]	Van Assen, A. Hendschel. Published by Boruwlaski, 1788.	1787	Dwarf Joseph Boruwlaski in uniform with sword holds a bird on his hand towards baby held by lady with large hat (B., wife and child).	Print Room, British Museum.	Ateleiotic dwarf.
144[b]	Van Assen. Engraved by R. Cooper. Published by Robins and Co.	1822	Practically same engraving as above.	" " " (Also in Eugenics Laboratory).	" "
145	Van Assen, A., delin. et sculpt. Published by Boruwlaski.	1788	Very similar to the above two, except that B. rests his elbow on a table, and the lady has discarded large hat for a floral decoration.	Print Room, British Museum.	" "
146[a]	I. Downman, A.R.A., delt. W. T. Fry, sculpt. Published by George Andrews, Durham.	1821	Joseph Boruwlaski. "The little Count was born in Polish Russia, 1739, married in 1780, he has three children." Hatless, ordinary costume, showing to waist, holding up one finger and addressing a lady in profile.	" " "	" "
146[b]	Published by James Dundee.	1809	Mr O'Brien and Count Boruwlaski (Giant and Dwarf).	Origin? Print in possession of Eugenics Laboratory.	? if really Boruwlaski although so entitled. See *Eccentric Mirror*, Vol. I. No. 2, London, 1813.
147	?	?	Dutch Engraving of Jean Hannema ('Admiral van Tromp').	Reproduced in Garnier, *Les Nains et les Géants*, Paris, 1884, p. 225.	See Fig 735. Probably ateleiotic.

SECTION III. *continued*

	Artist	Dates of Artist	Subject	Place	Description of Dwarf
148*	Mills, J.	Early part of 19th century	Engraving of Thos. Allen and Lady Morgan. 1803.	Copy in the possession of E. J. Wood in 1868.	They are represented holding each other's right hand. Thos. Allen was aged 35 and was 3 ft. 3 in. high. Lady Morgan, aged 45 was 3 ft. in height.
149	Painted Walker, engr. Clamp.	1795	John Jarvis, picture of a statue. From the original statue in the possession of George Walker, Esq. Published by J. Caulfield, March 1, 1796.	Caulfield's *Portraits, Memoirs and Characters of Remarkable Persons from the reign of Edw. III. to the Revolution*, Vol. I. p. 8, London, 1813. Print Room, British Museum (C. X, P. 2, 1851. 3. 8. 363).	He was page of honour to Queen Mary. Height 3 ft. 8 in. Died 1558 or 1560, aged 57. The statue was carved in oak and was for 200 years in the family of George Walter, Winchester Row, Lisson Green. He is an ateleiotic dwarf or a case of infantilism. Probably ateleiotic. See No. 117[b].
150	Painted Mytens, engr. G. P. Harding and James Slow.	Dec. 1, 1810	Jeffrey Hudson, with dog, from Woodford picture. See No. 45.	Print Room, British Museum (C. X, P. 5, 1851. 3. 8. 334).	Ateleiotic Dwarf: see our Plate HH (66).
151	Engraved by J. Droeshout.	1596—1652	Jeffrey Hudson, standing beside a table with a curtain behind to left of picture. Latin lines above and below.	Print Room, British Museum (and Eugenics Laboratory).	Miniature copper engraving. *New Year's Gift by Microphilus*, 1636. Far the best of all the Hudson prints.
152	?	?	Another copy with verse: "Gaze on with wonder and discerne in me, The abstract of the Worlds Epitome," and Latin lines.	Print Room, British Museum (as in No. 150).	A copy in Granger, Vol. II. p. 403, 1792. Also copied by Trettel in broad frame.
153	?	?	John Wormberg.	Caulfield as in No. 149.	Achondroplasic dwarf.
154	?	?	Nannette Stocker and Johann Hauptmann.	Kirby's *Wonderful and Eccentric Museum or Magazine of Remarkable Characters*, Vol. v. p. 228, London, 1804—1820.	See our Plate II (69). Ateleiotic Dwarfs. Nannette Stocker was 33 in. and Hauptmann 36 in. high.
155	H. Gravelot.	c. 1742	"Owen Farrel, the Irish Dwarf." Inscribed to Cromwell Mortimer by James Hulett. O. F. stands in street with a staff in his hand, two boys behind point at him and three figures with a wheelbarrow looking at him.	Print Room, British Museum (C. X, P. 8, 1860. 6. 23. 12). There are several variants of this. Reproduced Kirby as in No. 154. Vol. v. p. 364.	See our Plate KK (74). Probably achondroplasic, but this has been questioned. His skeleton which was preserved would settle the question, but it is untraceable.
156	?	?	Simon Paap (with the Giant James Toller).	Kirby: see No. 154, Vol. II. p. 145.	See our Plate RR (98). ? Myxoedematous Dwarf.
156[a]	?	?	George Romondo.	Kirby: see No. 154, Vol. III. p. 113.	See our Plate KK (73). Rickety Dwarf.
157	?	1815	Gulliver in the Island of Giants.	Engraving. Reproduced in Garnier, *Les Nains et les Géants*, Paris, p. 205.	Gulliver and 3 dwarfs.

158	B. Smith Pinx., H. Burgh fecit.	?	Owen Farrel standing with staff in one hand and hat in other. In two copies, in one is written "The celebrated Irish Dwarf of a surprising strength." The other is *reversed* and has printed on it "The Strong Dwarf."	Print Room, British Museum (C. X, P. 8), Eugenics Laboratory. Caulfield's *Portraits, Memoirs and Characters of Remarkable Persons from the Revolution to the reign of George II.*, Vol. III. p. 230, London, 1819.	Probably achondroplasic. Both these as well as No. 155 differ from the Kirby print: see No. 155.
159	?		Little Will at the Turk's Head Coffee House.	Print Room, British Museum (C. X, P. 8). Reproduced in Caulfield (see No. 158).	Described as a perfect Ragoten, of a squat figure, large head and very clumsily limbed, yet a man of sound sense and discernment. The figure is certainly not typically achondroplasie. It has short legs, but very long arms; head looks full size and trunk nearly full size; head not least resemblance to usual achondroplasic type; chin is slightly receding and large nose. Legs are hidden by apron so nature cannot be considered.
160[a]	?	?	Cromwell's Porter and Jeffrey Hudson.	Folio copper engraving without letters or date. Eugenics Laboratory.	Giant and Ateleiotie Dwarf.
160[b]	R. Page.	?	Jeffrey Hudson and Charles I.	Wilson's *Wonderful Characters*, Vol. I. p. 88 and p. 216, Vol. II. p. 375, Vol. III. p. 385, London, 1821—22.	Possibly a reproduction of some unlocated picture. Ateleiotie Dwarf.
161	,,	1822	Jeffrey Dunstan, Mayor of Garrett. (Probably also as an independent engraving. Copy in Eugenics Laboratory.)		He was dwarfish in size and knock-kneed and his head was disproportioned to his body. He looks rickety.
162	R. Cooper.	Last half of 18th century	Wybrand Lolkes and his wife.		Copied (?) from Wilkes' engraving, see No. 143.
163	Van Assen.	,,	Boruwlaski, wife and child.		Reproduced from van Assen's engraving, see No. 144[a].
164	R. Cooper.	,,	Wybrand Lolkes and his wife.		Copied (?) from Wilkes' engraving, see No. 143.
165	Van Assen.	,,	Boruwlaski, wife and child.	Smeeton's *Biographica Curiosa*, pp. 1, 38, 75, 205, 235, London, 1822.	Reproduced from van Assen's engraving, see No. 144[a].
166	?	?	Jeffrey Dunstan.		Same picture as is given in Wilson.
167[a]	?	?	Simon Paap.		Full-length picture: cf. our Plate RR (97).
167[b]	Worlidge?	c. 1773	Madame Teresia, the Corsican Fairy.	? Copied by R. Cooper.	Ateleiotie Dwarf, height 34 inches.
168	?	?	Tom Thumb and his carriage.	Lithograph of the time. Reproduced in Garnier, *Les Nains et les Géants*, Paris, 1884, p. 211.	Ateleiotie Dwarf. Cf. our Plate AA (43).
169	?	?	Part of the Piazza San Marco with a dwarf.	Yriarte's *Venise, Histoire, Arts, Industrie, La Ville, La Vie*, Paris, 1877, p. 141.	Possibly a photo or picture done by Yriarte.
170*	?	1st half of 19th century	Robert Skinner and his wife Judith standing one on either side of a tall man.	A rude woodcut referred to by Wood: *Giants and Dwarfs*, London, 1868, p. 351.	Probably ateleiotic. See our p. 361.

SECTION III. *continued*

	Artist	Dates of Artist	Subject	Place	Description of Dwarf
171[a]	? (G. Wright? R. Hancock?)	No date	"Edward Scofield," 3 ft. 2 in. high. Deputy clerk of St Chads, Shrewsbury.	Print Room, British Museum (C. X, P. 9, 1851 . 3 . 8 . 627).	This dwarf is probably ateleiotic, but he has on a long coat, and no certain diagnosis can be made.
171[b]	G. Beckham.	Reign of George II.	Cornelius Caton, of the White Lion, Richmond.	Copy in Eugenics Laboratory.	Possibly achondroplasic. C. J. Caulfield's *Portraits*, Vol. III. p. 173.
172	S. I. ?	1787	Kelham Whitelamb, 34 inches high, born at Wisbech in Cambridgeshire. Standing beside his "cupboard."	Print Room, British Museum (C. X, P. 9, 1851 . 3 . 8 . 719).	Type doubtful.
173	Printed by Rowney and Forster.	?	John Tarr, aged 67 years, 4 ft. 3 in., born at Bampton. No date.	Print Room, British Museum (C. X, P. 10, 1851 . 3 . 8 . 673).	Appears to be achondroplasic. Print says he was 15 years ostler at the White Horse, 12 years Boots at the Three Tuns, Tiverton.
174	Published by H. Rowe.	1821	Andrew Whitson, born Feb. 10, 1770, died 1826.	Print Room, British Museum (C. X, P. 10, 1851 . 3 . 8 . 723).	A crippled dwarf. ? A case of birth palsy.
175	Painted by J. H. Desvignes. Engraved by Charles Hunt, published by J. Moore.		The Miniature John Bull, etc. (see our p. 363).	Print Room, British Museum (C. X, P. 12, 1872 . 10 . 12 . 4455).	Probably ateleiosis.
176	Drawn on stone from Nature by A. Lambert, printed by R. Martin.	1840—1860	George Trout, one of the Porters to the Honourable, the House of Commons.	Print Room, British Museum (C. X, P. 12, 1851 . 3 . 8 . 653).	Probably achondroplasic.
177	A. Walker, sculpt. (in reign of Charles I.).	?	Richard Gibson, with a small portrait of Mrs Gibson in corner.	*Collectanea Biographica*, Vol. XLII. No. 45.	Adult face without hair; long hair on head in Stuart fashion, collar hides neck, bridge of nose not sunk. Probably ateleiotic.
178[a]	Anon.	After 1709	The wonderful and surprising English dwarf, 2 ft. 8 in. high. Born at Salisbury, 1709. Has been shown to most of the Nobility and Gentry of Great Britain.	Print Room, British Museum (C. X, P. 7, 1872 . 10 . 12 . 4339).	Probably ateleiotic, female dwarf, features of condition not well shown.
178[b]*	Anon.	First half of 18th century	The wonderful, strong and surprising Persian Dwarf, 3 ft. 6 in. high, etc.	Copy recently on sale in London.	Type unknown, bandy legged. Cf. Wood, *Giants and Dwarfs*, p. 310.
179	Engraved by James Roberts, sold by Hawksworth and by H. Roberts.	Published 1771	The Exact Representation of those two very Remarkable Persons, Mr Bamfield the Staffordshire Giant and Mr Coan the Norfolk Dwarf, etc.	Print Room, British Museum (C. X, P. 9, 1851 . 3 . 8 . 73).	Coan was probably ateleiotic. The print states that it is drawn by Mr Rackstraw's permission, as he has moulded them from the life, and that the dwarf did not exceed 3 feet and died at 36 years of age. His later days were spent in Chelsea.
180	Published by John Bowles.	First half of 18th century	Grotesque Pigmy Figures.	South Kensington Museum (95. H. 13).	Folklore Dwarfs, male and female, in a series of 12 plates, entitled: *The Lilliputian Dancing School, etc.*

181	Mantegna Andrea.	1431—1506	Dwarf handing a packet to one of two men engaged in conversation. A boy stands watching.	The original is said to be in a Collection at Padua. It is reproduced in Charles Yriarte's *Venise, Histoire, Art, Industrie, La Ville, La Vie*, Paris, 1877, p. 141.	The dwarf is not deformed, but the head is slightly too large for the body.
182[a]	Callot, Jacques.	1592—1635	Etchings of single dwarfs.	These etchings are in the Print Room, British Museum and are numbered 748, 749, 752, 753, 754, 756, 758, 761, 763 in Meaume's Catalogue.	Deformed dwarfs with short legs. Probably models were achondroplasic.
182[b]	Rubens, Peter Paul.	1577—1641	Drawing of Earl of Arundel's dwarf Robin.	National Museum, Stockholm.	Probably ateleiotic. Sketch for or ? *from* No. 41.
183	Persian artist.	16th—17th century	Drawing of dwarf. Old man with white beard, bent with age or stooping to the dwarf.	Original ? Reproduced in Charcot and Richer, *L'Art et la Médecine*, Paris, 1901, p. 251.	Ateleiotic Dwarf.
184	Tiepolo, Giovanni Battista.	1693—1770	Two studies of male and female dwarfs and dogs.	Reproduced in C. Jacobi's *Acqueforti dei Tiepolo Collezione di* 100 *Tavola*. Plates 90 & 91. Ongagia's Edition, Venice, 1879.	These dwarfs are very similar to those in his pictures and are in semi-recumbent positions : see Nos. 75—79.
185	?		Water colour in sepia. King in crown and robes uniting hands of dwarfs ; above oval bust portraits of Richard Gibson and his wife.	Royal Library, Windsor.	Ateleiotic Dwarfs. Seems to be original of No. 127.
186	Richard Gibson.	?	Painting of Gibson by himself.	Print Room, British Museum.	Neck looks short and face appears younger than in No. 127.
187	Du Plessis (James Paris).	1730—1733	John Grimes.	3253. Sloane Manuscripts, British Museum.	John Grimes of Newcastle-on-Tyne, aged 57, 3 ft. 8 in. in height. He was a short and very thick man as broad as he was long from hand to hand. He died 1736, was dissected and his skeleton set up. The picture represents him fully clothed, he appears well-proportioned with straight legs, but he has no beard and has a boyish appearance.
188	„ „ ,	„ „	Anne Rouse.	5246. Sloane Manuscripts, British Museum. A Short History of Prodigious and Monstrous Births, of Dwarfs, Sleepers, Giants, Strong Men, Hermaphrodites, Numerous Births, and Extreme Old Age.	"Anne Rouse, born near the city of Norwich, 24th June, 1690, aged 27, but 2 ft. 2 in. high. Being very well-shaped, well-proportioned and very strait." In the picture the legs look rather short and the arms rather long for the body.

SECTION IV. *continued*

	Artist	Dates of Artist	Subject	Place	Description of Dwarf
189	Du Plessis (James Paris).	1730—1733	John Worrenbergh or Wormberg.	5246. Sloane Manuscripts, British Museum. A Short History of Prodigious and Monstrous Births, of Dwarfs, Sleepers, Giants, Strong Men, Hermaphrodites, Numerous Births and Extreme Old Age.	He was of Hartshousen, in Switzerland, aged 39 and 2 ft. 7 in. in height and was seen by Paris, 1689. "He was as big in all his members as any grown man and as strong." The legs look very thick but are not short in proportion to the trunk. The arms do not look short. See Nos. 130—3.
190	,, ,, ,,	,, ,,	A dwarf black man mounted on dwarf horse.	Ditto. (Cf. also Granger, *A Biographical History*, Vol. III. p. 48, London, 1806, who refers to *Spectator*, No. 271.)	He was but 3 ft. high and aged 32 when Paris saw him in London, 1712. "He was strait, well-shaped and proportionable, he had a wife which was not 3 foot high, at 30 years of age, strait and proportionable as any other woman, she c^{d} dance extraordinary well, though big with child, and also the little Turkey horse that was but 2 ft. odd inches high and above 12 years old." Paris also says that in 1715 he saw a little black man 3 feet high and 25 years old.
191	,, ,, ,,	,, ,,	Hannah Weston.	Ditto.	"She was born at Leeds in Yorkshire in 1685, was 20 years of age and but 2 ft. 2 in. high, very straight and well-shaped, she c^{d} sing dance and play with the castanets excellently well." Represented fully clothed.

Addendum. No. 35*. Engravings from Jan van der Straet's pictures (besides Nos. 136 and 137) frequently show dwarfs. Thus in the Print Room, British Museum we find: (1) By P. Galle, *Christ before Pilate.* Dwarf with helmet holds Christ's robe, thick-set, but hardly achondroplasic. (2) By C. and T. Galle, *Herod and Salome.* In right-hand corner, achondroplasic dwarf with cap, sword and extended arm. (3) By A. Collaert, *Christ in the Manger.* Right-hand corner, small figure blowing a horn, might be an ateleiotic dwarf. (4) By Collaert and Galle, *Christ coming away from Pilate.* Two dwarfs, the first, possibly achondroplasic, holds a wand in one hand and points jeeringly at Christ with other; the second, much taller but still a dwarf, has a sword and holds the rope which binds Christ's hands.

Corrigendum. No. 46*. Just as this sheet goes to press a letter has been received from Mr A. W. Drury stating that Lord Fitzwilliam's picture is a full length portrait of Queen Henrietta Maria with the Dwarf by her side and that it was painted by Vandyck. A copy of the same is in the possession of Mr George C. Wentworth Fitzwilliam at Milton. Cf. No. 49.

PEDIGREES.

SECTION I. ACHONDROPLASIA.

PLATE LI. ACHONDROPLASIA. PLATES LII. AND LIII. ACHONDROPLASIA AND PROBABLE ACHONDROPLASIA.

PLATE LI. Fig. 608. *Rischbieth's Case.* Fig. 620 below was given by Boeckh, and published 17 years ago (1893). There has recently been a collection of 53 dwarfs on exhibition in London, at Olympia. Amongst these were five achondroplasic individuals, three females and two males. One of the former I recognised as Elizabeth or Kathie Kipke, one of Boeckh's cases, remembering her appearance from the photograph shown in his paper. She gave me the details of the following pedigree. In order to avoid mistaken identity I then asked her:—Whether she had ever been in the Heidelberg Frauenklinik, and Whether she had ever undergone any operation for the birth of her children? To both the answer was "Yes." To the second it was "At each birth." For the first at Heidelberg. Boeckh's photograph of this woman is shown in his paper, and it can be compared with a photograph of her taken at the present day. From the resemblance of these and from the pedigree she gives (though this does not, precisely, agree with that of Boeckh) and from the family name there can be no doubt as to the identity of the individual. This pedigree does not differ in any essential from that of Boeckh (except as to the nature of IV. 12, who though a dwarf is not achondroplasic), but represents the family 17 years later. It was made January 18th, 1910. This account was given by IV. 14, Elizabeth Dorffler (*née* Elizabeth or Kathie Kipke), whose photograph, with those of her daughter, V. 8, and of her elder sister, IV. 11 (all three of whom are achondroplasic) is shown in Plates Q (9), (10) and FF (62). With them are photographs of IV. 12, husband of IV. 11, and their son, V. 6. These two are ateleiotic. IV. 14, Elizabeth Dorffler, *née* Kipke, aged 42 years, born in Pomerania. Height[1] approximately 3′ 6″, head and face disproportionately big for height. Head square, with prominence of parietal eminences. Frontal eminences less prominent than usual in achondroplasia. Bridge of nose not much depressed, nose large, with wide nostrils but not tip-tilted. Cheek-bones slightly prominent. Looks about her age. Hair of scalp not thin and in normal condition. Her arms are very short; the distal extremities of the fingers do not extend beyond the level of the region of the great trochanters as far as could be judged. The hands are disproportionately large. They are very broad, short and thick with thick fleshy fingers. The fingers show an approximation to equality in length but this is not complete. They show the hand "en trident," and the peculiar formation of the segments, as it were three cylinders of progressive diminution in diameter placed end to end. The pulp of the fingers extends beyond the nail. Gait "rolling" or "waddling." Movements fairly quick. Those of the hands look clumsy but are quite accurate. Very intelligent and quick mentally. Reads and writes well. As this individual could not be examined without her clothes no other points could be made out. (For these see Boeckh's photograph and account.) By IV. 13, a man of ordinary size, she had a female illegitimate child, V. 7, delivered by Caesarian section in Heidelberg Frauenklinik. (Boeckh's account.) She married a German, of height about 5′ 7″ and by him had three children, all born in Zürich (V. 8, 9 and 10). Of these, V. 9, and V. 10, died aged 5 months and 3 months respectively. V. 8, aged 17 years, is an achondroplasic dwarf. (Plate Q (10).) She resembles her mother in stature, length of arms, hands and in all other respects, that of age (she looks her age) and that the shape of her head is more typical, the frontal as well as the parietal eminences being prominent and the bridge of the nose somewhat depressed. The nose is large, with wide nostrils, but is not tip-tilted. She is very intelligent and reads and writes well. IV. 11, a sister of IV. 14, aged 43 years, Dagmar Huther (*née* Kipke, Plate Q (9)), of the same height and like her achondroplasic. Typical in all respects except that, like her sister, the frontal eminences are not very prominent and the bridge of the nose not much depressed. She married IV. 12, a dwarf, described by Boeckh as a true dwarf of proportioned members. His photograph is shown here and it will be seen that he shows none of the features of achondroplasia, but is an ateleiotic or true dwarf. He died aged 44 years, of some disorder accompanied by palpitation of the heart (Herzklopfen, ? exact nature of this malady[2]). V. 6, aged 18 years (the son of IV. 11 by this dwarf, IV. 12), is described as "the smallest man in the world." His height is a little over 2′. (Plate FF (62).) He has none of the features of achondroplasia, but is an ateleiotic or true dwarf[3].

[1] No measurements were permitted. The height had to be inferred by comparison with surrounding objects.

[2] Boeckh says he died, aged 47, of Herzschlag.

[3] If the account of the parentage of this individual be correct, he is unique. (1) Because he is one of the very few cases on record of a true dwarf being produced by a true dwarf parent of either sex, and (2) because one parent is achondroplasic and the other ateleiotic. An achondroplasic dwarf of either sex may produce children. These may be achondroplasic or normal. Ateleiotic dwarfs, on the other hand, are usually sterile, but have been known to produce offspring. In most recorded instances, however, where this has occurred, such offspring, if they survived, have grown to normal dimensions. The parentage of this individual may have been adopted for show purposes. The alleged father, however, has growth of hair about the face and may therefore have been potent.

IV. 11 has had no other children. IV. 9, a normally grown sister of IV. 11 and 13, is now aged 45 years. She is married to a man of ordinary size, IV. 10, and has had five children, V. 1, 2, 3, 4 and 5. Of these V. 1, 2 and 3 are sons, still living and normally grown. V. 4, a daughter, achondroplasic, died aged 3 years. V. 5, a daughter, normal, died aged 3 years. Besides these three sisters, in generation IV., there are three brothers, IV. 6, 7 and 8, and one sister, IV. 5, all living and all normal. ("Wir sind sieben, fünf gross und zwei klein.") Nothing is known of their descendants, if any. In Generation IV., also, four normal children, IV. 1, 2, 3 and 4, died in infancy. III. 8, the father of IV. 11 and 12, was a dwarf like these, but was not quite so small. He died in Berlin aged 85 years. He had two brothers and two sisters, III. 9, 10, 11 and 12, all of normal size. Nothing is known of their descendants, if any. His wife, III. 7, was of ordinary size. She had three brothers and three sisters, all of ordinary size, as were their parents, II. 1 and 2. Both the father, II. 4, and the grandfather, I. 1, of this achondroplasic dwarf, III. 8, as well as one of his aunts, II. 5, were "little" people. They were all of about the same size. "They were small but not so small as we" (Sie waren klein, aber nicht ganz so klein wie uns) and did not show the peculiar shape that her sister, her daughter and herself show. She indicates by raising her hand their supposed height which is 4′ to 4′ 6″.

Unfortunately permission was not given to make measurements, X-ray examination or scientifically accurate photographs of any of these individuals and therefore the above account is of less value than it might be. (Unpublished.)

Fig. 609. *Rischbieth's Case.* IV. 2, Maud S——, aged 15 months. Achondroplasia, one of twins, her twin brother being of normal growth. A large, square head, equal in circumference to that of her twin brother. Frontal and parietal bosses prominent. Bridge of the nose depressed, but not much flattened. Nose slightly tip-tilted. The lower part of the face compared to the forehead is small and shows fairly well the "inverted pear" appearance spoken of by French authors. The length of the trunk is about equal to that of her brother, but the limbs are markedly shortened (micromelia). The finger tips do not extend beyond the great trochanters in full extension. The shortening affects the arms and thighs more than the forearms and legs, i.e. the micromelia is of rhizomelic type. The limbs are not massive, as is the rule in achondroplasia, but are rather thin. The subcutaneous tissues, however, seem to be somewhat increased and the normal skin folds are all exaggerated. All the long bones of all four limbs are curved. The curvatures occur at the junction of epiphysis and shaft. The epiphyses are enlarged. The hands and feet are relatively short, broad and thick. The hands show, particularly well, the approximation to equality in the length of the fingers. The separation of the distal extremities of these in complete extension is also well shown, but better in the right hand than in the left. The abdomen is prominent, but there is no lordosis. On the contrary there is an angular kyphosis in the lumbar region, this is apparently painless, is not tender on palpation and there is no rigidity in it. It is a rickety curvature. But the ribs are not beaded and there are no other signs of rickets. The first tooth appeared at the age of 13 months. The infant was bottle-fed since birth. She is not a fat child, as is usual in achondroplasia. Intelligence apparently normal; she is beginning to walk and talk. IV. 3, twin brother of the last. A well grown and perfectly healthy child. No evidence of rickets. Cut first tooth at the age of 6 months. Beginning to walk and talk. Marked internal strabismus of left eye, a condition which his father, III. 2, also shows. IV. 1, born dead at full term. No peculiarity. III. 2 is of average size and is in good health. Nothing is known concerning his brethren or parents, all of whom "are living abroad." (Statement of III. 3 and II. 8.) III. 3, aged 28 years, mother of the twins, is of height 5′ 6″; she shows no peculiarities and is in good health. III. 4, 5 and 6, aged 27, 25 and 21 years respectively, are of medium height and ordinary proportions. III. 7 and 8 died aged 6 weeks and 4 weeks respectively; cause of death not known; but they showed no peculiarity. III. 9, 10, 12, 13 and 14 were all seen. They are of average stature for ages (16 years, 15, 11, 10 and 6 respectively, and show no peculiarities. III. 11 died in infancy. Cause of death unknown, but he showed no peculiarity. II. 7 and 8 were both seen. II. 7 is of almost medium height (5′ 5″) and says his brethren and parents and all known relations as shown were of ordinary size. II. 8 is a little woman of height 4′ 9″. She is fairly intelligent, though illiterate. No appearances of achondroplasia either in hands, feet, arms or legs, cranium or face. She had 15 siblings, as shown. Four were males and one female. She has forgotten the sex of others. They were all of ordinary size and proportions. I. 1, 2 and 3 were "tall" or of medium size. I. 4 was "very short and stout." I. 5 was of medium height. (Unpublished.)

Fig. 610. *Rischbieth's Case.* Louisa D——. The account of this family was given by II. 11, the mother of the achondroplasic child, III. 3. The grandparents, I. 1, 2 and 3, were "tall," I. 4 was "short," but not a dwarf. Nothing is known of the great-grandparents. But none of the siblings of I. 1 or 2, nor any children of these were dwarfs. Those described as "tall" were as tall as herself or taller. She is a big and strong woman aged 35 years, of height, approximately, 5′ 8″. She is the youngest of a family of 11 children. None of these were small or in any way peculiar or deformed. The surviving brethren are nearly all as tall as herself, or taller. II. 1, 2 and 3 died young after measles. None of the children of II. 4 to 10 are in any way like her dwarf child. (She knows all about the peculiar points of this individual, having frequently heard doctors "demonstrating" the case.) II. 14, her husband, is an ex-soldier with

several war medals. Photograph was shown. An individual of medium height and good physique. He is the third son in a family of 7 children. None of these are "tall," some are of medium height, others rather "short," but none are dwarfs or in any way peculiar in build. None of the children of these are dwarfs, or in any way peculiar. II. 11, this woman herself, the mother of the dwarf, has had no miscarriages and no illness of any kind that she remembers. She is not alcoholic and shows no signs of syphilis. The achondroplasic child was born at full term. During the later months of this pregnancy she suffered considerably from abdominal distension, which was excessive. She had no trouble of this kind in her other six pregnancies. The labour was a very easy one, but "the waters drenched everything, there were such a lot of them" (Hydramnios). All the children, III. 1 to 7, were seen and examined except III. 1, aged 15 years, who is in domestic service, and III. 5, who died aged 11 weeks of epidemic enteritis. Their ages are :—11 years, 7, 5, 3 and 1 year 3 months. The youngest is well grown, he has facial eczema, but is otherwise healthy. All the others are healthy and well grown with the exception of the dwarf, III. 3, aged 7 years. She is a typical achondroplasic child. Her sister, two years younger, of about average size for age, stands taller by a head and neck. The head is relatively large, and square. Parietal bosses very prominent, but the frontal bosses not extremely so. Bridge of nose depressed but not much broadened. Nose somewhat tip-tilted or retroussé. All the limbs massive and markedly shortened. The finger tips do not extend in full extension beyond the great trochanters, instead of, as in the normal, to mid thigh. The shortening affects the arms and thighs (proximal segments) more than the forearms and legs (mesial segments), i.e. there is micromelia of rhizomelic type. All the long bones of the limbs are somewhat curved. The curves occur at the junction of the epiphyses with the shafts, rather than in the shafts themselves as occurs in rickets. The epiphyses are enlarged. The hands and feet are short, broad and thick. The fingers are short and thick. They do not show, very well, the usual approximation to equality in length. Their distal extremities remain separated from one another in complete extension (hand "en trident") and the segments of the digits form, as it were, three cylinders, of progressive diminution in diameter, placed end to end. The subcutaneous tissues are increased in thickness, giving the child an appearance of general adiposity. All the normal skin folds are exaggerated. There is marked lordosis, the buttocks are prominent and the abdomen very prominent. The trunk, relatively to the limbs, is of great length. It shows no peculiarities, with the exception of, possibly, some slight bulging of the sternum, convexity forwards. The dentition is normal for age. The face does not show the feature usual in this condition, namely smallness of the lower part in comparison with the upper, the "inverted pear" of French writers, or as it were an inverted triangle. Intelligence normal, but the mother states that she has a peculiar disposition unlike that of her other children. She is not mischievous, but is "full of tricks," delighting in such acrobatic feats as leaping from tables and chairs and the sudden springing from hiding with a shout, in the endeavour to startle the unwary. (Unpublished.)

Fig. 611. *Rischbieth's Case.* III. 1, Lili W——, aged 25 years, Parisienne. Music Hall Singer. Height about 3′ 6″. She has a large square head with marked prominence of the frontal and parietal bosses. The bridge of the nose is depressed and flattened; the nose is tip-tilted with large nostrils. The smallness of the lower part of the face when compared with the upper, i.e. reproduction of the shape of an "inverted pear" or triangle, is in her case not very marked. The length of the trunk is that of an individual of almost medium height; mammae well developed. The arms and legs are markedly shortened (micromelia). This affects the proximal segment more than the mesial, i.e. it is of rhizomelic type. The finger tips do not extend beyond the level of the great trochanters in full extension. All four limbs are curved and very massive. The epiphyses of the long bones are all enlarged. The hands and feet are short, broad and thick. The hands show marked separation of all the finger tips when the fingers are extended "main en trident." The fingers, which are of nearly equal length, are short, and each shows all appearance as of three cylinders, of progressive diminution in diameter, superimposed, corresponding to the proximal, mesial and distal segments of the digit, a condition which has also been described as "a conical shape of the fingers." Lordosis and abdominal prominence fairly marked. Intelligence good, vivacious. She answers all questions very promptly, laughs and jokes a great deal. She has a healthy complexion and says she is never ill. She wished to know the reason for this, since nearly all the other dwarfs she knows seem to be almost constantly ailing and attending a doctor. She "volunteered" the statement that she has had no children. The following family history was obtained from her mother, II. 3, aged 58 years, a woman considerably above the average height of her sex and very intelligent. She has 14 other children all living, 7 boys and 7 girls. (The order of birth of these is not shown.) All are of ordinary growth and proportions. Some are married and have children, all these are of ordinary growth and proportions. Her husband, II. 2, is of medium height. She is quite certain that all his brethren (of whom there are several), as well as all her own, are of ordinary stature and proportions. The parents of both, I. 2, 4, 6 and 7, were, she is positive, of ordinary growth, as also were their brethren as far as she knew them. She is an intelligent woman; she says that she has frequently been questioned with regard to the family history and knows of no one amongst the number of her relatives showing the peculiar characteristics of her daughter. She wishes to know why this member of her family alone shows these peculiarities. She "has asked many doctors why it is and they cannot tell her." (Unpublished.)

Fig. 612. *Apert and Sevestre's Case.* The father, II. 1, was described in detail by Apert, and the daughter, III. 2, by Sevestre. II. 1, M. Sicard, aged 37 years, circus clown or "eccentric comic artist." Height 1 m. 29 cm., but the length of the trunk is as great as that of the average normal, being 59 cm. from the upper border of the symphysis pubis to the episternal notch—a very great length for so short a man. In the normally proportioned adult the symphysis pubis is equidistant between the vertex and the soles of the feet. In this individual the measurement from vertex to symphysis is 85 cm., which if proportions were normal would correspond to a height of 1 m. 70 cm., instead of, as in this case, 1 m. 29 cm. The measurement between the upper border of the symphysis pubis and the soles of the feet is only 44 cm. instead of 85 cm. The upper limbs show similar shortening. In the adult of normal proportions the distal extremity of the middle finger reaches to the lower third of the thigh, whereas in this individual it extends no further than the antero-superior spine. All the segments of the limb take part in this shortening, but it occurs especially in the long bones of the thigh and leg, arm and forearm; the hands and feet on the contrary are less affected. *Measurements:*—arm, forearm, metacarpus and middle finger, 18, 14, 6 and 9 cm. respectively. In an adult of 1 m. 70 cm. these measurements would be 27, 24, 8 and 12 cm. respectively. The fingers and the metacarpus are thus shortened by $\frac{1}{4}$, the arm by $\frac{1}{3}$, the forearm by more than $\frac{1}{3}$. A peculiarity observed in the fingers is their approximation to equality in length, which is almost complete, and their separation from one another. In the lower extremity measurements taken from the usual points show that the thigh and leg have each the lengths 28 cm. instead of 47 cm., and the foot, from extremity of heel to tip of great toe, 18 cm. instead of 25 cm. Thus the length of the long segments is diminished by more than $\frac{1}{3}$, the foot by nearly $\frac{1}{3}$. The head is large, not only relatively but absolutely. It is 57 cm. in greatest circumference. Very muscular indeed (measurements are here given). Intelligence equal to the average as is shown by his profession. He is an accomplished gymnast and horseback acrobat. *Skeleton.* Radiographs show the bones to be very thick. They show no curvatures such as are seen at times in rickety bones. They are short, and thickened relatively to their length. Sites of muscular origins and insertions prominent. The normal angles of bones are exaggerated, articular surfaces broadened and enlarged, not only relatively to the length of the bones but absolutely. II. 2, wife of the last. She was of average size and proportions. (The account of the family is now carried on from that of M. Sevestre published some four years later.) III. 1 was the eldest daughter of II. 1 and 2, then aged 12 years. She was bigger than is usual for her age and of ordinary proportions. III. 2, Hortense Sicard, aged 7 years. Height, very small for that of a child of her age, 87 cm., which corresponds to that of the average of $3\frac{1}{4}$ years. A very obese child, weight 18 kg. 70 cg. The reduction in height is produced more by the limbs than the trunk. The latter measures, from episternal notch to upper border of symphysis pubis, 35 cm., about the average normal for her age. But the limbs are remarkably short, especially the upper extremities. The legs are longer than the thighs. (Normally the thigh is longer than the leg.) *Measurements.* Thigh from great trochanter to external condyle, 16 cm. Leg, external condyle to external malleolus, 18 cm. The femora was abnormally curved and enlarged at its upper extremity. A similar condition is shown by the bones of the leg. The upper end of the fibula shows greater length than is normal relatively to the tibia. There is genu valgum. An analogous condition is shown in the upper extremities. The distal extremities of the fingers reach to the level of the great trochanter but no further. (In the normally proportioned individual they extend as far as the lower third of the thigh.) It is the arm, rather than the forearm, that is shortened. *Measurements.* ("From the usual points.") Arm 10·5 cm., forearm 12·5 cm. The radius and ulna are incurved and are thicker than normal in their upper parts. Movement of pronation is more complete than normal and complete extension is impossible. Hands. These are short, broad and fleshy; the fingers are short, thick, solid and sausage-shaped; they are all of about the same length, 4·5 cm. The little finger is, however, a little longer than the others. This, combined with an exaggerated obliquity of the fingers, gives the hand a peculiar, special appearance. The abdomen is enlarged. The head is very large, globular; its greatest circumference is 55 cm., the frontal and parietal bosses are markedly prominent. The face is large and square, with large features and is fairly expressive. The nose, which is large, is depressed at the bridge and flattened; its extremity is upturned; the nostrils are large. The palate is contracted, it is high arched (Gothic). The teeth are well developed and dentition is normal. Two molars are present. Two median incisors shed in the upper jaw have not yet been replaced. In the lower jaw three incisors have been shed and a fourth is very loose. Two new incisors have appeared. They are well developed. Began to walk late, at about 2 years of age. She is now firm upon her feet but has a peculiarity in her gait dependent upon genu valgum. Intelligence good and corresponds to that of a child of her age, 7 years. The accouchement had been difficult because of the size of the head which was larger than normal. *Illness.* Suppurative adenitis in the right submaxillary region at the age of 2 years. No other illnesses. The mother stated that this child's father was achondroplasic. "This statement of the mother concerning his appearance was confirmed by several of my students, who, having seen this man in certain institutions in Montmartre observed a close resemblance in it to that of this child." No note of any brethren of III. 1 and 2, nor of the parents, brethren or collaterals of II. 1 or 2. (Bibl. No. 386, p. 288, and Bibl. No. 492, p. 574.)

Fig. 613. *Guéniot and Potocki's Case.* Three accounts of this case have been given, each account

giving further details. The first two accounts were published by Guéniot see Bibl. Nos. 288 and 289. The third was given by Potocki during the discussion on Lepage's Case (Bibl. No. 474) and he then stated he had assisted Guéniot in the operations. II. 1, said that her father, I. 1, was very small, not more than 1·5 metres in height, that he was aged 74 when she was born, while her mother, I. 2, was aged only 18 or 20. The family was alcoholic. Her mother, I. 2, had subsequently several well-formed children, II. 3, by another husband. These details are given by Potocki, Guéniot's earlier accounts make no mention of them. II. 1, was achondroplasic, Guéniot in his first account calls her a rachitic dwarf. She was aged 19 when she came for her first confinement. Her height was 1·15 metres, she had a prominent forehead and incurved limbs. Caesarean section was performed 17 Dec. 1892, and a girl, III. 1, extracted who weighed 3150 grammes and had the same pathological formation as her mother, II. 1. Her forehead was prominent and her four limbs very short with their upper segments shortened like those of her mother but they were not incurved. The second Caesarean section was performed in 1893 and a boy, III. 2, was extracted who weighed 3000 grammes and was deformed like his mother and sister. The father of these children, II. 2, was tall, strong and well-formed. Potocki in the last account does not give date of the third operation which resulted in a girl, III. 3, who was not achondroplasic, but he states the girl was aged 8, so it must have been 1896. The mother, II. 1, died of peritonitis the fourth day after the last operation. III. 1, had died aged 2½ months of pulmonary congestion, but III. 2, was alive in 1904. III. 3, resembled her father. (Bibl. No. 288, p. 99; No. 289, p. 16; No. 474, p. 277.)

Fig. 614. *Lepage's Case.* II. 11, aged 23 years; married to II. 12, a man of ordinary size and proportions; when first seen was 5 months pregnant. She showed all the signs of achondroplasia, which, amongst other skeletal deformities, had produced a contracted pelvis, on account of which Caesarian section was performed. I. 2, mother of the last, a big, well-proportioned woman, gave the following family history. By I. 1, her first husband, a man of ordinary height and proportions, who died of gastric cancer, she had five births. II. 1 to 5. II. 1, was an abortion at three months and was followed, in the same pregnancy, by a small child at full term, II. 2, now aged 36 years, big and well-proportioned. II. 3, was born at full term, normal, died aged 25 years of "articular rheumatism." II. 5, born after the death of her father, a daughter, normal, died aged 28 months of "cancer of the stomach, ascites, and umbilical hernia." By her second husband, who was below average height, she has had seven pregnancies, II. 6 to 14. II. 6 and 7, were abortions[1]. II. 8, was a premature child, born at seven months, "after trauma," female, still living, "very delicate." II. 9, a male child, still living, "suffers from haemoptysis." II. 10 and 11, a twin pregnancy. II. 10, aborted at four months. II. 11, a female was born at full term, achondroplasic. II. 13, a female, and II. 14, a male, were born at full term. All the members of the second family were full sized except II. 11, above referred to as achondroplasic, now aged 23 years. At birth her head was very large, her body small. Head inclined to the side or rear. Breast fed two years. Began to walk at 1 year 8 months. At four years the marked shortening of her legs, when compared with those of children of her own age, was first observed. Scarlet fever in infancy. No other illnesses. Menstruation began at 19 years. She grew somewhat after puberty. Her height is now 1 m. 21 cm. Periods quite regular and in every way normal. After admission to hospital, when five months pregnant, she suffered considerably from dyspnoea. General condition: Rather obese and very muscular. The distal extremities of the fingers extend for 2 to 3 cm. below the great trochanters. The arms, in contrast to the condition in the normal individual, are a little shorter than the forearms. The wrists are somewhat enlarged, but the lower ends of the bones of the forearm are of normal size. The hands, a little enlarged, are of almost normal appearance, but in extension the appearance of a trident as described by P. Marie is shown. The arms are very muscular. There is no abnormal curvature of the upper extremity. Movements normal; pronation and supination complete. Flexion of the elbow greater than normal. Cubitus varus. Arms abducted from the trunk more than normal; this is owing to the great size of the head of the humeri and to the situation of the scapulae. But these are not displaced backwards as has been described in some cases. *Lower extremities.* The lower extremities are short; from great trochanter to sole the measurement is 53 cm. As in the upper extremity the proximal segment is shorter than the mesial (*i.e.* the thigh is shorter than the leg). The thigh measurements from great trochanters to the line of the knee joint, as indicated by marks in crayon on the skin, are: R. 24 cm., L. 25 cm. The legs, measured in a similar way from the line of the knee joint to the external malleolus, show:—R. 24·525 cm., L. 25·526 cm. The feet are of moderate size and length, sole 20 cm., dorsum 12 cm. and fairly broad, 8 cm. Knee joints large. Patellae prominent, with a depression below each. Ankle joint normal. Malleoli somewhat enlarged. Radiographs. Head of humerus enlarged, no surgical neck, but all the upper portion of the shaft is very thick. The part of it covered by the deltoid muscle is very short. The humerus is manifestly shorter than the forearm bones, the radius and ulna. Knees: the tibial surface of articulation of the knee joint is enlarged. The head of the fibula extends into the knee joint and forms part of the articular surface of this joint. The cranium is large. Its diameters are:—*A*, transverse, (1) biparietal, 16·5 cm., (2) bitemporal, 13·0 cm. *B*, antero-posterior, (1) occipito-mental, 23 cm., (2) occipito-

[1] Owing to an oversight several of the symbols denoting early death and disease have been omitted from this figure on Plate LI.

frontal 18·5 cm., (3) sub. occ.-fr. 18·0 cm. The frontal and parietal eminences are very prominent. The face is large. The nose, apart from its bridge, which is depressed, is fairly large and prominent; the nostrils are large. The cheek bones are not very prominent. Lower jaw well developed; angle of lower jaw somewhat prominent. Mouth: palate somewhat high-arched, otherwise normal; teeth more or less carious, but normal in number and situation. Ears, normal; lobules fairly large. Neck, rather short. Trunk: thorax normal but rather short. Abdomen somewhat prominent. All the normal curves of the spine very marked. A very slight scoliosis in the mid-dorsal region, convexity to the right. Length of trunk, from episternal notch to upper border of symphysis pubis 46 cm. Pelvis contracted in its antero-posterior diameter. (Measurements given.) All organs normal. Mental condition normal. III. 1, the first child of II. 11, of female sex, delivered by Caesarian section. Resembles its mother in many ways. Head large. Limbs somewhat shortened. Manifest shortening of thigh and arm segments. At birth the head showed exaggeration of the transverse diameters. Frontal and parietal eminences very marked, occipital eminence small. Fontanelles very large; the anterior is prolonged forwards between the two frontal bones; its two lateral angles are prolonged between the frontal and parietal bones of either side; its posterior angle is prolonged for about 1 cm. into the sagittal suture. There is a supplementary parietal fontanelle behind the anterior fontanelle. The posterior fontanelle is not much enlarged. The sagittal suture is not very marked. The mastoid and temporal fontanelles are not palpable. *Upper extremity.* The arm segment is obviously shorter than the forearm. *Measurements.* From acromion process to styloid process of radius: Total, 15 cm.; arm, 6 cm.; forearm, 6·5 cm.; hand, 2·5 cm. Movements normal. No curvature of bones. *Lower extremity.* Shows the same features. *Measurements.* From great trochanter to external malleolus: Total, 16 cm.; thigh, 7·5 cm.; leg, 8·5 cm. Trunk: moderately developed. It measures 18 cm. from episternal notch to the upper border of symphysis pubis. It is larger than that of an infant at term of 3250 grammes' weight. Thorax very broad. Abdomen prominent, enlarged. The spine shows no peculiarities. Face: the nose is large; its bridge is depressed; the nostrils are large. Malar bones prominent. All organs normal. Child well developed. There is no note of any other child of II. 11, nor of descendants, if any, of any of the other children of I. 2, nor of her parents, brethren or collaterals. (Bibl. No. 474, p. 270 and Bibl. No. 494, p. 109.)

Fig. 615. *Launois and Apert's Case.* This case was first described by Launois and Apert in 1905, further additions were made to the pedigree by Apert in 1909. I. 1 and I. 2, were tall and well proportioned, I. 1, died of asthma. There is no note of their parents, brethren or collaterals. They had eight children of whom four, II. 6—9, died young, three, II. 3—5, were healthy and well made. II. 1, aged 34, height 138 cm., had a large round head, maximum circumference 59 cm. The nose was depressed at the bridge with upturned end. Nares large, teeth carious but of normal form and situation. The trunk very muscular showed nothing peculiar in its conformation. The genital organs were well formed. All the limbs were short especially the upper segment (rhizomelic micromelia), and their shortness contrasted with the normal dimensions of the trunk. The man was a typical achondroplasic individual but his hands were not trident-shaped nor were the fingers of equal length. In the upper limb the length from the acromion to the epicondyle was 22 cm., from the epicondyle to the styloid apophysis of the radius 21 cm. and from this point to the tip of the middle finger 15 cm. The lower limbs were also very short, length from the antero-superior iliac spine to the middle of the patella 36 cm. and from the middle of the patella to the ground 35 cm. He was deeply pigmented in the situations where this is usual but large leucotic spots ('vitiligo') showed in the middle of the most pigmented parts, and small brown spots were scattered over the trunk and thighs. There was a supplementary nipple on the left side. The right eye showed traumatic cataract present since infancy. The left eye was normal. There was no sign of hereditary or acquired syphilis. II. 2, his wife, was well formed but showed tuberculous signs which had necessitated amputation of the right leg. They had five children, III. 1—5. III. 1, aged 5 in 1909, was achondroplasic and like her father had large head, bulging forehead and very short limbs. At the age of 26 months in 1905 her height was 70 cm., and maximum circumference of head 52 cm. III. 2, born three weeks before term was small and weak but normally formed; he died a few days after birth of umbilical haemorrhage. III. 3, also normal, died of gastric enteritis aged 4 months. III. 4, aged 14 months, was brought to Apert shortly after birth and appeared normal. But as she grew older, micromelia became pronounced especially in the upper segment of the arms. Her height at 14 months was 65·5 cm., length from the acromion to the epicondyle 8·5 cm., from the epicondyle to the tip of the middle finger 15·5 cm. A table is given contrasting the measurements of this child and of her elder sister when aged 16 months. [The measurements of the elder girl in the 1905 paper are given for age of 26 months, so possibly 16 is a misprint.] This table shows that micromelia existed in the younger but in a less degree. Apert says III. 4 is a case of "achondroplasie attenuée." III. 5, aged 2 months, is merely stated to have been a case of typical achondroplasia. (Bibl. No. 493, p. 606 and Bibl. No. 610, p. 35.)

Fig. 616. *Comby's Case.* III. 2, father, aged 39, healthy, height 1·66 m. III. 3, mother, aged 36, medium height and also healthy. Maternal grandfather, II. 3, very small, squat (trapu) and with very short hands and feet. There is no note of other relatives. Child, IV. 1, a boy aged 5½, born at term, difficult confinement, breast-fed for 12 months, walked at 18 months. Head very large, 53 cm. in

circumference, face large, countenance intelligent. Fontanelle closed. Weight 15,300 grammes. Height 85 cm. Upper arm and forearm 11 cm. Hands large with divergent (écartés) fingers. Length from radio-carpal joint to extremity of medius 9 cm. Total length of lower limb from iliac crest to malleolus 32 cm. Leg longer than thigh 15 and 14 cm. Bust long and seemed normal 35 cm. Considerable lumbar curvature with lateral depression of ribs. Abdomen large (57 cm. in circumference at umbilicus). Muscles of body well developed. Genital organs normal. Had adenoids. "We are therefore in the presence of a micromelic dwarf, not at all rickety, having a large head, a normal bust, lively intelligence and flourishing nutrition." (See Bibl. Nos. 408, p. 955 and 409, p. 551.)

Fig. 617. *Lauro's Case.* In 1882—3, II. 1, Anna Piacenza a washerwoman came to be confined to the Obstetrical Clinic in Naples. She was not very intelligent, said she had had an illness when a few months old, but could give no particulars of it, she called it typhus. She had been confined to bed for about three years. She lived in a damp, badly aired house, worked several hours a day and was badly fed. *Measurements.* Total height 121 cm., trunk including head 70 cm., humeri[1] 18 cm., radii 17 cm., hands 14 cm., femora 23 cm., tibiae 20 cm., feet 20 cm., spinal column 61 cm., bi-acromial diameter 31 cm., sterno-dorsal diameter 146 mm. She had a very large head and measurements of it are given. She was operated on, symphysiotomy, hysterostomatomy and forceps. A girl, III. 1, was born apparently normal weighed 2400 grammes. It had a large swelling on the right occipito-parietal region. After 24 hours it was sent to the "brefotrofio[2]," where unfortunately it was lost sight of. In 1886—7, II. 1, came for another confinement. The foetus, III. 2, which was of male sex was dead. Superficially it showed no abnormality but when the soft parts were taken from the skeleton, it was evident that it was a true case of intrauterine rachitis. There is no note of any relatives of II. 1. (Bibl. No. 235, p. 386.)

Fig. 618. *Lannois' Case.* I. 4, paternal grandfather of the achondroplasic individuals, is aged 76 years and is still living; a tall man of normal proportions. I. 3, the paternal grandmother, aged 74 years, is still living. She is a very big woman, normally proportioned. I. 2, the maternal grandmother died aged 80 years. I. 1, the maternal grandfather, died aged 63 years. There was nothing peculiar about the size or proportions of these individuals. II. 4, father of the dwarfs, aged 55 years, and II. 3, their mother, aged 54 years, are normal. All their uncles, aunts and cousins are either tall or of medium height. III. 1, aged 31 years is normal; III. 2, aged 30 years is very tall ("un colosse," 192 cm.); III. 3, aged 26 years, is achondroplasic; III. 4, aged 25 years, is achondroplasic; III. 5, aged 14 years, is very big for his age; III. 6, aged 12 years, is big for her age; III. 7, normal, died of convulsions in her third year. III. 3, Thérèse Faug, aged 26 years, height 99 cm., exactly resembles her brother, III. 4, in all respects, except for difference of sex. ("Elle est absolument calquée sur le modèle de son frere.") III. 4, Paul Vincent Faug, aged 25 years, height 111 cm. comes from a village near Bordeaux. Profession, singer at café concerts. Born after a normal labour. Precocious in speech, otherwise normal in infancy, as it was supposed. But at about 5 or 6 years of age it was observed that he did not seem to be developing like the ordinary children but rather like his dwarf sister. His intelligence is good. He is further, a wit. The face is quite symmetrical, the mouth and teeth normally formed. The frontal and parietal eminences are markedly prominent on both sides. The trunk is normal. In the upper extremities the muscles are of normal form but all the bones are shortened; there is no curvature of these bones. The hands are large and show the characteristic trident form (la déformation caractéristique en trident). All the long bones of the hands are shortened, especially the phalanges of the fingers. The skin is thickened, wrinkled and cracked and the subcutaneous tissues are thickened as in myxoedema. "The hands would have made excellent battledores." The lower extremities showed the same features as the upper. In general appearance he is somewhat old for his years. The face assumes a jeering expression when he smiles. Hair of the scalp normal. A small growth of hair about the face. Pubic hair abundant. Genital organs normal. Voice normal. Thyroid gland easily felt. All organs normal. Intelligent. (Bibl. No. 414, p. 893.)

Fig. 619. *Houston Porter's Case.* I. 1, is described by his eldest son and two grandsons as having exactly resembled II. 1. I. 2, was of ordinary size. II. 1, aged 80 years, had worked as a bargeman on the River Thames until 70 years of age. Gait "rolling" or "waddling." Micromelia of rhizomelic type. Curvature of all the long bones of the extremities; epiphyses thickened and nodular. Feet short, broad and square. Hands "en trident." Palms of hands reach to great trochanters. (Thus the arms were longer than is usual in achondroplasia, but shorter than normal.) Pelvis small. Sternum curved, gladiolus convex forwards. The typical features of the achondroplasic skull were present but were not very marked. Bosses prominent. Upper part of face large, lower part small. Face thus roughly triangular with vertex of triangle placed below, as is usual in achondroplasia, but bridge of nose not depressed or broadened and tip not upturned. Very little lordosis. Intelligence normal. He had one brother, II. 3, just like himself, but no sisters. II. 2, wife of II. 1, was of ordinary stature. III. 1 and 2, sons of II. 1 and 2, both over 50 years of age, showed the same features as II. 1. The father and these two brothers say that their brother, III. 3, who was drowned, was just like themselves. There were no sisters in this generation. The three living members of this family were all about the same height, 4′ 4″. Thus in three generations

[1] Original gives these measurements as 18, 17, 14 etc. *mm.*, which must be a misprint.
[2] "Ospizio de allevare bambini" = orphanage.

there were six achondroplasic individuals, all males. There were no daughters in generation II. or III. "The nature of the condition seems certain, though it is not quite typical, in the following points:—the shape of the bridge of the nose, the length of the arms, the absence of lordosis, and the limbs are not so massive as usual." (Bibl. No. 532, p. 12.)

Fig. 620. *Boeckh's Case.* The account of this family was given to Boeckh by V. 6, whom he calls K. She stated that her great-great-grandfather, I. 1, was a dwarf, that her great-grandparents and grandparents, II. 1 and II. 2, III. 1 and III. 2, were normal. Of the five children of III. 1 and III. 2, one, IV. 5, was a dwarf, he was rather stout with disproportionately large head and much curved short limbs. He died aged 47 of "Herzschlag." From his marriage with a normal sized woman, IV. 6, eleven children were born, four died soon after birth (in his pedigree Boeckh only puts † to three), five grew to normal size whilst two, V. 6 and V. 4, remained very small in consequence of rachitis. Boeckh says he saw V. 4, that she had a typical rickety skeleton but the curvatures were less than in the case of V. 6. Her genitals were normally developed. Her husband, V. 5, was "ein echter Zwerg von proportionirten Körperbau, sein Membrum war relativ gross, die Testikel lagen in den Leistencanalen. Seine Frau behauptete dass in der einjährigen Ehe eine vollständige immissio penis nie vorgekommen sei." Another normal sister, V. 2, was married to a big man, she had four children, VI. 1—4, of whom one a 10 year old girl, VI. 4, whom Boeckh saw was extremely small and suffered from typical rachitis V. 6, according to her relatives, was of normal size at first and showed no abnormality. She grew up in a Pomeranian village. As her parents travelled about to earn their bread by exhibition, she and her brothers and sisters were left with their grandparents and according to V. 6, were badly fed. V. 6, began to walk first in her third year, from which time a gradual increasing curvature of the extremities with retardation of growth developed. The curvature increased continuously whilst the general growth was noticeably retarded. At school she made rapid progress. She menstruated at 19. She had lordosis of the upper lumbar and lower dorsal spine with compensating kyphosis of the upper dorsal spine. The muscles of the extremities were remarkably soft. The hands and feet were short and broad. The epiphyses were enlarged. Her measurements were:—total height 97 cm. Length of arm from the acromion to the tip of the middle finger 36 cm.; from the acromion to the olecranon 18 cm.; from the olecranon to the styloid process of the radius 12 cm. Length of hand from the styloid process of the radius to the tip of the middle finger 12 cm. Total length from the iliac crest to the ground 43 cm.; from the antero-superior spine to the lower edge of the patella, 17·5 cm.; from the lower edge of the patella to the "Hautfurche am Sprunggelenke" 15 cm. Length of the foot from the heel to the tip of the second toe 11 cm. She became pregnant by a normal young man, and a miscarriage was brought on. The child, VI. 5, was a female of normal structure and build who weighed 345 grammes and was 27 cm. in length. Boeckh gives the pedigree as reproduced. (Bibl. No. 280, p. 347.)

Fig. 621. *Baldwin's Case.* II. 1, Mrs J. F. W., aged 24 years. Height 3′ 11½″. Weight 7 stone 2 pounds. She is described by Baldwin as "a typically rachitic dwarf." She is, however, undoubtedly achondroplasic and as such has been regarded by Porak, Cestan and others. Her photograph is shown by Baldwin. It shows a woman with a large, square head, prominent frontal and parietal bosses and nose with depressed bridge. The lower part of the face is small in comparison with the upper and the face is thus roughly triangular in shape with the vertex downwards. The limbs are markedly shortened in proportion to the length of the trunk and size of the head. The tips of the fingers in extension do not extend quite as far as the great trochanters. The mid-point between the vertex and the soles of the feet would obviously fall above the umbilicus. This shortening affects the proximal segments (thigh and arm) more than the mesial segments (leg and forearm). The limbs are all very massive. They all show curvatures and these, at least where they affect the lower limbs, involve the sites of junction of the epiphyses with the shafts of the long bones rather than the shafts themselves (as would be the case in rickets). The hands and feet are short, broad and thick, but no more than this can be seen from the photograph. The shape of the fingers is not to be observed. All the normal skin folds are exaggerated. There is a dark line, as of the scar of an old cicatrix, in the middle line of the abdomen, extending from just above the symphysis pubis to above the mid-point between this and the umbilicus. This might well be the scar of an operation of abdominal section, such as Caesarian section. There can be no doubt that the case is one of achondroplasia, as shown by:—micromelia (or shortening of all limbs) of rhizomelic type, associated with a trunk of medium size and a head and face of the characters above described, with curvature of limbs of the peculiar kind described, exaggeration of all the normal skin folds, massiveness of limbs and peculiarly short thick hands and feet. These features are much more than enough to show that the case is one of achondroplasia and not one of rickets. III. 1, a female child of II. 1, delivered by Porro-Caesarian section, it was "of full size but with the peculiar deformity of the mother." It lived nine months and died of Angina Ludovici. There is no note of any other children. There is no note of II. 2, nor of the parents of II. 1, nor of her brethren or their descendants, if any. (Bibl. No. 254, p. 138.)

Fig. 622. *Decroly's Case.* Ch. B. aged 28 years, III. 4, is a shoemaker, but he is unable to support his family and receives private charity. His father, II. 2, was small like himself, he says not more than 1 m. 0·5 cm. A photograph showing this individual at the age of 30 years standing near a table is to

hand and his height did not exceed that of his son in the same position. He had four sisters and two brothers, II. 1, 4, to 8; he was the second of the family and married at 32 years. He died at 41 years of age, of pleurisy. The mother, II. 3, is of normal height and still lives; she is 66 years of age. III. 4, had a brother and two sisters; the brother, III. 1, is of normal height; he is crippled by an accident (fracture of the leg). One of his sisters, the elder, was small like himself, but she died at the age of 7½ years, of bronchitis. The other is of normal height and shows no anomaly. He is the youngest of the family; his mother was 38 years of age at his birth. At the birth of III. 4, the large size of his head was remarked. He has had two accidents, one to the head from a fall upon the forehead and the other to the right knee, which was dislocated and is functionally imperfect as a result. In consequence of this infirmity he uses a crutch and his capacity as craftsman is thus reduced. Went to school at the age of 9 years and remained for 2 years; did fairly well. Apprenticed to a shoemaker at the age of 11 years but on account of his feeble physique was unable to do sufficient work to satisfy his employers. Height 110·5 cms., weight 36 kgs. Maximum circumference of head 58 cms. Antero-posterior diameter 196 mm. Transverse diameter maximum 159 mm.; length of R. arm (acromion to epicondyle) 150 mm.; radius 140 mm.; hand (from distal skinfold of wrist to extremity of middle finger) 125 mm.; R. thigh 255 mm.; R. leg (from internal condyle to extremity of malleolus) 175 mm.; R. foot (from heel to extremity of great toe) 170 mm.; trunk (from episternal notch to pubis) 485 mm. The hands are squat, the fingers of nearly equal length; the hands are "in form of a trident." The thighs are markedly abducted, the pelvis tilted forwards. The arms are abducted and the forearms are held in a position of partial flexion. The epiphyses are much enlarged, particularly those of the wrists and digits; there is, further, marked laxity of ligaments. He can easily place the whole hand on the flexion surface of the forearm. The musculature is greatly developed, the muscles are prominent and short. The face is somewhat bony but shows nothing peculiar, neither relative smallness or exaggeration of the volume of the head nor depression of the bridge of the nose. Sexual development normal. Intelligence seems to be well up to the average. As regards his social capacity this is diminished by the above physical infirmities. He married, at the age of 20 years, a normal person of his own age, and has had three children by that marriage, IV. 1, 2 and 3. The eldest, IV. 1, a boy aged 6 years and 4 months, shows the same anomalies as his father; there are two little girls, aged 4 years and 18 months, normally formed. The boy attends the village school and is regarded by his master as of average intelligence for his age; but he has to be placed in a special class on account of his physical condition. The labour was a very difficult one and delivery was effected by forceps. His peculiarity was noted by his father at birth, *i.e.* the head was relatively large and the limbs short. He now has the appearance of a dwarf with the physiognomy and body of an infant of his age. Height 815 mm.; weight 13 kgs. (Photograph shown in memoir.) It is that of an achondroplasic child typical in all respects except that he has marked genu valgum ("knock knee"). Tables of measurements follow but it is unnecessary to give them all. As in the father he can place the whole hand on the front of the forearm, thus showing marked laxity of ligaments. Both these cases are undoubtedly achondroplasic. (Bibl. No. 515, p. 19.)

Fig. 623. *Peloquin's Case.* IV. 1, Pierre P., aged 30 years. Tailor. Height 121 cm. The eldest of three children; breast-fed; began to walk at 18 months of age. His small height was remarked in his early years but he was well formed. His childhood passed without incident. His intelligence developed like that of other children. He learned to read and write at 6 years of age. In spite of his small height his head and trunk are those of an ordinary man. The face, with an abundant growth of hair, is intelligent and without stigmata of any kind. He answers questions well and gives the impression of a man of much intelligence for his social station. Head: Round and globular, of brachycephalic type; without exaggeration of the frontal and parietal eminences. The nose is somewhat depressed and broadened at its bridge. Upper extremities very short; the distal extremities of the fingers only extend a few centimetres beyond the great trochanters. On palpation the bones are very thick but not deformed. The hands are small, the digits of different lengths; there is no deformity "en trident." Musculature powerful he can easily carry a weight of 15 kgs. with one arm. *Measurements*:—Upper extremity: Total, 50 cm.; arm 17 cm.; forearm 19 cm.; hand 14 cm. Lower extremity, very short, very muscular; a curvature of the tibiae with very pronounced internal concavity. Lower extremity: Total length, 54 cm.; thigh 28 cm.; leg 27 cm.; foot 21 cm. The trunk is that of a man of medium size; the distance between the episternal notch and the upper border of the symphysis pubis is 54 cm. All the organs are normal. He is a healthy man, quick in his movements, a good workman and a good walker. IV. 2, sister of this man, aged 27 years. Her height is 118 cm. Breast-fed until age of 12 months. No peculiarity was noted in her early infancy. She began to walk at the age of 13 months. Menstruation began in her 15th year. Chlorosis 17th to 20th years. Very intelligent. Has a very fine memory; "is of a gay and happy temperament well contented with her lot." On first observation one is struck by the disproportion shown between the size of her head and the shortness of her limbs. Her head is brachycephalic, like that of her brother. It is the head of an ordinary medium sized woman of her age. The limbs are very short, especially the lower extremities. *Measurements*:—Upper extremity: Total, 44 cm.; arm 15 cm.; forearm 16 cm.; hand 13 cm. Lower extremity: Total (from great trochanter to sole), 47 cm.; thigh 23 cm.; leg 24 cm.; foot 18 cm. Trunk, from episternal notch to upper border of symphysis pubis, 52 cm. The hand "en trident" can hardly be made out. She

is strong, muscular and in excellent health. Weight 43 kgs. Organs normal. She is unmarried "but it must be said with insistence that this is not from lack of opportunity, but solely from motives of convenience." IV. 3, sister of these, aged 25 years. Normal. Height 1·65 m. Health perfect. Breast-fed. III. 2, aged 48 years, the mother of these children is normally proportioned. Her height is 140 cm. She says that her husband, III. 1, who died aged 52 years "of some gastric trouble," and his father, II. 1, were both very short and of about the same height, 135 cm. approximately. In both the body was long, the lumbar curve marked. The head was of ordinary size. They had short arms and legs. IV. 1, corroborates these statements. There is no note of II. 2, or any other relatives, antecedent or collateral. This case was described by Peloquin and also by Poncet and Leriche. Peloquin did not give the measurements of IV. 2, who, he says, refused to be examined. (Bibl. No. 417, p. 28, Bibl. No. 442, p. 174, and Bibl. No. 443, p. 202.)

Fig. 624. *Litchfield's Case.* There is a general description of the chief characteristics of achondroplasia given but no particular description of any individual in the pedigree except III. 1. Litchfield did not see the mother, II. 3, who had been delivered by Caesarian section and had died shortly afterwards. He only gives a photograph of her and her husband and states that the photograph shows she was a typical achondroplasic dwarf. A photograph of III. 1, is given and it is stated he was achondroplasic, that his ribs were not beaded but that distinct Harrison's sulcus and a slight kyphotic curve of the dorso-lumbar spine pointed to superimposed rickets. At the age of 1 year 11 months he showed the following features. Short bowed legs, very thick and pudding like, epiphyses enlarged, skin folds exaggerated, subcutaneous tissues increased. Arms shortened; tips of fingers do not extend beyond great trochanters. Hands short and broad. Fingers stumpy, "main en trident." Feet short and broad. Head large, bridge of nose broad and depressed. Intelligence normal. "He also showed signs of rickets." Photographs of these three individuals are shown in the above paper. (Bibl. No. 533, p. 624.)

Fig. 625. *Herrgott's Case.* II. 6, Ernest K——, aged 53 years, of height 160 cm. Head large, as big as that of a normal adult of his age. The line of the nose continues directly, in profile, that of the forehead; it is slightly depressed. Teeth regular and normal; palatine vault shows no peculiarities. Neck rather short; thyroid gland clearly palpable. Clavicles normal. Thorax measures 20 cm. from episternal notch to xiphoid cartilage. Vertebral column straight. Hands short, broad and thick and roughly square; they are hairy. The fingers, especially those of the right hand, look as though they had been equalised by a blow of a hatchet; they are as thick as those of an ordinary adult of the same age, but are extremely short; the middle and ring fingers of the right hand are of equal length and measure 4 cm. from the margin of the interdigital fold; on the left the middle is clearly shorter than the ring finger. The little finger and the index are of equal length; they measure a little more than 3 cm. The "main en trident" is well shown. Micromelia very marked, upper and lower limbs being very short. Forearm from line of extension of wrist to olecranon measures no more than 14 cm. and the arm, from acromion to olecranon, measures 17·5 cm. The upper extremities are very muscular and show little limitation of movement. He states that he is one of a numerous family in which there are several dwarfs. His *father*, I. 1, was a dwarf who died in old age having had, by several females of normal height, numerous children. I. 3, a *maternal* aunt of II. 6, was also a dwarf like himself; she had become pregnant twice, II. 8 and 9; in the first confinement embryotomy of the infant was performed and the mother was saved; incontinence of urine followed this confinement; in the second confinement she died. II. 2, one of the brothers of II. 6, much older than himself was a dwarf considerably smaller than himself. This brother married and had numerous children, mostly dwarfs, both boys and girls: the latter all died in childbed. II. 4, one of the sisters of II. 6, was also a dwarf. She married a man of normal height and became pregnant but died during confinement at the age of 22 years. She had been operated upon, he states, by "the doctor of the Empress" and her skeleton is conserved in the museum of one of the hospitals of Paris, probably the Tarnier Clinique. Ernest K——, II. 6, is intelligent; he can read and write, he follows the profession of pedlar, leads a wandering life in a caravan, playing comedy and music; he is the originator and director of a troupe formed, in part, by members of his own family. He married, at the age of 19 years, a big and strong woman of 21 years, who died at the age of 42 years, having presented her husband with twelve children and three abortions. Of the twelve children four are dead: twins, III. 5—6, the one aged five days the other aged five months; a daughter, III. 7, of three years and another, III. 8, of five years both of whom seem to have been normal. Eight children are alive, of whom five are of normal development and three (daughters) are achondroplasic like their father. Their ages are 23, 21 and 19 years respectively; they follow three children of normal height, aged 27, 26 and 25 years respectively, and are followed by two others, also well formed, who are in their 17th and 15th years. III. 12, Marie Louise, aged 23 years, had been breast-fed for two years. Menstruation began at the age of 17 years and had been quite regular. Age of commencing to walk uncertain, but it seems to have been between $3\frac{1}{2}$ years and 4 years. At 15 years of age her height was no more than 50 cm. Since the age of 19 years her growth seems to have completely ceased. Her height is now 95 cm. There is a slight dorsal kyphosis which adds to the oddness of her appearance. The head, relatively to her age and not to her height, is normal. The vault of the palate is regular; the teeth are regular, the thyroid gland is very

slightly developed, the clavicles are well formed. The hands are short, the fingers short and broad; in the left hand the medius is the longest but in the right the ring finger is longer than the middle. The "main en trident" is typically present in the left hand but not in the right. The forearm measures 11 cm. from the extremity of the olecranon to that of the styloid process of the ulna and the arm 18 cm. from olecranon to acromion. The epiphyses are large and the diaphyses are very slightly incurved. This woman, as well as her father and two achondroplasic sisters, has a peculiar gait. As she walks she holds the arm slightly abducted from the trunk and, in walking, carries to the front alternately all the side of the body corresponding to the leg advanced to the front. Marie Louise is very intelligent, reads, writes and calculates well. In the troupe which her father directs she is an actress, singer and musician (violinist). III. 13, Lucie K——, aged 21 years, height 110 cm. Head of normal size, in disproportion to her small height. Upper limbs very short, wrists and elbows large. Humerus seems slightly incurved. Arm (from acromion process to extremity of olecranon) measures 13 cm.; forearm is about the same length, the distance from the extremity of olecranon to styloid process of ulna is 13·5 cm. Hands short and show the disposition "en trident" very markedly. The ring finger and not the medius is the longest. Movements of extension as well as of supination are limited. Complete adduction of the arm to the thorax is impossible and the obstacle seems to be the great size of the humeral head relatively to the articular cavity. The lower extremities, as well as the upper, are of remarkable shortness. (A complete description of this individual, with three photographs, is given; she is a typical achondroplasic female with a scar of what was probably an operation for Caesarian section in the supra-pubic region. It does not seem necessary to give this description here in full.) There was no history of syphilis or other infection. She began to menstruate at the age of 15 years. She had had a miscarriage in the second month, 11 months before attending hospital, when again pregnant in the eighth month. Caesarian section performed and a male child, IV. 2, delivered. Normal like its father, a member of the troupe. Aborted once more, and then again underwent Caesarian section, a female child, of normal proportions, IV. 4, being born. III. 15, Leontine K——, aged 19 years. Breast-fed like her sisters; like them ignorant of the age at which she began to walk. Has menstruated regularly since the age of 15 years. Height 106 cm. The head is of the size of that of a normal person of her age. Thyroid gland easily felt. Hands short and the fingers, like those of her father, of nearly equal length. The wrists show marked enlargement. Complete extension of the elbow is impossible. The measurement from the styloid process of ulna to extremity of olecranon is 13 cm.; that between olecranon and acromion is 18 cm. Her intelligence is normal and like her two sisters she is a member of the troupe of which her father is the director. (See Bibl. No. 516, p. 8.)

PLATE LII. Fig. 626. *Balme and Reid's Case.* I. 1, and I. 2, were healthy. Of their children, II. 1, died aged 6 of convulsions. II. 2, died aged 2 of convulsions. II. 3, was alive and healthy. II. 4, died aged 4 of diphtheria. II. 5, and II. 6, were both alive and healthy. II. 7, aged 20 was in good health. II. 8, died aged 4 of measles and bronchitis. II. 9, was a miscarriage in the third month (three years after the eighth child). II. 10, aged 12, was born when his mother was 44; three years after the miscarriage, II. 9. The mother stated that throughout the whole length of time while she was pregnant with the patient, II. 10, she "menstruated" regularly every month; also that during the period she was subject to several "shocks." The confinement was a difficult one, owing to the large size of the child's head and the fact that it was a breech presentation. No instruments were employed but artificial respiration had to be resorted to, as the child was in a state of partial asphyxia. As soon as the child was born he was noticed to have a large head and very tiny arms and legs. Up to the age of 15 months the child was solely breast-fed and appears to have been quite healthy, though he is said to have had a fit when 9 months old. Dentition occurred at the ordinary time, but the mother did not allow the child to attempt to walk, until he was $1\frac{3}{4}$ years old as "he was so fat." He then made rapid progress and had always been particularly strong, never suffering from any of the ordinary children's ailments with the exception of measles. He had always been very bright mentally. At age of 12, his height was only $38\frac{3}{4}''$; his head was abnormally large and square measuring $23\frac{3}{4}''$ in circumference but was free from bosses or other signs of disturbed growth. The bridge of the nose was very depressed and the mouth was usually kept open. (The child had adenoids in abundance.) The arch of the palate was distinctly high, the thorax was well developed and measured 24" after deep expiration. The clavicles were normal in length though rather more curved than usual, the scapulae and sternum also appeared normal, and there was marked beading of the ribs. The vertebral column measured $20\frac{3}{4}''$ from the occipital protuberance to the top of the coccyx and there was well marked lordosis. The thyroid body was palpable and there were no supra clavicular pads. The arms were very short, so that the fingers only just reached the bottom of the great trochanter when the boy was standing upright. The humeri were short, thick and slightly curved and the elbow joints could not be extended beyond an angle of about 160°. The hands were small and fat and the fingers all of the same length, they showed very clearly the characteristic separation between the middle and ring fingers. The femora were short and thick, the lower epiphyses appearing relatively broad and massive. There was marked lateral curvature of the left tibia and fibula, but the corresponding bones on the right side were quite straight. The feet were somewhat deformed, the middle toe of the left foot being curiously deflected laterally so as to lie over the second toe:—*Measurements.* (Those in brackets

are measurements of normal boy of same age.) Stature $38\frac{3}{4}''$ ($55\frac{1}{4}''$). Height when sitting 26″ ($27\frac{1}{2}''$). Crown of head to umbilicus 20″ ($27\frac{1}{2}''$). Umbilicus to sole of foot $18\frac{3}{4}''$ ($32\frac{3}{4}''$). Suprasternal notch to symphysis pubis $16\frac{1}{2}''$ ($15\frac{3}{4}''$). Occip. protuberance to tip of coccyx $20\frac{3}{4}''$ (24″). Circumference of head $23\frac{3}{4}''$ ($20\frac{3}{4}''$). Tip of mastoid to tip of mastoid (across vertex) $16\frac{3}{4}''$ ($15\frac{1}{4}''$). Clavicle $5\frac{1}{8}''$ ($5\frac{1}{8}''$). Humerus (acromion to external condyle) $5\frac{1}{2}''$ ($10\frac{1}{4}''$). Radius 5″ ($7\frac{1}{4}''$). Ulna $5\frac{7}{8}''$ (8″). Wrist joint to tip of middle finger $5\frac{1}{4}''$ ($5\frac{3}{4}''$). Circumference of hand $5\frac{3}{4}''$ (7″). Thumb $1\frac{5}{8}''$ (2″). First finger $1\frac{5}{8}''$ ($2\frac{5}{8}''$). Second finger $1\frac{5}{8}''$ ($2\frac{7}{8}''$). Third finger $1\frac{5}{8}''$ ($2\frac{3}{4}''$). Fourth finger $1\frac{3}{8}''$ ($1\frac{3}{4}''$). Circumference of upper end of humerus $8\frac{1}{2}''$ (10″); around crest of pelvis 21″ ($22\frac{1}{4}''$). Ant. sup. spine to knee joint $7\frac{5}{8}''$ (16″); ant. sup. spine to int. malleolus $15\frac{1}{2}''$ ($28\frac{1}{4}''$). Great trochanter to sole $18\frac{1}{2}''$ ($29\frac{3}{4}''$). Femur (great trochanter to ext. condyle) $8\frac{1}{4}''$ ($13\frac{1}{2}''$). Tibia $6\frac{3}{4}''$ ($11\frac{3}{4}''$). Fibula $7\frac{1}{4}''$ ($12\frac{1}{2}''$). Length of foot $6\frac{1}{2}''$ ($8\frac{1}{4}''$). Circumference of lower end of femur 10″ ($11\frac{3}{4}''$). (Bibl. No. 465, p. 780.)

Fig. 627. *Osiander's Case.* I. 2, was an unmarried dwarf, aged about 27 and scarcely 49 "pollices Parisienses" in height. She was healthy, fleshy and obese and except that the femora were curved and the pelvis too much inclined, she was well formed. She menstruated at 18, and menstruation continued (emansit) after conception. There is a long description of the confinement. The child, a girl, was born almost four weeks too soon, her weight was $5\frac{1}{2}$ "librae civiles" and length 17 "pollices." The smaller diameter of the head was 3″ 1‴, the larger diameter of the head was 4″ 2‴, the breadth of the shoulders (latitudo humerorum) 4″. The measurements are presumably in French inches and lines. A picture of the dwarf is given, the arms look a little short, the fingers just reaching a little below the top of the thigh and the legs look rather short. (Bibl. No. 51, p. 1.)

Fig. 628. *Scharlau's Case II.* Of I. 1, no statement is made. I. 2, was a strong woman of 36 when II. 6 was born and she had already borne five healthy children, II. 1—5. She had cholera during pregnancy. II. 6 was born a few weeks too soon, it was nearly dead and died. In external appearance it was almost exactly like *Case I*, Fig. 646. When dissected it was found the sutures were patent and the fontanelles large. The spine was of normal length, the clavicles well developed, shoulder blades small and soft. The pelvis was very small. All four extremities were remarkably short and much curved but not fractured. The length of the upper arm was 1″ 6‴, of forearm 1″ 7‴, of thigh 1″ 9‴ and of leg 1″ 5‴. The epiphyses were much enlarged[1]. (Bibl. No. 135, p. 412.)

Fig. 629. *Kirchberg's and Marchand's Case.* I. 1, and I. 2, were healthy, no trace of struma, tuberculosis, syphilis or alcoholism in them. II. 1, was the first child. Born dead or died soon after birth. The length of the trunk was 31·5 cm. The head was very large, its circumference being 37 cm. The face was remarkably flat, the nose perfectly flat, the nostrils extremely small and eyes rather protuberant. The hard palate was cleft from the middle posteriorly, the soft palate and uvula were also cleft. The extremities were very short and thick. The length of the upper extremities from the acromion process of the scapula to the tip of the middle finger was 12 cm., that of the lower extremities measured $7\frac{1}{2}$ cm. (internally). The femora and tibiae were curved. The feet very short and the skin of the limbs was in thick folds. The thorax was very short, fairly broad and sunken in the middle line, the sternum extremely flexible and moveable. The abdomen was much distended. Measurement from vertex to umbilicus was 22 cm., from umbilicus to heels $9\frac{1}{2}$ cm. The pelvis was small and narrow. Maximum length of left femur 4·8 cm., of right 4·7 cm. The bones of the legs were very thick. The tibia was 4 cm. in length. The scapula was extremely thick and short, its greatest height being 3 cm. The clavicle was long and narrow $4\frac{1}{2}$ cm. in length. The bones of the upper extremities were short and thick; the radius and ulna being somewhat curved anteriorly. Length of ulna 4·1 cm., of radius 3·3 cm., of humerus 4·4 cm. (Bibl. No. 243, p. 183.)

Fig. 630. *Von Franqué's Case.* No statement is made with regard to I. 1. I. 2, had only learned to walk at 2 years of age, and had been bandy-legged as a child, but she had been always healthy; showed no noticeable rickety symptoms. She had five children, II. 1—5. II. 1, was still-born and its arms and legs were not perfect. II. 2, born a year later lived for $\frac{1}{4}$ year and had club feet. II. 3, born two years after II. 2, was normal, healthy and well-grown. II. 4, born two years after II. 3, had the same curvature and shortening of the extremities as II. 1 and II. 5. II. 5, is the case described by von Franqué. It was born before full term, according to the mother's reckoning within the 30 weeks. It was 34 cm. in length and the circumference of the head was 30·5 cm. It was well nourished, but died[2] because it was a breech presentation and there were difficulty and delay in delivery of the after-coming head. The defective formation and curvature of the extremities were at once evident. The arms and legs were much shortened. Length of humerus 4 cm. instead of 6·75, and of ulna 2 cm. instead of 5·75. Length from great trochanter to the ext. malleolus 7 cm. The hands and feet were relatively well developed. The expression of the face could not be described as cretinoid, the root of the nose was not specially sunken. The convexity of the curvature was backwards in the humerus and was backwards and towards the middle in the ulna and forwards and outwards in the femur and tibia. In the middle of the latter there was an acute angular curvature, which could be felt through the soft parts. The feet were in the position of equino-varus. An examination of the radius, ulna, humerus and costa showed great hardness and lack of

[1] In Fig. 628, II. 6 and not II. 2 should be marked as dying at birth.

[2] Not indicated in Fig. 630.

the medullary cavities in the diaphyses but no special thickening of the epiphyses. There was in the ribs a conformation similar to the "rosary" in rickets. (Bibl. No. 278, pp. 80 and 88.)

Fig. 631. *Meynier's Case I.* I. 1, suffered from sciatica (ischialgia). No statement is made with regard to I. 2, except that all the ancestors and collaterals of II. 4, were of medium height. II. 2, also had sciatica. II. 3, had convulsions and a cousin of II. 4 (what degree of cousinship and on which side is not stated) had bronchial asthma. II. 4, was of middle height, he suffered from sciatica and lumbago. Syphilis was absolutely denied. He married II. 5, who was healthy, she had a brother, II. 6, who died aged 2 and who had rickets; of her parents, I. 3 and I. 4, nothing is stated. II. 4 and II. 5, had seven living children and there was a miscarriage of 30 days, III. 7, between the sixth and last child. III. 1, suffered from eczema, otherwise the first six children, III. 1—6, were healthy. They were nursed by their mother from 12 to 15 months and were all of normal stature except III. 6, aged 3, who was of rather less than normal stature. II. 8, was an eight months' child and at birth the parents noticed she had very short legs, her hair was long and very thick and her skull soft. When seen in 1902 she was aged 7 months and 19 days. One was struck by the shortness of the lower limbs, the large head, the size of the anterior fontanelle, the long hair, the skin in folds, the large and fleshy hands and feet, the exaggerated lumbar curvature, the well-formed thorax and surprising intelligence of the baby who even then could say a few words. A second examination was made in 1903 at the age of 10 months. She was then measured. She had whooping cough in May 1903 and died in June. (Symbol of early death omitted in Fig. 631.) As she died in a private house, an autopsy could not be made at once, but the skeleton was obtained five months after and measurements were again taken. Both sets of measurements are given. Only the first measurements are given below. Length from episternal notch to upper border of symphysis pubis 25 cm.; from episternal notch to distal extremity of xiphoid process 9 cm.; from distal extremity of xiphoid process to the umbilicus 8 cm.; from umbilicus to upper border of symphysis pubis 8 cm. The lumbar curvature was very pronounced, the lumbar column projecting straight from the glutei. *Upper limbs.* From the angle of acromion to the distal extremity of the middle finger on the outer side, L. 23 cm., R. 25·5 cm.; length of arm from angle of acromion to olecranon, L. 9 cm., R. 10 cm.; forearm from the olecranon to the styloid process of radius, L. 8 cm., R. 9 cm.; hand from the styloid process of the radius to the extremity of middle finger, L. 6 cm., R. 6½ cm. The fingers were very large with an evident tendency to the "main en trident." The middle and index fingers were of the same length in the right hand, in the left hand the ring and middle fingers were of the same length and the index and little finger of the same length. It was almost a square hand. The greatest circumference was 12 cm. *Lower limbs.* Length from antero-superior iliac spine to external malleolus of ankle joint, L. 23 cm., R. 22 cm. Length of thigh from antero-superior iliac spine to head of fibula, L. 14 cm., R. 13 cm.; leg from head of the fibula to the external malleolus, L. 9 cm., R. 9 cm. Distance from the external malleolus to the sole 3·5 cm. The feet were large and fleshy, with large toes. She was radiographed, and it was seen that the diaphyses in the lower limbs were normally ossified and were especially thick at their extremities; they showed no nodosities or signs of fracture. The curvature in the bones of the legs was more marked than in the femora, but was regular, uniform and symmetrical. The distance between the diaphyses of the femora and those of the bones of the legs was very ample and allowed one to suppose that the epiphyses were much enlarged and completely cartilaginous. (Bibl. No. 466, p. 470.)

Fig. 632. *Hunter's Case.* No note is given of Gen. I. II. 3, was aged 74, alive, she suffered from bronchitis. She had two elder sisters, II. 1, who died aged 58 of Bright's Disease, and II. 2, who died aged 71, also of Bright's Disease. No note is made of any descendants of these sisters. She married II. 6, who died aged 59 of chronic paralysis. II. 6, had an elder sister aged 71 who was in good health. It states she was the second born but mentions no elder brother or sister. A younger brother, II. 7, died, aged 16, of consumption. II. 3 and II. 6, had seven children, III. 1—7. III. 1, aged 49, healthy. III. 2, aged 46, healthy. III. 4, aged 42, healthy. III. 5, died of scarlet fever, aged between 4 and 5. III. 6, died of scarlet fever, aged between 2 and 3 years. III. 7, died aged 19, of consumption. III. 3, aged 44, who was not very intelligent, being described as "rather simple," was hardworking and thoroughly respectable. She married, at the age of 22, III. 11, who was then aged 20. His mother, II. 9, aged 76, suffered from chronic bronchitis, she had an elder sister, II. 8, aged 78 and healthy, a brother, II. 10, died of a stroke aged 68, a sister, II. 11, died of dropsy, aged 35. II. 12, was dead, but there was no information as to cause of death. II. 13, died aged 21, of a "twist in the bowels." No note is made of any descendants of these brothers and sisters. II. 14, father of III. 11, died aged 72 of senile decay, his brother, II. 15, died of pneumonia, aged 66. II. 16, aged 72, II. 17, aged 69, and II. 18, aged 65, were in good health. Nothing is said with regard to their descendants. III. 11, aged 42, said he had a kind of stroke at about age of 2 years but otherwise was healthy. His sister, III. 8, aged 54, and brother, III. 9, aged 49, were in good health. III. 10, died of meningitis aged 19. III. 12, aged 39, and III. 13, aged 35 were healthy. No note is made of any descendants of Gen. III. except the family of III. 3 and III. 11. They had seven children, IV. 1—7. Of these, IV. 1, M. C., aged 21, IV. 7, aged 6½, were in good health. IV. 2, W. C., died aged $1\frac{10}{12}$ years of meningitis. IV. 3, H. C., aged 16, was achondroplasic though unless her photograph was misleading the skull had escaped involvement. She was of average

intelligence with well-developed limbs and mammae, but no pubic hair. IV. 4, A. C., aged 15 (1910), height 4′ 5″. Weight 64 lbs. He suffered from diabetes for about seven years but seemed to have outgrown it. Had not been medically attended for about eight years. He only looked half his age and his lower limbs were shortened. IV. 5, W. C., aged 13 (1910), was born after long and hard labour, had fits in infancy and a "kind of stroke" when 2 years old. He shows distinct suggestions of achondroplasia and is in an imbecile asylum. IV. 6, M. J. C., aged 9, was very delicate, before she was 5 years old she had both thighs and her collar bone broken. IV. 7, F. C., aged 6, said to be in good health. The case is an interesting one as showing a typical achondroplasia, associated with mental defect, phthisis and defective growth in the same stock. (Unpublished case from Dr D. W. Hunter: see Plate SS (99)—(101), and note resemblance of hands in IV. 3 and IV. 5.)

Fig. 633. *Crimail's Case.* I. 1, died suddenly aged 40. I. 2, was strong and well. II. 1—6, were healthy. II. 7, walked when 13 months old and her development appeared normal except that the thorax developed greatly in proportion to the extremities. The vertebral column showed no curvature or lateral deviation; the limbs were small, but not deformed, being quite straight and without curvatures or trace of rickets. *Measurements.* Total height 1·28 m. Length of arm from the angle of the acromion to the distal extremity of the middle finger 0·51 m.; from the angle of the acromion to the proximal extremity of the olecranon process 0·19 m.; from olecranon to the styloid process of the radius 17·5 cm.; from the styloid process of radius to the distal extremity of the middle finger 14·5 cm.; length from the antero-superior iliac spine to ground 15·5 cm.; from the spina iliaca to head of fibula 28 cm.; from the head of the fibula to the external malleolus 27·5 cm.; from the external malleolus to the ground 4 cm. The pelvis was uniformly narrow (*i.e.* in all dimensions) like that of a non-rickety dwarf. Caesarian section was performed. The child was dead on delivery and no details of it are given (sign for death at birth omitted in Fig. 633). The woman recovered. (Bibl. No. 244, p. 272.)

Fig. 634. *Wood and Hewlett's Case I.* Of I. 1 and I. 2, nothing is stated, they had 16 children, II. 1—16, all well grown except II. 13. II. 1, aged 80, was blinded by an accident when a boy. II. 13, was the 13th child. He was a strong active dwarf in regular employment as a farm labourer. His general health was excellent. He did not waddle as he walked. The general appearance was typical of achondroplasia. He had a large head, long body, short arms and legs and marked lordosis. The bridge of the nose was hollowed, the palate very high arched, the back of the head vertical instead of convex. The extension of the elbow joint was markedly limited by the excessive bony development at the posterior part of the lower end of the humerus. The wrists and fingers were very lax, allowing a much greater range of extension than in the normal hand. The fingers were short, the genitalia well developed. *Measurements.* Abdomen at umbilicus 31″. Length of right femur from the trochanter to external condyle 9½″; foot 8¼″; hand from lowest crease in wrist 5½″. Circumference of calf 10½″. Extension of both arms was limited to an angle of 135°. (Bibl. No. 437, p. 17.)

Fig. 635. *Hutchinson's Case.* This is an account of the skeleton of the Norwich dwarf, I. 1, aged 35, height 4′ 2″. He was executed for the murder of his child, II. 1, and the attempted murder of his wife. His legs and arms were short, the tips of the fingers only just touching the great trochanters. All the larger bones of the limbs were thick and remarkably short but not curved in any way. *Measurements.* From the crown of the head to the top of the breast bone 13″. From the top of the breast bone to the extremity of the trunk 19″. Length of thigh 8″; leg 8″; upper arm 6½″; forearm 7″; hand and longest finger 5½″. Circumference of wrist 6½″; knee 14″; ankle 8″. Breadth of shoulders 4″ (?); across hips 12″. Circumference of skull 23½″; neck 14½″; trunk measuring at lower end of breast bone 32″. (Bibl. No. 242, p. 229.)

Fig. 636. *Turner's Case.* I. 1 and I. 2, were not related, married at 23 and were both alive and healthy. No history of syphilis was obtainable and no other members of the family had been deformed. II. 1, the eldest child was born within the first year of married life, the following year stillborn twins, II. 2—3. About a year later, II. 4, now well and normally developed; then II. 5, who died aged 12 months of diphtheria, then II. 6, now aged 3½ years, and lastly, II. 7, who died aged 3 months of "consumption of the bowels." The deformities of II. 1, aged 10, were noticed at birth but became more marked as the child developed owing to the increased size of the head and body as compared with the limbs. She had always been very intelligent and was considered much sharper than her sisters. She was unable to attend to herself during the ordinary calls of nature, unable to wash thoroughly and took about three hours to dress. She could write only with great difficulty owing to the deformity of her fingers and hands. She had no serious illnesses. The isthmus of the thyroid gland could be felt but the lateral lobes could not be detected. When sitting there was usually a single kyphotic curve, but on standing there was marked lumbar lordosis. She had slight genu valgum. Even when the arms were completely extended by the sides, the fingers could scarcely reach the iliac crests and could only just touch the umbilicus. The hands were spade-like whilst the fingers tapered to their extremities so that even when outstretched the ends were not in apposition. The index and middle fingers were widely separated from the ring and little fingers. The face was small like that of a cretin with coarse features and very depressed nose.

Measurements. Height standing 37″; sitting 24″. Fronto-occipital circumference of head 21¾″. Acromion to tip of olecranon 5″. Olecranon to tip of middle finger 8⅛″; olecranon to styloid process of ulna 4½″. Circumference of wrist 5″. Length of clavicles 5″. Occipital protuberance to tip of coccyx 16″. Antero-superior iliac spine to ground when standing 15½″. Great trochanter to external malleolus 12¼″. Antero-superior iliac spine to knee joint 7″. (Bibl. No. 354, p. 263. Photographs are given.)

Fig. 637. *Variot's Case.* I. 1 and I. 2, were healthy and well made. They had eight children, II. 1—8, of whom the six eldest, II. 1—6, were normal, and II. 8, had died, whether normal or not is not stated. There had been no miscarriage and no reason to suppose syphilis. II. 7, only began to walk at 3, since then her development had been slow but she had no serious illness. Her height was 1·24 m. or 15 cm. below normal. The lower limbs were remarkably short. Length of thigh from great trochanter to the line of articulation of the knee joint 27 cm.; leg from the articular line of the knee to the external malleolus 30 cm.; height of trunk from episternal notch to symphysis pubis 46½ cm. The femora were much shortened, and the internal condyles abnormally developed. There was genu valgum on both sides. The feet were very deformed, short and squat, the plantar arch being almost absent, the metatarsals seemed shortened, the transverse arch of the foot normal. Length of upper arm from acromion to extreme epicondyle 18 cm.; forearm from tip of the olecranon to styloid process of the radius 21 cm. There was no incurving of the bones of the forearm or of the leg. The hands were similarly deformed to the feet and appeared rather square. There was little difference in the length of the fingers which hardly tapered at the extremities. The trunk seemed extremely long and well made, and there was no lumbar curvature. The head was well made and the intelligence well developed. (Bibl. No. 438, p. 268.)

Fig. 638. *Davidson's Case.* Details of this case were sent to Ballantyne by Dr Samuel Davidson. I. 1, was healthy but much addicted to use of alcohol in excess. I. 2, was healthy, she had had seven children, II. 1—7, all but II. 7 were born alive at full term and breast-fed. II. 1, died aged 13 months from "convulsions." II. 2, died aged 1 month from "bowel hives." I. 2, was aged 32, and had been married 15 years at birth of II. 7; there was no hyramnios and the infant died during delivery (not indicated on Plate). It weighed 2160 grammes and was of the female sex. The lower limbs were fixed in an unnatural position; the thighs were sharply abducted and passed outwards almost at right angles to the pelvis, the legs were partly flexed and showed a marked concavity on the inner aspect and the feet were turned sharply inwards. *Measurements of Infant.* Length with lower limbs in position described 38 cm. Distance from finger tip to finger tip with arms extended 32 cm. Occipito-frontal circumference of head 33 cm. Occipito-mental circumference of head 37 cm. Both upper and lower extremities seemed slightly shorter than normal and on both was some deepening of the natural flexures. The head was broader than usual, the nose short and somewhat flattened with a depressed bridge. The thyroid and thymus were normal. (Bibl. No. 405, p. 338.)

Fig. 639. *Osler's Case.* I. 1 and I. 2, were healthy French-Canadians. They had 14 children, II. 1—14, of whom the eldest was aged 27 and the youngest 4. Five children had died in infancy, II. 10—14. With the exception of II. 5—6, the dwarfs, the remaining children were all healthy and well grown. II. 5, aged 16, was 86·5 cm. in height. The mother did not remember anything abnormal about her as a young infant. She walked at 18 months old. Her head seemed large, and the mother said the fontanelle did not close till her sixth year. When between 3 and 4 years of age it was noticed she did not develope naturally and that her joints were very large. She was bright-looking and intelligent but somewhat full and coarse featured. The head measured 56 cm. (presumably the horizontal circumference, but the writer does not say what he is measuring!); the teeth were well formed. She talked fluently and well, had learned to read a little and was beginning to write; but was backward for a girl of her age. The most remarkable phenomenon was the condition of the joints of the long bones. The shafts were short and looked thin and the articulations very short and irregular. The shoulders were not much affected, but the elbow joints, wrist joints, knees and ankles were enormously enlarged. She was a little knock-kneed when she stood. The mobility in the joints was perfect. II. 6, aged 11½, was 87 cm. in height. His mother did not notice anything special about him, except that he was late in walking and the anterior fontanelle did not close till between the third and fourth years. He did not seem to grow much after the fourth year. He resembled his sister. His head was large and well formed, he was very intelligent looking, bright and good-tempered. The articulations were extraordinarily large and contrasted with the smallness and shortness of the shafts of the bones. He was somewhat pigeon-breasted and when he stood, knock-kneed. Osler states there is no doubt these were achondroplasic and not cretins. (Bibl. No. 331, p. 190.)

Fig. 640. *Meckel's Case.* I. 2, and I. 3, were healthy, and no such deformity had occurred in either of their families, although both had been married before. The mother, I. 3, of the children thought that the rough treatment which she had received in her third confinement with a normal child was the only cause as far as she knew why the first three children, II. 2—4, were normally formed, and the last three, II. 5—7, were deformed. But the fact that during her pregnancy with II. 6, she fell down the stairs twice, and during her pregnancy with II. 7, fell once, might have had something to do with it. Meckel only examined II. 7, a female foetus (not indicated as stillborn on Plate). The skull was very large in proportion to the face and was very high relative to its diameters. The bones of the skull were

remarkably thin, small, and broken in many places. The bones of the face were also broken. The fractures were fresh and had probably occurred during birth. The epiphyses showed hardly any traces of ossification. The clavicles were much curved, thick, and defectively ossified for age. Some of the arm bones were fractured. The bones of the hand, especially the fingers, were abnormally short, thick and broad. The femora were much curved. The fibulae were abnormally thick and broad. The knees were everted, the legs and feet inverted. The limbs were abnormally short and thick. The weight was 4 lbs. ("4 Pfund Medicinalgewicht"). There were 43 fractures in the ribs. The nose was flat. *Measurements.* Length from vertex to sole of foot 9″ 6‴ (? seems inconsistent with measurements below); vertex to buttocks 8″ 9‴; of head from vertex to chin 3″ 4‴. Breadth of head between parietal eminences 2″ 9‴. Length of neck 0; trunk 5″ 5‴. Maximum circumference of the trunk at the upper part of the abdomen 8″ 3‴. Length of the upper extremities 3″ 9‴; hands 1″ 3‴; carpus and metacarpus 10‴; fingers 5‴. Maximum circumference of the upper limbs 4″ 6‴. Breadth of the hand 1″ 1‴. Circumference of finger 10‴. Total length of lower extremity 3″ 10‴; of foot 1″ 8‴. Breadth of foot 1″. Length of toes 6‴. Circumference of toes, with the exception of the great toe which was twice as thick as the normal, 9‴. From the general account of this case, it seems probable that it was one of achondroplasia: the multiple fractures suggest rickets or rickety pseudo-achondroplasia. The distinction had not been made at that day, and most of the measurements give little help; no statement is made of the amount of force used in delivery, and the fractures may have been due to this. (Bibl. No. 69, pp. 45 and 9.)

Fig. 641. *Wood and Hewlett's Case II.* No statement is made with regard to I. 1. I. 2, said she was worried and had received a shock in the fifth month of her pregnancy with II. 3. Her two elder children, II. 1—2, were normally developed. At birth of II. 3, the labour was difficult and forceps were used. The nurse noticed that his arms and legs were very short. At 12 months old he was unable to hold his head up and his back was very much curved. Massage improved the condition of his neck muscles. He walked for the first time at 2 years of age. His nasal breathing was obstructed at birth. When seen at age of 7, he was an achondroplasic dwarf, 3′ 1″ high, and weighing 2 stone 10½ lbs. He was a bright, active boy, who went to school, and could read small words and write his own name. He could bend down and kiss his toes without bending his knees. The circumference of his head was 22½″, the forehead was overhanging, the bridge of the nose very depressed, the palate very highly arched. The short ribs with normal sternum and normal spine produced deformity of the chest. Extension at the elbow could not occur beyond an angle of 135°, on account of the large bosses at the ends of bones. The hands were spade-like, the fingers tapering to the extremities and spread out like the ribs of an open fan. The wrists and fingers were very supple. There was marked lordosis of the back both in the erect and recumbent position. The lower limbs measured 13″ from the antero-superior spines to the inner malleoli. The femora were straight. The hair was fine and inclined to be curly, with well-marked "cow-lick" in the frontal region. *Measurements.* R. arm: acromion to external epicondyle of humerus 4¾″; greatest length of ulna 4¼″; length of hand from styloid process of radius 3¾″. Thorax: at nipple line 20″; in furrow below nipple line 19¼″. Back from seventh cervical spinous process to tip of coccyx 15¼″. Waist at level of umbilicus 18½″. Lower extremity: R. femur from trochanter to external condyle 7¼″; R. fibula 5¾″; length of foot 5½″. Greatest circumference of upper arm 7″; calf 7½″. Distance between inner angle of eyelids 1¼″. The boy was under observation for nine months and increased 1″ in height and 2½ lbs. in weight. (Bibl. No. 420, p. 90.)

Fig. 642. *Wood and Hewlett's Case III.* I. 1, died aged 48 of cerebral palsy. Nothing is stated with regard to I. 2. I. 1, and I. 2, had seven children, II. 1—7. II. 6, died aged 4 months. II. 7, died at birth. II. 3, aged 8¾, was the third of the five survivors. He crawled till he was 4 years old, but since then had walked. He went to the State School, knew his letters, could spell a few words and count. The bridge of the nose was depressed, the palate high-arched, the forehead overhanging. He had had necrosis of the left upper maxilla with scarring and contraction of the lower eyelid. The toes were well formed, the fingers thickened, tapering to a point and diverging as in other cases. There was a well-marked thyroid present. There was marked lordosis when standing or lying down. The head and waist were very small, the abdomen and buttocks very prominent. He walked without a waddle. He was unable to extend the elbows beyond 135°. The fingers extended two inches below the crest of the ilium with arms hanging by sides. The mother said he had grown one inch in three years. *Measurements.* Weight 2 st. 10 lbs. 13 ozs. Height 3′. Circumference of head 22½″. From tip of acromion process to external epicondyle of humerus (R. arm) 5″. From tip of olecranon to tip of extended mid-finger 8¾″. Circumference of chest below nipple line 21″; abdomen at umbilicus 20¾″. From antero-superior spine to tip of internal malleolus (R. and L.) 12¾″. Umbilicus from ground in erect position 18″. From vertebra prominens to tip of coccyx 14″. Greatest circumference of the upper arm 6″; forearm 6½″; thigh 11″; calf 8½″. Circumference of chest high up in axilla 21½″; buttocks below crest of ilium 21″. Transverse diameter of chest 9″; buttocks 7½″. (Bibl. No. 420, p. 392.)

Fig. 643. *Wood and Hewlett's Case IV.* Of I. 1, and I. 2, nothing is stated except that no history of any similar dwarfing existed on either side of the family. They had five children, II. 1—5, all of whom were well developed except II. 5, the youngest, aged 11. She was said to have been hydrocephalic at

9 months old; she walked at 18 months and began to talk at same time. She had had otorrhoea and had always been a restless sleeper. She was a bright achondroplasic dwarf, with large head and extremely depressed nasal bridge, the end of the nose was markedly retroussé. She went to the State School, and could write an excellent essay. She could kick her forehead when sitting down, and bend down to kiss her toes. There was marked lumbo-sacral lordosis when standing or lying. The extension of the elbows was limited to 135°. The fingers were thick, conical and diverged from the mid-line. *Measurements.* Weight 3 st. 11½ lbs. Height 3′ 6½″. Distance between inner angles of eyes 1½″. R. humerus from acromion to lower part of external epicondyle 6¼″. R. ulna, greatest length 5½″. Circumference at nipple line 22″. Spine from vertebra prominens to tip of coccyx 17″. Waist at umbilicus 19¼″. R. femur from trochanter to external condyle 8½″. L. fibula 7⅜″. Length of foot 6¾″; hand 4½″. Circumference of calf 9½″. The fingers extended 3″ below the crest of the ilium as the arms hung by the sides. (Bibl. No. 420, p. 393.)

Fig. 644. *Miller's Case.* I. 1, and I. 2, were well formed and healthy, there was no history of alcoholism, syphilis, or any other disease or malformation in either them or their progenitors. They had five children, II. 1—5, of whom four, II. 1—2 and II. 4—5, were healthy. II. 3, aged 7 years, was the third son. Instruments were used at his birth owing to his large head, but there was no special difficulty. The abnormal size of the head and the short limbs were noticed at birth. He was breast-fed till the 12th month and was a healthy baby though fatter than the other children of the family. He walked at 18 months. Dentition was normal, he had twenty sound teeth, of which eighteen were milk and two permanent teeth (lower incisors). He never had any illness save chicken-pox and measles, and was especially strong, well and active. The head was large, especially the cranial vault, the nasal bridge depressed (but less so than is usual in achondroplasia), the forehead prominent, the palate not arched. The trunk was normal, the thorax somewhat depressed laterally and the ribs slightly beaded. The clavicles were normal; there was marked lordosis and the buttocks were very prominent. The arms were very short, the finger tips not extending to the great trochanter. The humeri, ulnae and radii were all short, thickened and curved, the two latter more than the former. The elbow joints could not be fully extended (150° to 160°). The hands were very characteristic, being short, thick, and flat with digits of almost equal length, the middle and ring fingers tending to separate from one another in extension, constituting the "main en trident" of Marie. The legs were short and thick, the femora especially so, while the tibiae and fibulae were curved laterally, particularly on the right side. The gait was somewhat waddling. The muscles were well developed, the skin and hair soft and natural. Apart from a slight tendency to flat foot, the feet were normal. The sexual organs were well developed and the thyroid gland seemed natural. Mentally he was quite bright, but was very shy and sensitive. *Measurements.* Patient aged 7 years 2 months. (Those in brackets are measurements of normal boy aged 7 years 3 months.) Height standing 36½″ (46″). Height sitting 23″ (23″). From vertex to umbilicus 18½″ (19¼″); umbilicus to sole of foot 18″ (26¼″). Circumference of head 21¾″ (21″). From mastoid to mastoid across vertex 14¾″ (14″). Length of clavicle 4″ (4″); humerus 5″ (8½″); radius 4½″ (6⅜″); ulna 4⅝″ (6⅝″). Distance from wrist joint to tip of middle finger 4⅜″ (5¼″). Circumference of hand 5″ (5¾″). Length of second finger 2¼″ (2¾″); third finger 2⅛″ (2½″). Circumference of middle phalanx of third finger 1⅝″ (1⅜″); of lower end of humerus 6½″ (6¾″); round crest of pelvis 18″ (22⅜″). Antero-superior spine to internal malleolus 14″ (26¼″). Length of femur (great trochanter to external condyle) 6¼″ (10½″); tibia 5¾″ (9¾″); fibula 6¼″ (10¾″). Circumference of lower end of femur above condyles 9″ (9⅝″); of middle third of leg 8¼″ (11″). (Bibl. No. 484, p. 34.)

Fig. 645. *Dide and Leborgne's Case.* I. 1, and I. 2, were normal. They had seven children, II. 1—7, of whom six, II. 1—6, were born dead. The seventh, II. 7, aged 63, was only 1 year old when he lost his mother. He began to walk late and only spoke when about 4—5 years old. He had a large head. He remained at an institution till he was 20, was then apprenticed to a shoemaker, but had to give up the trade as his sight was bad. He travelled about and at one time acted as a clown. He could read and write a little. Finally he was put in an Insane Asylum at Rennes. The remarkable things about him were the size of his head (the maximum circumference of which was 62 cm.), and his small height (134 cm.). The arms were very short. When he stood with arms extended by his sides, the palm of the hand was on a level with the great trochanter. There was a great disproportion between the length of the upper arm and forearm, the upper arm being 22 cm. long, the forearm 26 cm. long. Similarly the leg was longer than the thigh. Length of trunk from the episternal notch to the upper border of symphysis pubis was 63 cm. The lumbar curvature was very pronounced. *Measurements.* Length of humerus 22 cm.; forearm 26 cm. Total length from antero-superior iliac spine to ground 66 cm. From antero-superior iliac spine to spine of patella 27 cm. Length of fibula 27 cm. Circumference of calf 34 cm.; thigh 48 cm.; waist 72 cm. Perimeter of thorax 88 cm. The hand looked square; the fingers were of nearly equal length and divergent in extension ("main en trident"). The arms were rather incurved. The lower limbs were curved. The bridge of the nose was depressed, the intelligence very backward. (Bibl. No. 467, p. 200.)

Fig. 646. *Scharlau's Case I.* I. 1, and I. 2, were healthy and had 13 children, II. 1—13. II. 1—12, are stated to have been healthy. Nothing is said with regard to the health of II. 13. II. 14, her husband was a confirmed drunkard. II. 13, and II. 14, had four children. III. 1, the eldest was born when the

mother was aged 27, she was alive but very scrofulous, III. 2, also had a scrofulous appearance, and had an enormously thick head and exophthalmos, the third child, III. 3, a very strong boy with flat feet died, aged 1½ years, of diarrhoea and vomiting. The fourth child, III. 4, was born when the mother was 37, about three weeks before it was expected: she was not quite dead but died (not indicated on Plate) in spite of attempts at resuscitation. She weighed 3120 grammes. Her total length was 43 cm., 17 cm. of which length was the height of the head. The middle of the length was at the processus xiphoideus sterni. She was hydrocephalic, the fontanelles and sutures were widely patent, the eyes appeared small, the nose flat, the thorax broad and short, the abdomen large. The upper extremities, on which the skin lay in thick folds, were unusually short, with short broad hands. Externally no articulation could be perceived. The lower extremities were similarly shortened and were very much curved. (Bibl. No. 135, p. 411.)

Fig. 647. *Charpentier's Case.* I. 1, I. 2, and II. 1, the father, mother, brothers and sisters of II. 2, were all very tall. II. 2, aged 20, was only 1·15 m. in height. Her head and trunk were normal, whilst the extremities, particularly the lower, were short in a marked degree. She had walked at the age of 9 months and had had no disease during childhood. Her muscular power was considerable and her intelligence intact but to some extent puerile. Her very short limbs were markedly muscular and quite straight. The fingers and toes were remarkably short. The vertebral column was straight; the trunk, bust, skull and face were perfectly developed and corresponding to her age. Her head was enormous, with projecting forehead and very pronounced facial asymmetry. *Measurements.* From clavicle to vertex of skull 21 cm.; clavicle to great trochanter 50 cm.; great trochanter to patella 20 cm.; patella to sole of foot 24 cm. Upper limbs: length of upper arm 17 cm.; forearm 14 cm.; hand 12 cm.; middle finger 5 cm. She came to the Hospital for her confinement. Instruments were used and the child extracted alive, but it died shortly after. It presented the characteristics of a foetus of 6½ months old, and weighed 1520 grammes. *Measurements.* Total length 38 cm. From vertex of skull to umbilicus 22 cm.; umbilicus to heels 16 cm. Diameter occipito-frontal 10 cm.; mento-occipital 12 cm.; bi-parietal 4½ cm. (at moment of birth), 8 cm. (half-hour after). Sub-occipito-bregmatic 8½ cm. (Bibl. No. 162, p. 45.)

Fig. 648. *Lange's Case.* I. 1, and I. 2, were normal, and all the confinements of I. 2 had been normal. The pelvis of II. 2, who had been to the Hospital for her confinement, was normal. II. 3, aged 26, had been bottle-fed, but according to her mother was no different from other children at first. She began to walk at 9 months old. In her fourth year she had scarlet fever, measles and quinsy. At about this time the curvature of the lower limbs and the remarkable shortness of her arms were noticed, but walking was neither impossible nor painful. The doctor said she was rickety. She menstruated at 14. In order to avoid being noticed in the streets she had, since the age of 12, always been carried or driven, consequently she was easily tired and could scarcely walk twenty paces without help. In 1882 she had intercourse with a man, II. 4, aged 22, and 191 cm. in height. The pregnancy was normal. She was 92 cm. in height and a fairly well nourished person, and showed a certain degree of intelligence in her method of answering questions. The size of her head and the length of her trunk were noticeable in contrast with the shortness of the extremities. The head was rather dolichocephalic, the lower jaw showed no sign of rickets. There was no deviation of the spinal column, the chest was normal except for a slight rosary, the clavicles were normal. The lower limbs were shortened, curved forwards and remarkably hairy. The thighs were curved forwards. The lower epiphyses of the thigh-bones were slightly enlarged, those of the tibiae very much enlarged. The upper limbs were short, both arms and forearms showed slight curvature with concavity forwards. The epiphyses of the bones of the forearm were greatly thickened. The abdomen was somewhat distended. The external measurements of the pelvis were: distance of spines 18·8 cm.; distances of crests 19·2 cm.; external conjugate 13·7 cm. The child, a female, III. 3, was dead before birth. There was not much difficulty in its extraction. It was normally formed: weight without brain 2110 grms., length 49 cm., circumference of shoulders 34 cm. *Measurements of mother*, II. 3. Circumference of head 49·5 cm. Length of trunk from the tip of the spinous process of the seventh cervical vertebra to the tip of the coccyx 50 cm. Bi-acromial breadth measured in front 29 cm. Lower limbs, from the highest point of the great trochanter to the heel 35 cm. Thigh from the highest point of the great trochanter to the lowest point of the external condyle of the femur 18·5 cm. Length of leg from the external condyle of the femur to the heel 16·5 cm.; foot from the heel to top of the great toe 15·5 cm.; upper arm from tip of the acromion to tip of olecranon 19 cm.; forearm from tip of olecranon to the styloid process of the ulna 13·5 cm.; hand from the radio-carpal joint to the distal extremity 8 cm. Weight 26 kilos. II. 3, recovered from her confinement and left the Hospital. (Bibl. No. 201, p. 753.)

Fig. 649. *Auché's Case.* I. 1, I. 2, I. 3, and I. 4, were all well made and there had never been a dwarf in their families. II. 2, died from an accident at the age of 32. II. 3, aged 26, was healthy, and neither she, nor II. 2, was tuberculous, syphilitic or alcoholic. II. 3, was well made, and her height was 1·62 m. III. 2, the younger child, aged 18 months, was normal. III. 1, was born at term, the confinement was normal. At birth the large size of the head and the shortness of the legs were noticed. The child began to walk at the age of 14 months, but quickly became tired. The first tooth appeared at the age of 9 months; the last of the milk dentition at 2 years. The child began to speak late, but the intelligence

appeared normal. It had congenital inguinal hernia. The forehead was high and prominent, the face small in proportion to the cranium, but with large features. The bridge of the nose was broad and depressed, the extremity large and slightly retroussé. The mouth was kept almost constantly open. The palatine vault was high-arched. The trunk was normal, there was marked lordosis but no abnormal curvature of the vertebral column. When standing with arms extended by the sides, the palm of the hand was a little above the iliac crest. The hand was fleshy and square, with fingers nearly equal in length, and exhibited the features of the "main en trident." The foot was fleshy. The limbs showed no curvatures. The muscles were well developed and genital organs normal. *Measurements.* Height 73 cm. Circumference of head 53 cm. Bi-parietal diameter 17·5 cm. Maximum fronto-occipital diameter 21 cm. Total length of upper extremity 30·5 cm. Length of upper arm 11 cm.; forearm 11 cm.; hand from radio-carpal joint to distal extremity of middle finger 8·5 cm.; lower extremity from great trochanter to sole of foot 31·5 cm.; thigh 13 cm.; leg 13·5 cm.; foot 12 cm. (Bibl. No. 517, p. 116.)

Fig. 650. *Porak's Case II.* I. 1, and I. 2, were healthy. II. 1, aged 27, was their only child. Her height was 115 cm. She possessed the trunk and head of an adult; the head seemed too large, the root of the nose was depressed and she was very stout. Her extremities were very short and incurved, her mental faculties were intact. The upper extremities measured 40 cm. from the acromion to the end of the fingers; the lower extremities measured 48 cm. from the great trochanter to the soles of the feet. The clavicles were normal, the joints of the limbs large and the vertebral column straight, but there was an exaggerated lumbar curvature. She came to Hospital for her confinement. She said it was her first child. The child, III. 1, was extracted in pieces and therefore could not be examined. She went for a second confinement to Dr Ribemont Dessaignes, who told Porak he had performed Caesarian section and extracted a living child, III. 2, which exactly resembled its mother. The child was a girl and weighed 3650 grammes. Porak obtained the following particulars. *Measurements.* Foetal length 48 cm. Length from crown of head to umbilicus 30 cm.; umbilicus to soles of feet 18 cm. Head: occipito-frontal diameter 11·5 cm.; mento-occipital diameter 12 cm.; bi-parietal 11 cm.; sub-occipito-bregmatic diameter 10 cm. The mother died, the father took the child but afterwards abandoned her, and she died at a charitable institution for children. (Bibl. No. 247, p. 21.)

Fig. 651. *Marconi's Case.* No statement is made with regard to I. 1. I. 2, had always enjoyed good health, although she had suffered from venereal disease ("affezione celtiche"). She had had five children, II. 1—5, all breast-fed. Four of them, II. 1—4, were alive and healthy. II. 5, had died aged 2, the cause of death was not remembered by the mother. When seen I. 2 had been pregnant six months, she came for advice because she had floodings (décollement) of water followed by a considerable loss of blood at night between 2 and 3 p.m. Her general condition was good, her conformation regular and normal. A medical examination was made and a decision with regard to treatment arrived at. Finally an enormous placenta was expelled normally. The foetus was of the female sex and weighed 700 grammes. It looked like a soft raspberry-coloured mass of gelatinous texture, with a form roughly human and rudiments of hands and feet. What was most remarkable was the disproportion between the various parts of the body, the shape and shortness of the limbs compared with the almost normal size of the trunk, and the enormous head. The foetus was radiographed and Marconi states he believes it was a typical case of achondroplasia. (Bibl. No. 485, p. 634.)

Fig. 652. *Cestan's Case.* I. 1, and I. 2, were healthy. No syphilis, no alcoholism. II. 1, aged 12, was well made. II. 2, aged 9½, was born at term. The mother noticed the arms were short at birth. She walked and spoke at the age of 16 months. *Measurements.* Height 0·93 m. Height of head 21 cm. Breadth of head at the parietal eminences 17 cm. Upper arm 13 cm. Forearm and hand 22 cm. The back was flat, but the buttocks projected so that dorso-lumbar curvature was produced. The scapulae projected slightly; the pelvis exhibited an apparent arrest of development. The head was large, the nose large and flattened. The limbs were very short and "main en trident" was very pronounced. (Bibl. No. 385, p. 277.)

Fig. 653. *Winkler's Case.* I. 2, a strong muscular woman, had five children, II. 1—5. II. 1—4, were healthy. II. 5, who died at birth achondroplasic. When born it weighed 3850 grammes. *Measurements.* Length to umbilicus 28 cm.; to buttocks 35 cm. (of which 15 cm. was head height); to foot 44 cm. Transverse diameters: at shoulders 11·5 cm.; at buttocks 10·5 cm.; of head (ant.) 9·5 cm.; of head (post.) 11·5 cm. Occipito-bregmatic diameter of head 10·5 cm.; occipito-frontal diameter 13·5 cm.; sub-occipito-frontal diameter 10·5 cm. Circumferences of head (occipito-bregmatic) 4·1 cm.; (sub-occipito-bregmatic) 3·9 cm. The bones of the head were remarkably well ossified, the spine defectively ossified. Chest normal, clavicles normal. The scapula was less ossified, the cartilaginous parts predominated. The cartilages at the joints were remarkably thickened. Upper limbs: The epiphyses were everywhere thickened, although without all the centres of ossification. The diaphyses were all very short with an enormously thick cortical layer. The curvature of the humerus and the ulna was towards the front (nach vorn), that of the radius outwards. The pelvic girdle was smaller than normal, in shape a transverse oval. Lower limbs: The epiphyses were remarkably thickened but purely cartilaginous. The diaphyses very

short, with very thick compact outer layers. The femur and tibia were curved somewhat inwards below, the fibula curved a little backward. (Bibl. No. 143, p. 101.)

Fig. 654. *Megnier's Case II.* This is probably a case of achondroplasia with myxoedema. I. 1, died aged 60, of diphtheria. I. 2, died aged 64, of cerebral apoplexy. II. 4, died in childbirth, her son, III. 1, aged 17, was of low stature. II. 5—7, were alive and of low stature. II. 1—2, died at an early age. II. 8, aged 36, was 1·60 m. high, and had had syphilis, but had shown no signs of it for 12 years. He had been married seven years. His wife, II. 9, aged 30, suffered from sciatica and habitual headache. Her mother, I. 4, had died aged 57, of paralysis of the bladder. Her father, I. 3, had died aged 57, of a tumour in the loins; of her brothers and sisters, a brother, II. 11, died aged 17, of meningitis; a sister, II. 10, alive, was cardiopathic, and of the other four living brothers, one, II. 12, had disease of the bladder. They were all tall. II. 9, never had syphilis. Her first child was III. 2, an achondroplasic girl, after three years she had a miscarriage at three months, III. 3; and about two years later another miscarriage, III. 4. III. 2, aged 6 yrs. 3 mths., was born at term and breast-fed for three months. When born, the mother noticed that her legs were short. She began teething at 18 months old, and the teething continued till she was 5 years old. At the age of 2, she had gastro-enteritis for a month, otherwise had had no illness, but was dyspeptic. When a year old she could not lift her head from the trunk, at 4 years the erect posture was possible but when seen it was still difficult. She had umbilical hernia, but it was almost cured. From the age of 2 to 5 she did not appear to have much intelligence. Her general appearance was cretinoid. The thorax was compressed at the sides and projecting in front, and unduly wide at the base, and no rosary existed. There was no lateral curvature of the spinal column. The abdomen was very prominent. *Measurements.* Length of the sternum 10·5 cm.; clavicle 10 cm. Length from the episternal notch to the umbilicus 20 cm.; episternal notch to the upper border of symphysis pubis 48 cm.; distal extremity of xipho-sternum to upper border of symphysis pubis 18·5 cm.; vertex to the upper border of symphysis pubis 33 cm.; upper border of symphysis pubis to the ground 31 cm.; antero-superior iliac spine to base of feet 37 cm. Maximum pelvic circumference 48 cm. The limbs appeared thick and solid, the calves greatly developed, the hands square, the toes large, and the feet thick and fleshy and rather flat. Length of thigh from the antero-superior iliac spine to the external condyle 18·7 cm.; great trochanter to external condyle 16·5 cm.; leg 16·5 to 17 cm. Maximum length of feet 12·8 cm. Length of first toe 2·4 cm.; second toe 2·3 cm.; third and of fourth toes 2·2 cm.; fifth toe 1·9 cm. Length from acromion to distal extremity of middle finger 31 cm. Length of upper arm from acromion to olecranon 11·5 cm.; forearm from olecranon to the styloid process of radius 9·4 cm. Transverse diameter of hand to the beginning of fourth finger 6 cm. Length of index finger 3·8 cm.; middle finger 4·1 cm.; ring finger 3·7 cm.; little finger 3·1 cm. Radiographs of the lower limbs showed that the bones were regular in form, that the epiphyses although somewhat deficient in the part which was still cartilaginous, were notably enlarged, whilst the diaphyses had their volume much reduced in the middle part. (Bibl. No. 466, p. 486.)

PLATE LIII. Fig. 655. *Horand's Case I.* I. 1, and I. 2, were normal and had never suffered from rheumatism, tuberculosis or syphilis. Of their children, II. 4 was well-made; II. 3 was very small, but nothing further is said of him; II. 2 was of medium height, well made and had never had rheumatism, tuberculosis or syphilis. She married II. 1, who had never been ill, was very tall and robust and was neither alcoholic, syphilitic, nor tuberculous. They had three children, III. 1—3. III. 1, a girl, aged 8½ was tall, handsome and very intelligent, height 1·29 m. III. 3, a girl of 10 months old, was healthy and well made. III. 2, aged 6, was born when II. 1 was aged 30 and II. 2 aged 27. The birth was normal; he was breast-fed and weaned at 14 months old, he then had violent headaches. He had his first tooth at 6 months old, began to speak at 1 year and to walk at 14 months. At 4 years of age he became deaf in both ears for a while. His head was large in comparison with the trunk; all the fontanelles were closed. The frontal eminences were very prominent, he had a large forehead, slight facial asymmetry and short nose flattened at the bridge. The eyelids had no cilia; the palate was high-arched and the neck short. The trunk was of normal dimensions, the limbs very short. When standing with arms extended by the sides the extremity of the middle finger reached to just below the great trochanter. The humerus was thick and compact with slight outward curvature; its epiphyses were broad and thick. The arms were somewhat abducted from the trunk. The hands were large, thick, fleshy and almost square; the fingers were nearly equal in length, short and thick, and diverged in extension so as to form the "main en trident." The lower limbs were very short and thick. The patellae were much nearer the inguinal folds than is normal. The lower extremities had two curvatures, the one with an anterior concavity from the antero-superior iliac spine to the great toe and one with an external concavity from the antero-superior iliac spine to the external malleolus; the curvature was more marked on the left side owing to a slight degree of genu valgum with an inward rotation of the tibia. The feet were large and broad, the plantar arch rather flattened. The great toe diverged markedly from the second toe. The buttocks were prominent and the joints of the limbs large. There was exaggeration of the lumbar curvature. His intelligence was fair; he went to school. The genital organs were well developed. The thyroid gland was not enlarged. *Measurements.* Weight 17·50 kg. Height given twice and differently as 85 and 89 cm. L. humerus 12½ cm.; radius 12 cm.; ulna 12½ cm. R. humerus 13 cm.; radius 11 cm.; ulna 13½ cm. R. lower extremities from antero-superior iliac

spine to external malleolus 36 cm. L. lower extremities: antero-superior iliac spine to external malleolus 36 cm.; femur (from the antero-superior iliac spine to the line of articulation of the knee joint) 21 cm. The R. tibia 13½ cm. L. tibia 14½ cm. (Bibl. No. 486, p. 927.)

Fig. 656. *Parhon, Shunda and Zalplachta's Case.* This is an undoubted case of achondroplasia, associated with kyphosis in the lower dorsal region and kypho-scoliosis above this. Probably these are rickety in origin; they may be statical, or of adolescence, or possibly but not probably of achondroplasic origin. I. 1, was alive and of medium height. I. 2, was dead, cause unknown. They had nine children, II. 1—9, of whom three, II. 1—3, were dead; five, II. 4—8, were alive and of normal height. II. 9, aged 33, was exhibited in a fair. The palatine vault was high-arched; the hair was long and glossy; beard and moustache were well grown. The limbs were very short. When the arms were extended by the sides, the hands just reached the level of the upper border of the great trochanter. *Measurements.* Height 105·5 cm. Circumference of head 57 cm.[1] Anterior semi-circumference 31·4 cm. Posterior semi-circumference 20·5 cm. Antero-posterior circumference 37·5 cm. Transverse circumference (measured from insertion of one ear to that on the opposite side or from one zygomatic apophysis to the other) 35·5 cm. R. middle finger 6·8 cm. R. hand (from styloid process of radius to base of index finger) 6·5 cm. R. forearm (from the summit of the olecranon to the styloid process of the ulna) 16 cm. R. upper arm (from internal epicondyle to acromion) 16·5 cm.; (from acromion to external epicondyle) 16 cm. R. foot (from posterior surface of calcaneum to distal extremity of great toe) 16·8 cm. R. leg (from the line of the knee joint to the external malleolus) 17 cm. R. femur (from great trochanter to external condyle) 18·5 cm. The "main en trident" present. Kypho-scoliosis in the upper half of the dorsal region and very pronounced kyphosis in the lower. The lumbar curvature appeared exaggerated. The mental condition was rather puerile. (Bibl. No. 488, p. 539.)

Fig. 657. *Macewen's Case.* I. 1, and I. 2, were normal as were all their other relatives. Of their eight children, six, II. 1, were normal. II. 2, who immediately preceded II. 3 in age, was slightly under medium height and was affected by rickety curves of the bones of the lower extremities. She did not walk till she was 7. II. 3, aged 16, did not speak till she was 2½ years old and was 4 years old before she began to walk, crutches being required at first to aid her. Besides the usual illnesses of childhood she suffered from general ill-health and was always feeble; the deformity of the bones began to appear when she began to walk. She was 3′ 6″ in height, markedly stunted and of heavy build but active and with her intelligence practically perfect. Her head was large and the root of the nose depressed but prognathism was only slightly marked. The limbs were markedly affected, the humeri, radii and ulnae being much curved with greatly enlarged extremities. The trident hand was well marked though the fingers were a little long. The curving of the femora and tibiae was pronounced. There was marked lordosis with consequent protuberance of the abdomen. The central point of the body was well above the umbilicus. (Bibl. No. 533, p. 1646.)

Fig. 658. *Rankin and Mackay's Case.* No statement is made with regard to I. 1. I. 2, was healthy. She had three sons, II. 1—3. II. 1, aged 12, was normal. II. 2, died of "wasting" aged 3. II. 3, aged 9, was born normally but was misshapen at birth. Apart from deformities he was healthy and vigorous and his intelligence was above the average. He was well nourished with a healthy and smooth skin. His head was abnormally large, the dome of the cranium high and the occipital region unduly prominent. The nose was flattened and depressed at the bridge, the mouth partially open. He was pronouncedly prognathous. The palate was arched and high and the voice nasal. The trunk was of average size, the sternum projected forwards, and there was distinct costal rosary and well-defined Harrison's sulcus. The abdomen was protuberant partly on account of lordosis of the spine. The gait was clumsy. The arms were thick and short, and when extended barely reached the tips of the great trochanters. All the joints were prominent. The shaft of the humerus was short being only ⅝″ longer than the clavicle, and the bones of the forearm were shortened, with a decided increase in the normal curve of the radius. The hands were "remarkably foreshortened," the fingers tapering towards points and deviating from one another like the spokes of a wheel. In the legs there was a general shortening of the bones, an increase of normal curvature and enlargement of the extremities. The longitudinal arch of the foot was destroyed giving rise to flat foot. *Measurements.* Height 2′ 11″. Sitting 2′ 1″. Weight 2 st. 7½ lbs. Circumference of head 20¾″. Tip of mastoid to tip of mastoid 15″ (? over vertex). Clavicle 4″. Acromion to external epicondyle 4⅝″. Radius 3⅜″. Olecranon to styloid process of ulna 4¼″. Antero-superior iliac spine to internal condyle 7¼″. Internal tuberosity of tibia to malleolus 5½″. Chest, full expiration 20½″. (Bibl. No. 518, p. 1522.)

Fig. 659. *Romberg's Case I.* Nothing is stated about I. 1, and I. 2. II. 1—2, were twin foetuses of the ninth month in the Royal Anatomical Museum of Berlin, one male and one female. Both had large heads; in the female foetus, II. 2, the occiput protruded considerably as a distended sac. The chest and abdomen were unusually large, the extremities excessively short, about one-fourth of the usual length. The feet appeared to be turned in. The head was larger than usual and bulging laterally, but the ossification was normal. The neck was of the usual length. The thoracic cavity was proportionally bigger than normal, for the ribs formed a relatively large arch as they approached the sternum. Both ribs and

[1] Does not appear to be sum of two semi-circumferences.

clavicles were normally ossified. The extremities were markedly abnormal. In the upper the humerus formed an angle with the scapula which was more obtuse than acute and was curved, the convexity being external. The ulna and radius, throughout their middle parts, showed a curvature with convexity outwards in the same line as that of the humerus, but then curved inwards, so that the whole arm was excessively shortened and curved. The pelvis was normal. The femur was curved so that it had an anterior concavity and formed an obtuse angle with the pelvis. The tibia and fibula almost formed a semi-circle, with external curvature to middle portion and then curving in. (Bibl. No. 61, p. 28.)

Fig. 660. *Romberg's Case II.* I. 1, and I. 2, were healthy. I. 2, said that in the first months of her pregnancy she had been liable to spasms which affected her throat. II. 1, was born with deformed limbs. The head was bigger than normal, the face swollen and reddish in colour. The fontanelles were widely opened, especially the posterior. In the posterior superficies of the skull the defect of the occipital bone was supplied by a thick cartilaginous elastic membrane. The calvarial bones were separated by membranous spaces. The neck was very short. The thorax was flat and depressed in shape and the arch of the ribs looked less convex than in healthy children. The limbs were greatly deformed. Both humeri were curved, with convexity externally, so that the arms appeared contracted and short. The L. humerus was much depressed at the site of insertion of the deltoid muscle. The forearm formed an obtuse angle with the arm. There was an external convexity as far as the middle third of the limb, and it then again curved inwards. The femora almost formed a semi-circle and the tibia showed curvatures with convexity outwards ; the soles of the feet were turned in. The skin of the whole body was flaccid and soft to the touch. Romberg saw her again six months later and she appeared worse. The fontanelles were still wide open. There was no sign of teeth. She had difficulty in breathing and was very emaciated. (Bibl. No. 61, p. 22.)

Fig. 661. *Treub's Case.* I. 1, and I. 2, were of normal size and no case of achondroplasia was known among their ancestors or collateral relatives. It does not state whether I. 1 and I. 2 had any normal children. II. 2, was achondroplasic, she lived with a man of normal stature, II. 1. She was intelligent enough but troublesome and it was with great difficulty she was photographed. Caesarian section was performed and a normal girl, III. 1, was extracted who weighed 3000 gms. and whose length was 49 cm. No other measurements of either mother or child are given nor does it state whether they lived or died. II. 3, a younger sister of II. 2, was said to resemble her in every particular, and there was also a brother, II. 4, who had died aged 2, and who was said to be a dwarf. (Bibl. No. 471, p. 58.)

Fig. 662. *Daniel's Case.* II. 2, denied ever having had syphilis and said there was no degeneracy in his parents or ancestors, or collateral relatives ; his father, I. 2, had a twin sister, I. 1. I. 4, aged 52, was healthy; I. 3, aged 55, had when 12 years old an affection of the joint of the right leg, in consequence of which it was shorter than the left by "quatre travers de doigts." II. 4, aged 25, was well formed. The original runs "La femme (II. 3) a une sœur parfaitement bien constituée. Au point de vue obstétrical, elle a eu deux accouchements, à sept mois, enfants mort-nés." As further on it states II. 3 was a primipara, presumably it was the sister, II. 4, who had the two still-born children, III. 2—3, and they have been so entered in the pedigree. II. 3, aged 29, a "crocheteuse," came to hospital for her confinement. She had walked at 1 year old, never had had any serious illness, had measles in infancy and rheumatic pains at age of 13. She had been married seven years, had no trace of syphilis or genital troubles and denied alcoholism. She was tall and robust and this was her first confinement. The trunk of the child was expelled normally, but when they were trying to extract the head, and were using no violence, it suddenly burst and the brain hemispheres were thrown on the bed. The child, III. 1, was a female and weighed 1250 gms., the trunk was almost normal, but the shortness of the limbs was noticeable. Two sets of measurements are given, in two cases they are not in agreement so both are recorded here. *Measurements.* Total length of body from the occiput to the heels 32·8 cm. (32 cm.). Distance of umbilicus from heels 13·8 cm.; from occiput 19 cm. (18·2 cm.). Total length from the coracoid process to tip of middle finger 11·1 cm. Length from the coracoid process to elbow (forearm bent) 4 cm. ; elbow to wrist (hand bent) 4 cm. Length of hand from the wrist to tip of middle finger 3·1 cm. Total length from great trochanter to heels 9·7 cm. Length of thigh from great trochanter to knee (lower part of leg bent) 4·7 cm.; from the knee to the heels 4·4 cm. ; of foot from heel to tip of great toe 5 cm. (Bibl. No. 456, p. 30.)

Fig. 663. *Poynton's Case.* I. 1, and I. 2, were healthy and had eight healthy children, II. 1—8. II. 9, aged 7, was a full-term child. He had a large, square head, circumference being $21\frac{1}{2}''$. His expression was intelligent and the intelligence was normal. The trunk was disproportionately long in comparison to the length of the extremities, which were very short. There was well-marked projection of the nates. The disposition of the fingers was ray-like. The external auditory meatus was directed inwards and not inwards and forwards. *Measurements.* Length of trunk $13''$; humerus $2\frac{1}{2}''$; forearm $3\frac{1}{8}''$. Distance from antero-superior iliac spine to adductor tubercle $9\frac{7}{8}''$. Distance from the knee joints to the internal malleoli $5\frac{1}{4}''$. (Bibl. No. 527, p. 431.)

Fig. 664. *Nijhoff's Case I.* I. 1, 2, 3 and 4, were normal. II. 1, was "a dwarf." II. 2, normal. These two had eight children, four boys and four girls, of which three boys and three girls were "dwarfs,"

and the last two girls, at any rate, were achondroplasic. III. 1, "a dwarf," died unmarried. III. 2, also "a dwarf," married a normal woman, by whom he had one male child, IV. 2, normal, and a child, IV. 1, who died young. III. 4, "a dwarf," died before 30 years of age "in parturition"; no note as to characteristics of child, IV. 3. III. 6, normal, married, III. 7, a normal woman, and by her had five normal children, IV. 4—8. III. 8, "a dwarf," married (presumably to a normal woman), has no children. III. 10, normal, married to a normal man, III. 11, has two normal female children, IV. 9 and 10; III. 12, "a dwarf," typically achondroplasic, married to III. 13, a normal man, has had one child, a normal female, IV. 11, delivered by Caesarian section. III. 14, "a dwarf," typically achondroplasic, married to a normal man, has one child, a female, achondroplasic, IV. 12, delivered by Caesarian section. Professor Nijhoff kindly furnishes the following details concerning III. 12 and III. 14. III. 12, Aaltje B. Height 122 cm. From excellent photographs (see Plate PP, (89), (90)), very kindly furnished by Professor Nijhoff, it can be clearly seen that she is typically achondroplasic. Married, Caesarian section performed for delivery of first child, IV. 11, in 1890, in Dr Sanger's Clinic, Groningen. Both mother and child survived. The latter, a normal female, lived 9 years. III. 14, Janna B., sister of III. 10, aged 41 years; height 123 cm. *Pelvic measurements.* Interspinous 22 cm. Intercristal 23 cm. Intertrochanteric 31 cm. External conjugate 17 cm. Diagonal conjugate 7 cm. Delivered of her first child, IV. 12, by Caesarian section by Professor Nijhoff, in the Groningen Clinic, on Oct. 14th, 1899. From very fine photographs (see Plate PP (91), (92)) kindly furnished by Professor Nijhoff it can be seen that she is typically achondroplasic. Professor Nijhoff also states that the child, a female, was achondroplasic, and furnishes the following measurements of it compared with those of a normal child: Weight 3170 gms. Total length 47·5 cm. (50 cm.). Length of arm above elbow 8 cm. (11 cm.), below elbow 7 cm. (9·1 cm.); middle finger 3·0 cm. (4·5 cm.); leg above knee 9·5 cm. (11·5 cm.), below knee 7·5 cm. (10·5 cm.); foot 7·0 cm. (8 cm.); big toe 1·8 cm. (2·6 cm.). (See Bibl. No. 396.)

Fig. 665. *Lunn's Case.* I. 1, was 6′ 2″ in height, I. 2, 5′ 10″ in height. They had five children, II. 1—5, of whom II. 1—3 and II. 5 were all in good health. II. 4, aged 53, had been stunted from birth; he lost the use of his limbs for six months at the age of 3, and since then his legs had been bowed. At the age of 17 or 18 he became a coal porter, which occupation he followed for nine years, since then he had been a road labourer in Paddington. He had always enjoyed good health except for bilious attacks, but eight years ago he began to suffer from sciatica which eventually compelled him to give up work. His forehead was prominent, the bridge of the nose rather deficient, the eyes deeply set, the palate arch rather high. The legs were very bowed, there being great curvature of the tibiae. The feet were short and square, the toes being nearly all of the same length. There was little curvature in the bones of the upper limbs. The fingers were more nearly of the same length than normal, and the middle and ring fingers diverged at the first interphalangeal joint. He had lordosis and prominence of the abdomen. The umbilicus lay midway between the crown of the head and the soles of the feet. The finger tips reached $1\frac{1}{4}$″ below the great trochanters. The genital organs were normal and the intellect good. *Measurements.* Height 4′ 6″. Sitting height 2′ 9″. Circumference of head 24″. Length of clavicle $5\frac{1}{2}$″. Distance from the acromion process to the external condyle $6\frac{3}{4}$″. Length of radius $6\frac{3}{4}$″. Distance from olecranon to styloid process of the ulna 8″. Distance from the antero-superior iliac spine to the internal malleolus R. $18\frac{1}{4}$″, L. $18\frac{1}{2}$″. Distance from the internal tuberosity of the tibia to the internal malleolus $7\frac{1}{2}$″. Chest full expansion 35″. (Bibl. No. 536, p. 252.)

Fig. 666. *Swoboda's Case.* Of I. 1, I. 2, I. 3, and I. 4, nothing is stated. II. 2, had been seven years in an insane asylum. II. 3, was a waiter, of middle height and very nervous. II. 4, a waitress, was tall and healthy as were also her eleven brothers and sisters, II. 5—15. There was no trace of syphilis and alcoholism was denied. II. 3, and II. 4, had six children of whom III. 1, aged 10, was the eldest, III. 2—6, were normal, and there had been three miscarriages, III. 7—9. When III. 1 was born, no shortness of limbs was noticed but she had congenital left-sided genu valgum and flat feet. The grandmother said III. 1 was small and thick at birth and so soft and flabby they were afraid to lift her. If the disproportion between the body and limbs existed it was not noticed; from photographs taken at age of 2 and 3 years one could only gather that the hands had the characteristic triangular form and only reached to the trochanter. The child was weakly at first, but learnt to walk at the end of the first year, she had a large head and was thought rickety. In consequence of sitting a great deal a high degree of lumbar kyphosis developed. The mother had her examined about 40 times by doctors who said she had rachitis. At age of 6 months she began to teethe, and when she began to walk the lumbar kyphosis changed to lordosis. She soon became strong and muscular. She was brought to hospital the last winter (? 1897) for infectious vulvitis and was recognised as an achondroplasic dwarf. She had a projecting lower jaw with prominent forehead and depressed nose. The trunk and neck were of normal length and the thyroid gland was normal. The sternum was broad and thick, the abdomen abnormally large. The genitals were normally developed with an abnormal amount of pubic hair for her age. The hands were short and broad and of characteristic main-en-trident shape. The three middle toes of each foot had also the triangular shape. The congenital genu valgum had cured itself. Her weight was 23 kilos and her intelligence well developed. *Measurements.* Height 104 cm. Circumference of head 54 cm. Length of sternum 14 cm.

Circumference of thorax 60 cm. Diameter of chest 11 cm. instead of a normal 19·75 cm. Length of upper arm 15·5 cm.; forearm 15 cm.; thigh 15·5 cm.; leg 20 cm. (Bibl. No. 429[b], p. 670.)

Fig. 667. *Keyser's Case.* There are very few details given of this case. I. 1 was 4′ 8″ in height. I. 2 was normal, only the one child is mentioned, II. 1, aged $2\frac{2}{12}$ years. The bridge of the nose was markedly depressed; with hanging arms the hands only reached to the umbilicus, the hands were trident shaped, kyphosis was present but no lordosis. *Measurements.* Height 25·75″. Circumference of head 19″. Length of humerus 3″; forearm 3·25″; femur 5·5″; leg 3·75″. (Bibl. No. 526, p. 1602.)

Fig. 668. *Durante's Case.* I. 1, and I. 2 died of phthisis. II. 2 had never been strong, at the age of 28 she had bronchitis with haemoptysis. She married when aged 29 and her first child, III. 1, was alive and healthy. At age of 32 or 33 she became pregnant a second time and was seized with violent vomiting day and night, which could not be stopped. The doctor tried to procure abortion on account of her health but failed. She came to hospital, and showed symptoms of tuberculosis and hysteria, and also had lateral nystagmus. She died in the fifth month of her pregnancy. After death the child was extracted by Caesarian section. It had short, cylindrical, sausage-shaped, incurved limbs covered with thick skin which lay in folds. Measurements of the foetus are given. (Bibl. No. 412, p. 812.)

Fig. 669. *Smeeton's Case.* I. 1 was a poor fisherman; he and his wife, I. 2, were of ordinary height and had eight children, II. 1—8, of whom seven, II. 1—7, were normal. II. 8, Wybrand Lolkes, was born at Jelst, in Western Friesland, 1730. He showed great mechanical talent and was apprenticed to a watchmaker and became a very clever workman. He went to Rotterdam and married, but business not being flourishing, he exhibited himself in many Dutch towns and then went to London and was exhibited by Astley in 1790. He was then 60 and measured 27″. His wife always appeared with him. They had three children, III. 1—3, one of whom, a son aged 23, was 5′ 7″ high. II. 8, died in Holland. His portrait is reproduced by Smeeton and is also in *Le Magazin Pittoresque*, 1839, p. 333. (Iconography, No. 142.) He is described by Regnault (Bibl. 411) as achondroplasic, also referred to in the Introduction (see our pp. 360 and 362). His picture in Smeeton's *Biographia Curiosa* looks achondroplasic but the trunk seems rather shorter than normal. Iconography, Nos. 162, 164. (See Bibl. No. 69[b], p. 38.)

Fig. 670. *Méry and Labbé's Case.* There was nothing abnormal about I. 1 and I. 2. I. 2, had 12 pregnancies. Two, II. 1—2, ended in miscarriages at 2 months and $3\frac{1}{2}$ months respectively. Four children, II. 3—6, died of gastro-enteritis before the age of 3 months. Six children survived, II. 7—12. II. 7, was healthy and serving as a soldier. II. 8—10, were healthy. II. 11, at the age of 5 months had Potts' disease which was cured, but left a fairly pronounced gibbosity. II. 12, aged 12, was a dwarf. Apparently he had had all the diseases of childhood, scarlatina, measles, varicella and meningital (?) attacks, and for many years had chronic blepharitis. He was born at term, and the abnormal size of his head and the shortness of his limbs were then noticed. His limbs were so short that his mother could not carry him comfortably on her arm till he was 5 years old. He had always grown slowly and was about the size of a child of 4 years of age. The trunk was nearly normal, the limbs short and thick, the head large. Standing with his hands hanging by the sides, the end of the hand did not reach the level of the great trochanter. The right hand hung a little lower than the left owing to spinal deformity. The clavicles were almost normal, with exaggerated curvature on both sides. The spinal column showed double scoliosis, very marked concavity to the right in the dorsal region and to the left in the lumbar region. In consequence of this deviation the thorax was deformed, its left side projected behind; on the right side, in the mammary region, there was a deformity as if a blow of an axe had been given; there was a sharp bend in the wall of the thorax forming a kind of dihedral angle. The right side of the chest projected in front, the right shoulder was lower than the left and there was lumbar curvature. The muscles of the lower extremities were greatly developed, the bones were thick and the epiphyses much hypertrophied. There was no abnormal curvature, but the normal curvature was much exaggerated. The feet were large and square, very large in proportion to the leg. In the upper extremities, the forearm was noticeably longer than the upper arm, the volume of the diaphyses was out of proportion to the length and the epiphyses were hypertrophied. The hand was fleshy, the fingers square at the end and pudding shaped and differed very slightly in length. The hand exhibited the peculiarities of main-en-trident and its size was disproportionate to the rest of the arm. The frontal and parietal bones projected, so that the forehead projected considerably and there was noticeable widening of the bi-parietal diameter and apparent flattening of the upper part of the skull. The nose was flattened and enlarged at its upper part. The face appeared rather large. The intelligence was fairly developed, he could read and write. The external genital organs were but little developed, they were not, however, abnormal considering the age of the child. *Measurements* (taken Nov. 1901). Total height 96 cm. Length of spinal column measured from the seventh cervical to fifth lumbar vertebra 36 cm. Circumference of thorax at level of nipples 53 cm. Length of clavicles 11 cm.; thigh from the tip of trochanter to the interarticular cleft of knee 20 cm. From the interarticular cleft of knee to external malleolus 19 cm. Circumference of base of thigh, R. 34 cm.; L. 33 cm. Length of foot from posterior part of heel to tip of great toe 17 cm.; upper arm from the large tuberosity of the humerus to the bend of the elbow (both sides) 10 cm.; forearm from bend of elbow

to upper fold of wrist (R.) 13 cm., (L.) 12 cm. Total length from acromion to middle finger 33 cm. Length of hand both sides 11·5 cm. Circumference of head 51·5 cm. Sub-occipito-bregmatic circumference 48 cm.; sub-occipito-frontal circumference 49·5 cm. Circumference corresponding to maximum diameter of Budin 56·5 cm. At the end of Dec. 1901, he had grown ·5 cm. He was treated with thyroid extract in tablets, and when seen in May, 1902, the measurements were as follows. *Measurements* (May, 1902). Height 110 cm. Length from the upper extremity of the sternum to umbilicus 29 cm. Circumference of chest at nipples 55 cm. Length of arm from the acromion to the end of the index finger 38 cm. From the antero-superior iliac spine to the lower extremity of the external malleolus 51 cm. (Bibl. No. 410, p. 543.)

Fig. 671. *Rudaux's Case.* This case was described by Rudaux after he had reported Le Lorier's Case (Pedigree 676). I. 2, aged 39, came for her seventh confinement. Her six previous children, II. 1—6, were all normal, five had been born normally, with one of them the forceps had been used. One of these six children, II. 1, had died aged 5 months of gastric intestinal trouble (*not noted on Plate*). The seventh child, II. 7, was a girl of the characteristic achondroplasic type. Her weight was 1920 grammes, length 32 cm. She died next day. Head diameters: occipito-mental 12·2 cm.; occipito-frontal 10·2 cm.; sub-occipito-bregmatic 8·5 cm.; bi-parietal 10 cm.; bi-temporal 8 cm. (Bibl. No. 524, p. 128.)

Fig. 672. *Lequeux's Case.* I. 3, was a primipara, there was no syphilis, rachitis, tuberculosis or intoxication of any kind in her family. I. 2, was rather given to absinthe and had lost a child, II. 1, by a previous marriage suddenly. The confinement was normal and II. 2 was born, weight 3500 grammes. *Measurements.* Total length 44 cm. Length of trunk 24 cm. Abdominal circumference 13 cm. Length of upper limb from acromion to the digital extremities 10·5 cm.; lower limb from the iliac spine to the heel 13 cm. Circumference of head 38 cm. Head diameters; bi-parietal 11·7 cm.; bi-temporal 11·3 cm.; occipito-mental 16 cm.; sub-occipito-frontal 11·3 cm.; sub-occipito-bregmatic 12 cm. The hand was trident-shaped, the limbs short and squat and marked by deep furrows. (Bibl. No. 472, p. 150.)

Fig. 673. *Laffargue's Case.* There is no other case among collaterals or ascendants. The individuals, II. 1—2, were brothers, of mixed race, a Berber negro race with predominance of negro. II. 1, aged about 30, had a peculiar gait, thick-set hands and feet and slightly curved limbs with concavity directed inwards. II. 2, aged about 25, hands and feet like those of his brother II. 1, short but of normal breadth. His gait was peculiar and the curvature of his lower limbs very pronounced with concavity directed inwards. They had no siblings.

	II. 1 cm.	II. 2 cm.		II. 1 cm.	II. 2 cm.
Measurements:			*Measurements:*		
Height from the ground			Breadths: span	105	116
to Vertex	128	114	,, from one acromion to the other	29	32
,, Acromion	105	88	,, from one iliac crest to the other	26	27
,, Epicondyle	78	70	Length of foot	20	20
,, Styloid process of radius	64	56	Length of thumb	5·5	9
,, Lower extremity of medius	53	41	Lumbar concavity	5	
,, Great trochanter	57	56	Skull and face. Maximum antero-posterior diameter	20	21
,, Interarticular cleft of knee	32	31	Maximum transverse diameter	14	16
,, Internal malleolus	7	7	Bizygomatic diameter	12·5	11
Length of trunk	58	52	Length of face	14·5	12
			Minimum frontal diameter		13
			Height of auditory meatus from the ground	120	104

(Bibl. No. 343, p. 515.)

Fig. 674. *P. Marie's Case.* I. 1, was well made and not alcoholic. He died aged 33 of acute meningitis. I. 2, was well made, and knew of no case of abnormal stature in her relatives, who had been remarkable for their height and longevity. Only two children are mentioned; II. 2, who was alive and well made and II. 1, aged 18. II. 1, was born at term, the birth was natural, he was breast-fed for 20 months, walked and spoke at 18 months. His head was too much developed, his palate very arched, and his hand trident-shaped. He was not intelligent and could not learn to read and write. *Measurements.* Height 107·5 cm. Length of upper extremity from the acromion to the tip of the middle finger 37 cm.; upper arm 11 cm.; forearm 15 cm.; the lower extremity from the great trochanter to the ground 43 cm.; thigh 18·5 cm.; leg 22·2 cm. Distance between the jugular notch and upper edge of pubis 41 cm. (Bibl. No. 371, p. 17.)

Fig. 675. *Herrman's Case.* I. 1 and I. 2, were Russians, and II. 11 was born in Russia. The family history was negative, no similar case had occurred in any branch of the family. I. 2, had had

nine children, of whom four, II. 1—4, died in infancy, she had no miscarriages. Four daughters were living, physically and mentally normal and in good health. II. 7 and II. 9, were married and had healthy children, III. 1 and III. 2. II. 11, aged 15, was born normally, was breast-fed for two weeks, and then artificially fed, while in care of another woman. After 10 months he was returned to his mother in poor condition. The mother noticed then that his head was large and his limbs short. He began to teethe at the age of 9 months, could sit up at 5 years, stand at 6 years, and walk at 7 years of age. He began to talk distinctly and intelligently at 7. His weight was 60 pounds. The bones of the skull were well developed, the root of the nose depressed, the chest well formed and the extremities very short. The fingers reached only to the great trochanter, they were of nearly equal length and had the characteristic trident form. The muscularity of the arm and the enlarged extremities of its bones gave it a peculiar knotted appearance. The lower extremities were short and muscular, the femur being shorter than the tibia. The marked curvature of the legs was entirely lateral. The genitals were well developed and pubic hair abundant. The intelligence was retarded. *Measurements.* Height 117 cm. Occipito-frontal circumference of head 54·5 cm. Length of upper extremity from the acromion process to the tip of the middle finger 45 cm.; upper arm 15·5 cm.; forearm 17 cm.; hand 14·5 cm.; the lower extremity from the antero-superior spine to the sole 53 cm.; femur 22 cm.; tibia 25 cm. Circumference of chest 69 cm.; neck 29 cm.; abdomen 58 cm. Distance from the vertex to the umbilicus 60 cm. (Bibl. No. 449, p. 18.)

Fig. 676. *Le Lorier's Case.* I. 1, died aged 56 of phthisis. I. 2, was healthy. II. 6, was well proportioned, she had five brothers and sisters in good health, II. 1—5, and had lost none. At 8 years of age she had measles. She married II. 7, a healthy cab-driver. There was no case of achondroplasia in the family. Her first child, III. 1, was alive. He was born in 1901 and the forceps had been used at his birth. The second, II. 2, was born normally in 1903 and died of broncho-pneumonia aged 22 months. The third, III. 3, a girl, was achondroplasic and weighed 3250 grammes. (Bibl. No. 524, p. 127.)

Fig. 677. *Moir's Case.* II. 2, aged 58, was a Chinaman. His parents, I. 1 and I. 2, natives of Hankow, were of normal build, but were dead. He was married and had a son and a daughter, III. 1—2, but they were not seen. He was intelligent, bright and alert. His head was large and globular, the bridge of the nose depressed, the palate normal. The lower limbs were short but remarkably well developed muscularly. He was somewhat flat-footed, but the legs were well formed; the ribs were normal. The hanging arms reached only to the crest of the ilium, the radius was not enlarged at the lower end (see our Plate R (11)—(13)). *Measurements.* Total height $42\frac{3}{4}''$. Height of umbilicus from the ground 19″. Circumference of head $22\frac{1}{4}''$. Length of clavicle $2\frac{3}{4}''$. Distance from tip of mastoid to tip of mastoid 7″ (how measured?). Length of radius $2\frac{3}{4}''$. Distance from olecranon to ulnar styloid process 5″; antero-superior iliac spine to internal condyle $9\frac{1}{2}''$; internal condyle to internal malleolus 8″. Chest, expanded, 27″. (Bibl. No. 605, p. 516.)

Fig. 678. *Charon, Degouy and Tissot's Case.* I. 1, was given to drink, I. 2, was insane, II. 1 and II. 2, were well preserved septuagenarians, they had 12 children, of whom four, III. 2, died young, of trivial diseases. Another, III. 1, lived only two days and the last, III. 5, was stillborn. One of these six had congenital club-foot, but the account does not state which. Five others, III. 3, showed no peculiarity. III. 4, aged 41, was the eighth child and was imprisoned for murder. He had no education and was mentally deficient. He suffered from double asymmetrical micromelia of the lower extremities (micromélie abdominale double et asymétrique). His height was 134 cm. The R. leg was shorter than the L. Length of R. leg 41 cm., of L. 68 cm., measured from the antero-superior iliac spines, as apparently the great trochanter was absent on one side. There were various anomalies in the bones of the legs. The feet were broad and short, the toes separated from one another, "sont élargis en battant de cloche, le premier en retrait sur les autres, le cinquième presque aussi gros et sur le même plan transversal que le premier." The trunk was well formed but he had dorso-lumbar scoliosis. The upper limbs were in harmony with the trunk, the hand was not trident shaped. The penis was normal. He had monorchism. His measurements were: Height of trunk 50 cm. Distance from the pubis to vertex 76 cm.; from the pubis to the ground 58 cm.; from the jugular notch to the xiphoid appendix 27 cm.; from the jugular notch to the umbilicus 36 cm. Circumference of the thorax at nipples 87 cm.; of the abdomen at the umbilicus 75 cm. Maximum circumference of head 56 cm. Antero-posterior maximum diameter of head 18 cm. Maximum transverse diameter of head 15·5 cm. Cephalic index 86. Total length of upper limb 69 cm., humerus 29 cm., radius 24 cm., medius 10 cm.; R. lower limb (from antero-superior iliac spine) 41 cm., femur 20 cm., tibia 16 cm.; L. lower limb 68 cm., femur 29 cm., tibia 34 cm. (Bibl. No. 563, p. 390.)

Fig. 679. *Sinnetamby's Case.* This is a case from Ceylon. I. 1 and I. 2, were of average size and build, they had several children, all of whom were of normal size except II. 2, by name Podi Nona. She was a woman of diminutive size, aged 20; the upper half of her body, though small, was well proportioned, but the lower limbs were short, out of proportion to the rest of the body and afflicted with marked genu valgum. The pelvis though well formed was not calculated to permit the passage of the foetus—so Caesarian section was performed. The operation was successful, for II. 2 lived, but no statement is made as to whether the child lived. *Measurements* of II. 2. Height standing 52″; when sitting 29″. Distance from crown of head to umbilicus 23″; from umbilicus to the sole of the foot 29″; from

jugular notch to symphysis pubis 19″; from occipital protuberance to tip of coccyx 26″. Circumference of head 21″. Inter-mastoid measurement across vertex 15½″. Clavicle 5″. Humerus (acromion to external condyle) 9″. Radius 7½″. Ulna 8¼″. Wrist joint to tip of middle finger 6″. Circumference of hand 7½″. Length of thumb 2¾″; of index finger 3¼″; of ring finger 3½″; of little finger 2⅜″. Circumference round crest of pelvis 25″. Interspinous 9″[1]. Intercristal 9½″. External conjugate 6¼″. Length from antero-superior spine to knee 14″; antero-superior spine to internal malleolus 24″; great trochanter to sole of foot 24¾″. Length of tibia 10″; of fibula 11¼″; of foot 8½″; of thorax 28″; of vertebral column from occiput to coccyx 29½″. (Bibl. No. 504, p. 72.)

Fig. 680. *Bramwell's Case.* No statement is made with regard to I. 1 and I. 2, except that they had 14 children, II. 1—2, of whom one died in infancy. II. 2, married II. 3, and had two children, III. 1—2. III. 1, aged 3 1/12 years, and whose height was 29½″, and weight 24½ lbs. She was brought to hospital for laryngeal diphtheria necessitating tracheotomy. There was a marked contrast between the length of the body and the limbs. The tip of the middle finger reached half-way between iliac crest and great trochanter. The head was very large, the forehead prominent, the bridge of the nose depressed, the space between the eyes normal. There was partial epicanthus. The chest was small and flat, the infra-sternal angle very large (120°), the abdomen large. When the hands were held loosely by the side or laid flat on the table the middle and ring fingers separated (trident shape). The hands were short and broad, the fingers short, broad near the base narrowing towards the tips (carrot-shaped). The general health was good. The mother said the child was backward compared with another child of hers, III. 2, who was two years younger. III. 1, was good-tempered and had a good memory for names and objects, apart from this, the mental power was not as well developed as in a normal child of that age. She was unable to speak properly. *Measurements.* Head: circumference 52 cm.; occipital protuberance to root of nose 35 cm.; auditory meatus to auditory meatus (over vertex) 36 cm. Trunk: chest circumference (through nipples) 45·5 cm.; circumference at abdomen (umbilicus) 52·5 cm.; occipital protuberance to tip of sacrum 36·5 cm. Upper limb: humerus 8 cm.; forearm 11 cm. Lower limb: external trochanter to external malleolus 26 cm. The arms could be raised to the side of the head but the elbows could not be fully extended. (Bibl. No. 483, p. 174.)

Fig. 681. *Cranke's Case.* I. 1 and I. 2, were Dutch Afrikanders of Griquatown, both normal. The account states they had five normal children older than II. 6, but does not say if they had younger children. II. 6, a boy aged 6, height 34″, was sturdy and intelligent. The limbs were short at birth, the cranium was dome-shaped, and he had well-marked main-en-trident. (Bibl. No. 531, p. 11.)

Fig. 682. *Franchini and Zamasi's Case.* II. 13, aged 35, was born at Cologne and a juggler by profession. His father, I. 1, was dead, cause unknown; he was a well-built man of strong constitution. I. 2, the mother, was alive and normal. She had had 13 children, of whom 12, II. 1—12, were dead. Two of these, II. 1—2, had died at birth, II. 1, of asphyxia, II. 2, of jaundice. The others died young of intercurrent diseases, all were normal. II. 13, born at term, was the 13th child. There was nothing remarkable in the ancestors or collaterals. II. 13, aged 35, was breast-fed, his weight was less than is usual at birth and he remained small and of frail constitution though he never had any serious illness. At the age of 15 his head began to increase in size, at 20 he had blennorrhagia and at 23 married II. 14. His height was 123 cm., he had a large head and short extremities and was a typical case of achondroplasia. His intelligence was above the average, he could read and write well. He had a malformation of the elbow (position antero-postérieure en extension complète avec un certain degré de rotation). The distal extremities of the metacarpals were much enlarged and the articular extremities of the ulna and radius much deformed. The latter descended lower than is usual and consequently the space corresponding to the fibro-cartilage was much diminished. The styloid apophysis of the ulna was much lengthened and developed and almost as thick at the point as at the base, that of the radius was not well defined. The epiphyses of the femur, tibia and fibula were also deformed. *Measurements.* Weight 50 kilos. Height 123 cm. Head measurements: maximum frontal circumference 61·5 cm. (?); fronto-occipital circumference 50·5 cm.; antero-posterior diameter 20 cm.; transverse diameter 16·5 cm. Cephalic index 82·5. Bi-orbital diameter 12·5 cm. Bi-malar diameter 12·3 cm. Circumference of thorax at nipples 81 cm.; abdomen at umbilicus 73 cm. Distance from jugular notch to pubis 49 cm.; pubis to ground 44 cm. Upper limbs: acromion to epicondyle, R. 19 cm., L. 19 cm.; epicondyle to styloid apophysis of radius, R. 20 cm., L. 19 cm.; length of hand from interarticular line to tip of medius, R. 14·5 cm., L. 14 cm. Lower limbs: from antero-superior iliac spine to ground 55 cm.; from great trochanter to external malleolus 48 cm.; from great trochanter to external condyle of femur 24 cm.; from external condyle of femur to external malleolus 24 cm. Length of right foot from hinder edge of heel to point of great toe 19 cm.

His wife, II. 14, aged 33, from the Canton of St Gall, was 105 cm. in height with large head, flattened nose, trunk of normal length, very short limbs and trident-shaped hands. At the age of 20, she was 1 metre in height. She was very intelligent but refused to be examined. Her parents, I. 3 and I. 4, were alive and normal. II. 13 and II. 14, had one child, a girl, III. 1, born 1½ years after marriage delivered by Caesarian section and now aged 12. Her height was 80 cm. (70 cm. is given in another

[1] We have interchanged author's interspinous and intercristal measurements.

place). She was breast-fed by mother, had large head, short limbs, was very intelligent and exactly like her parents. (Bibl. No. 644, p. 244.)

Fig. 683. *Schmidt's Case II.* (Jacob Hoeppner.) I. 1, I. 2, I. 3, and I. 4, were strong and of average height, as were also II. 2 and II. 3. II. 2 and II. 3, had eleven children, of these two III. 1—2 were dead (it does not say whether these two were normal or abnormal), eight, III. 8, were alive and of normal size. III. 5, aged about 65, was a dwarf and measured 126·2 cm. He was born with moustache and whiskers and had been a big strong child; at 6 years of age he had small-pox and measles, otherwise had been always healthy in early life. He thought that he had grown like other children for ten or twelve years and since then had grown no more. He maintained that the stoppage in his growth was due to too great physical exertion when a boy. From his 9th year he had carried heavy blocks of wood in a sawing mill and then he was a strong robust boy and took pleasure in carrying the largest blocks possible to beat the other boys. He had always remained healthy, with the exception of an attack of severe articular rheumatism about 10 years ago. Since then he had been sickly. At the age of 35 he married III. 6, then aged 27 and as small as himself; she had been delicate and suffered from disease of the chest (Brustkrank). They had one son, IV. 2, born a month too soon, who died in five days. He was not abnormally small for a premature birth. III. 5, had besides an illegitimate son, IV. 1, by a strong healthy girl, III. 4, and this boy was as big as other children. Given to strangers to take care of, he had been neglected and died in 20 weeks. Three years ago, III. 5 had been castrated; he said it was on account of a fall and that he had never had venereal disease. Since then he had become weaker. He was much troubled with "catarrh" of the respiratory organs, and suffered from a constant cough and want of breath. His mental faculties were normal. His dwarfishness was chiefly due to his short legs, his head was not stunted in growth, the trunk was more so. His whole appearance was agreeable, and the lack of proportion was not so noticeable, since the limbs considered alone had all parts in perfect proportion. Probably a case of achrondroplasia. *Measurements.* Total length of body 126·2 cm. Length of head measured from the glabella to external occipital protuberance 184 mm.; from glabella to the most prominent point of the occiput, parallel with the German horizontal plane, 192 mm.; measured, without paying attention to the horizontal plane, from a point in the middle of a line joining the tubera frontalia to the external occipital protuberance 182 mm. Breadth of head 159 mm. Perpendicular length of spinal column 64·5 cm. Length of sternum 17·6 cm. Circumference of chest measured through nipples, average with quiet breathing 72·5 cm.; at abdomen 61·2 cm.; hips at crests 64·1 cm.; at trochanters 69·8 cm. Length of clavicle 15·0 cm.; humerus 24·2 cm.; ulna 20·0 cm.; radius 17·1 cm.; hand not given. Circumference of middle of upper arm 19·9 cm. Maximum circumference of forearm 20·3 cm. Length of femur 24·0 cm.; tibia 28·2 cm.; foot 20·5 cm.; lower limb from trochanter to external malleolus 51·5 cm. Circumference of middle of thigh 35·2 cm.; of calf 267 mm. (Bibl. No. 270, p. 63 and pp. 69—74.)

Fig. 684. *Schmidt's Case I.* (Sophie Petersen.) I. 1, I. 2, I. 3, and I. 4, were of average height. II. 2, was a healthy man of ordinary size. II. 3, was also of average size. She had scarlet fever and measles in youth, later she had violent puerperal fever (immature five months birth), in addition she had two difficult confinements, but the children were healthy. She had 12 pregnancies, III. 1—12. III. 1, was a healthy child at first, though the confinement was difficult, but in her 10th year she got spinal disease (Wirbelcaries) of which she died aged 15½. III. 2, a girl born after an easy confinement, died in nine days of "Lungenschlag." III. 3, miscarriage at 3½ months. III. 4, a son born after an easy confinement, was a weakly child; at the age of 1½ he was attacked by "paralysis of the spine with convulsive attacks," but got better in three years. Later he had scarlet fever and articular rheumatism, but at age of 22 was fairly healthy. III. 5, aged 18, was born after an easy confinement and was chlorotic. III. 6, a boy, an immature birth at 5½ months. III. 7, born after an easy confinement, died aged 18 months from "teething convulsions." III. 8, a girl born after an easy confinement, died aged 10 months of whooping cough and affection of the brain. III. 9, miscarriage at 3 months. III. 10, immature birth at 5 months. III. 12 was a miscarriage at 4 months. III. 11, aged 11½, was according to her mother born after a most difficult confinement. Immediately after birth the shortness of her upper arms and the prominence of the parietal protuberances were noticed, otherwise she was like other children. In her ninth month the forehead began to expand and the head was remarkably large; and at the same time a rickety rosary and posterior curvature (Ausbiegung nach hinten) of the spinal column when sitting appeared, but the latter symptom disappeared at the end of the second year. After the first year the epiphyses became enlarged. She had her first teeth at 5 months old. She spoke soon, but walked late and with great difficulty, and could not walk alone till she was 2½ years old. She had always been delicate and weakly, had measles at 3, slight whooping cough at 5 and severe abdominal typhus at 9. She scarcely grew at all in her first year the head excepted, but the cranium had not altered since her second year, she then grew slowly till her third year and stopped till her sixth year, since then she had grown half a foot (einen halben Fuss). Mentally she was perfectly normal, even above the average for her sphere of life. Her cranium was remarkably large, with the tubera frontalia and parietalia very prominent. It was evident she was rickety and macrocephalic. The shortness of the extremities, especially the upper extremities and in particular the humerus, was remarkable. The arms hung down like short fins. The epiphyses of all the

long bones were much enlarged. The muscular system was well developed, the fatty portions of the thighs and buttocks being greatly developed. The elbow joints on both sides could not be perfectly extended. The abdomen was much enlarged by meteorism. In spite of the smallness of her extremities, the girl used them cleverly, ran up and down stairs with great swiftness, and her little fingers moved quickly when working. *Measurements.* Total length of body 97·9 cm. Length of head from glabella to external occipital protuberance 165 mm.; from glabella to the most prominent point of the occiput, parallel with the German horizontal plane, 178 mm.; measured without paying attention to the horizontal plane, from a point in the middle of the line joining the tubera frontalia to the external occipital protuberance 184 mm. Breadth of head 172 mm. Perpendicular length of spinal column 46·5 cm. Length of sternum 9·5 cm. Circumference of chest measured over the nipples, average of quiet breathing 53·5 cm.; abdomen at umbilicus 56·7 cm.; hips at crests 52·7 cm.; at trochanters 65·6 cm. Length of clavicle 11·1 cm.; humerus 11·7 cm.; ulna 11·9 cm.; radius 10·2 cm.; hand from end of radius to end of middle finger 11·9 cm.; hand from end of radius to beginning of first phalanx of middle finger 5·1 cm.; whole upper limb from acromion to end of the middle finger 31·1 cm. Circumference of middle of upper arm 17·9 cm. Maximum circumference of forearm 18·7 cm. Length of femur 19·7 cm.; tibia 17·6 cm.; foot 13·9 cm.; lower limb from trochanter to external malleolus 36·4 cm. Circumference of middle of thigh 39·5 cm.; calf 26·7 cm. (Bibl. No. 270, p. 62 and pp. 69—74.)

Fig. 685. *Porak's Case I.* No statement is made with regard to I. 1 and I. 2, but I. 2 must have been normal as Porak saw her. She had had seven children, II. 1—7, the first at the age of 19, all the pregnancies had terminated at term except the fourth, II. 4, who was born at eight months, this child was alive and so were all the others except two. I. 2, was aged 30 when II. 7 was born. She came to the Hospital for her confinement, and the child, a female, who weighed 1900 gms., was born dead and achondroplasic. The shortness and curvature of the limbs and the exaggerated development of the head and the part of the body above the umbilicus were very noticeable. The nose was flattened, the neck very large. The skin on the upper limbs was thicker than usual. The thighs were abducted and considerably curved, the legs were flexed on the thighs with considerable external curvature. The feet were in pronounced equino-varus position, which was however more apparent than real. The scapulae showed some abnormalities. The humeri, ulnae and radii were curved. The femora had cousiderable external curvature. The fibulae were only slightly ossified and the epiphyses of the long bones of the lower limbs were not ossified. *Measurements.* Head, occipito-frontal diameter 10 cm.; mento-occipital diameter 11 cm.; bi-parietal diameter 8 cm.; bi-temporal diameter 7 cm.; sub-occipito-bregmatic diameter 8 cm.; length of body 30 cm.; from vertex of head to umbilicus 20 cm.; umbilicus to soles of feet 16 cm.; upper limbs 13 cm.; clavicle 3·3 cm.; humerus 4·1 cm.; ulna 3·8 cm.; radius, 3·0 cm.; fibula, 1·5 cm.; tibia, internal face 2·7 cm.; tibia, external face 3·7 cm. (Bibl. No. 247, p. 560.)

Fig. 686. *Eckstein's Case I.* I. 1, had diabetes. I. 2, was healthy. They had 12 children, II. 1—4; nine children, II. 1, were healthy. Two sons, II. 2—3, had died, one aged 17 and the other 27, both had from birth symptoms of the same disease as II. 4. II. 4, aged 9, was 82 cm. in height. Her face was well formed, the nose slightly depressed, and the teeth peculiar. The incisors showed no edges (Kanten), but were pointed like the canines. Moderate bi-lateral keratitis existed, yet vision was almost normal. The thorax was much depressed; she had kyphosis and a prominent abdomen. Her extremities appeared shorter than normal, but the impression disappeared when one looked at the shortening of the trunk in consequence of spinal weakness. It was only when the body was straightened that the shortness of the extremities was noticeable. The flexibility of the wrists was very noticeable. She had genu valgum. (Bibl. No. 608, p. 1072.)

Fig. 687. *Spicer's Case.* II. 5, was sent to Spicer on account of mouth-breathing and panting of an exaggerated type. Its height was 35″. No measurements are given, but there was distinct shortening of the lower limbs with normal development of the trunk and main-en-trident. Many of the family had post-nasal adenoid hyperplasia. The eldest sister, II. 1, had a high vaulted palate, superior protrusion and had lost the upper front incisors at age of 21. The elder brother, II. 2, aged 20, height 6′ 2½″, had hands and feet of the acromegalic type, with vaulted palate and superior protrusion. He had lost the upper front incisors. The second sister, II. 3, had the thyroid gland enlarged. The second brother, II. 4, had tonsils and adenoids removed for obstruction and mouth-breathing. I. 2, the mother, and I. 1, the maternal aunt, had distinct acromegalic characters of nose, cheek-bones, lower jaw and lower lip. In short the morbid states of this family were chiefly those associated with pathological states of the bony cranial basis or the immediately overlying pituitary body or the subjacent Luschka's tonsil. (Bibl. No. 551, p. 57.)

Fig. 688. *Taylor's Case.* No statement is made with regard to I. 1 and I. 2. II. 2, was the seventh child, II. 1 being normal. It had a large head and short limbs. The trunk was comparatively normal, but there was a disproportion between the upper and forearm, the upper arm being very short. The hands were short and broad like those described by Marie, and it was enormously heavy compared to ordinary children of the same age. (Bibl. No. 434, p. 162.)

SECTION II. ATELEIOSIS.

PLATE LIV. Fig. 689. *Schmolck's Case.* Schmolck writes :—"During my Alpine tour this year, I came by chance upon a narrow very lonely valley, little frequented by tourists, named Samnauntal, one of the lower valleys of the Inntal. A short stretch northwards from Finstermünz, we came upon a somewhat rough way, impassable for vehicles, which branched off westwards from the Inntal. The eastern half of the valley belongs to the Tyrol, the western to Switzerland. The valley has six 'Ortschafts' which lie at a level of from 1500 to 1800 metres, and have 356 inhabitants altogether. Among them I found seven dwarfs, whom I photographed. A thorough physical examination, without clothes, could not be made. But in any case such examination could have been of no value, since no Röntgen photograph could be made and the pictures of the bones are of the first importance. I can therefore only give a quite superficial description of individual dwarfs, but I believe that these as well as a description of their origin will be of interest. I have made very careful inquiries concerning the latter. Until 1873 there had never been such cases among the inhabitants of the valley and no cretinism or goitre. These dwarfs were all descended from a brother and sister who lived in the valley at the beginning of last century and were mentally and physically perfectly normal, Christian Prinz and Marie Prinz. Marie Prinz, I. 1, married Nikolaus Jenal, I. 2, and had six normal children. Only the descendants of II. 3, a normal daughter, are given. She married Vincenz Messner, II. 4, and had three normal daughters. Of these, III. 2, Therese, married III. 1, normal, and had 10 normal children, IV. 1—10. III. 4, Aloisia, married III. 3, Josef Jenal, and had six normal children, IV. 11—16, and two dwarfs, IV. 17—18. III. 6, Jakoba, married III. 5, Florian Kleinstein, normal, and had five normal children, IV. 19—23, and three dwarfs, IV. 24—26. Christian Prinz, I. 3, married Marie Jenal, of Samnauntal, I. 4, and had six normal children. Of these, II. 8 married Pauline Willner, of Samnauntal, II. 9, and had four normal children, III. 7—10, several, III. 11, with obvious dwarf growth who died young, and III. 12, a dwarf still living. II. 14, Eduard Prinz, married II. 15, Josefa Jenal, of Samnauntal, and had two normal children, III. 13—14, and two dwarfs, III. 15—16. IV. 17, Suzanne Jenal, aged 30, height 108 cm. (see Plate BB (46), when aged 28), lives in the village of Raweisth, where she acts as housekeeper to her father who is village innkeeper. Her demeanour is somewhat foolish, but more marked defects of intelligence are not to be observed. The cranium is very large, the bridge of the nose is broad and depressed, the eyes are wide apart, the skin of the face is wrinkled, the neck very short. There is no goitre. The limbs are well proportioned and their movements adroit. IV. 18, whose sex is not stated, was born 30 years ago and died, aged 29. IV. 24, Julius Kleinstein, aged 30, height in shoes, 108 cm., has a large quadrate skull, somewhat bulging forehead, depressed bridge of the nose, slight growth of hair on upper lip, short neck and skin of face wrinkled. He has no goitre and a deep and somewhat peculiar voice. His limbs are well proportioned, and his movements animated and adroit. He is of average intelligence and friendly disposition. He is an independent tailor in the village of Plan and lives with his mother and two dwarf sisters. IV. 25, Marie Kleinstein, aged 26, height 93 cm. She has a very large angular skull, bulging forehead, depressed bridge of nose and a very short neck with wrinkled skin. She has no goitre. Her movements are very quick and adroit. She had done well at school and no defect of intelligence is to be observed. She occupies herself with needlework, as she is too weak for work in the village inn which her mother carries on. IV. 26, Julie Kleinstein, aged 14, height 86 cm., has exactly the same appearance as IV. 25, but the cranium is relatively even larger than in the latter. She is still at school and doing very well indeed (Plate BB (47) is a good photograph of the three Kleinsteins). III. 12, Josefa Prinz, in Compstock, aged 26, height 109 cm. in her shoes, has well-formed limbs. Her movements are quick, animated and accurate. She is of a friendly disposition and has a pleasant expression. The shape of her head is not in the least peculiar. The thyroid is not enlarged. She sings well but her voice is child-like. Her intelligence is obviously quite normal. She follows the occupation of a dressmaker, by which she supports herself and her mother and is considered a very capable "fashionable" tailoress. She has her clientele in Munich, Innsbruck, Bozen and St Moritz. By her side in the portrait is her mother, aged 70 (see Plate BB (44)). III. 15, Rudolf Prinz, aged 24, height 104 cm. in his shoes, without shoes scarcely 100 cm., has a quadrate shaped head. The parietal eminences are very prominent, the transverse measurements between the parietal eminences very great. The bridge of the nose is depressed. He has a small moustache, and a childlike, rather squeaky voice. The skin of the face is wrinkled. He has no goitre, his intelligence is not defective, his movements are animated and accurate. III. 16, Ulrich Prinz, aged 22, is of exactly the same height and similar personality, but has no trace of a moustache. He has learned tailoring but does not follow it as a trade, as there are already tailors enough in the district. Both these dwarfs live in the village of Laret, in the house of their well-to-do father. Their eldest brother stands besides them in the picture. (See Plate BB (45).) As to the nature of these instances of dwarf growth, one can naturally say nothing definite without accurate X-ray investigations. But if one considers the complete symmetry and proportion in build present in all these dwarfs, the absence of all curvature and shortening of bones, the lack of defect of intelligence

and so forth, one may well believe that they belong to the so-called 'real dwarfs' (echte Zwerge). As is well known, disturbance of bone growth may arise from defective function of the thyroid gland. How that is also concerned here cannot be said for certain, since abnormalities in the condition of the thyroid gland in these dwarfs were not observed on external examination. An anomaly of the thyroid gland, which is so common in high lying and secluded mountain valleys and leads to cretinism and goitre, is not thereby excluded here: but it must have remained confined to a single family, since there is neither cretinism nor goitre amongst the remaining inhabitants of the valley. All these are well formed people, who by agriculture and grazing live in moderate prosperity under very favourable hygienic conditions. In considering the appearance of dwarf growth in the Prinz family, one must take into account, besides geographical position, the many marriages which have been contracted between blood relations living in the same valley. In the Prinz family the surnames recur continually. Although the Catholic male inhabitants of the valley fairly often marry Tyrolese women from the neighbouring Inntal, marriages with the inhabitants of the neighbouring Unter-Engadin do not occur, on account of the Protestant faith of the latter, and as a result marriages between the few inhabitants of the Samnauntal are contracted. It is worthy of note that the Samnauntalers themselves now strictly shun any marriage alliance with the normal members of the dwarf family. They even express fear, lest their whole community should degenerate into dwarfs. It should be considered further that the cretinoid type of face, very obviously recognisable in many illustrations of dwarfs of normal intelligence, is not uncommon (see *Virchow's Archiv*, Bd. 94), and that this is to be referred to premature ossification and union of the basilar synchondrosis. The bones of the face remain small in consequence; the cranium, in contrast to it, appears to be enlarged, and it has, in compensation, really grown to excess. In the descendants of Marie Prinz the cretinoid formation of the face is very obvious, in those of Christian Prinz only slightly so, not being present at all in the case of Josefa Prinz. I have also seen misplaced teeth and persistent milk teeth (as seems to be common in cretins) in single individuals of these dwarfs. On the other hand I have, even after prolonged inquiry, found no noticeable defect of the intelligence in any of these dwarfs, at the most single individuals appeared somewhat childish and too little independent in disposition[1]." (Bibl. No. 538, p. 105. The photographs reproduced were most kindly sent to K. Pearson by Dr Schmolck.)

Fig. 690. *Rischbieth's Case* (Magri Family). The account of this family is given by III. 11, Ernesto Magri, known as "Baron" Magri, now aged 62 years. I. 1, his paternal grandmother, was "a little woman," of stature about 4′ 6″, but she showed none of the peculiarities seen in his dwarf brother and sister. II. 1 and 2, his parents, were of ordinary height. He knows nothing of their brethren, nor of his maternal grandparents. Of his own brethren, III. 1, 2, 3, 4, 5, 6, 7, 8 and 10 were of ordinary stature. III. 1, died aged 85 years. Of the others some are still alive and others are dead. He is uncertain of his facts about them and knows nothing about their descendants if any. The other two of his brethren, III. 9 and 13, were dwarfs like himself. III. 9, died aged 32 years. His brother III. 13 is still living. He himself married a woman of ordinary size. By her he had three children, two sons and one daughter, IV. 1, 2 and 3. Of his sons' descendants, if any, he knows nothing. But his daughter, IV. 3, married a man of ordinary size and has had two children of ordinary size and growth, but died during her second confinement. III. 13, Primo Magri, is now aged 60 years. Married to III. 14 (*née* Lavinia Warren or "Mrs Tom Thumb"), late in life, after the menopause, a female dwarf of his own height, now aged 67 years. She had a sister, III. 16, of her own size and type. Nothing is known of their parents or collaterals. III. 14, was formerly married to III. 15, Charles Stratton, "Tom Thumb"[2] (see Plate AA (43)). It is said that there was a child of ordinary size and growth, IV. 5, born of this union, but that it died in infancy. Of the parents or collateral relations or brethren of III. 15, nothing is known. But he himself was of markedly infantile appearance. He had a double row of teeth all round, the milk dentition having persisted, as well as the permanent set. In general he resembled Schaaffhausen's dwarf (see Bibl. 130 and our p. 388). He died aged 53 years. III. 11, Ernesto Magri, aged 62 years, music hall artist (see Plate CC (48)). Height 3′ 9″. Fairly strong moustache. Appearance infantile. Frontal and parietal bosses prominent. Bridge of nose slightly depressed, but not broadened or flattened; nose not tip-tilted. Skin normal. Hair of scalp thick and soft and beginning to grow grey (contrast Schaaffhausen's dwarf). All teeth present, normal. No persistence of milk dentition. Voice squeaky, childlike. The limbs show no curvatures and no shortening. Proportionally to trunk and head the length seems to be that of an ordinary adult, and the segments show this relationship to one another. The hands are not spatulate, but they are like those of an infant grown old, being broad and thick with relatively broad, thick and short fingers, as in the infant. The nails are set somewhat at an angle to the line of the fingers instead of in their plane. Nothing like a main-en-trident. Colour of face somewhat "waxy" and yellowish; looks much like that of pernicious

[1] The pedigree seems to indicate that true dwarfism might be recessive in a stock ancestral to both Prinz and Jenal families. A good piece of work might be done by a summer spent in the Samnaunthal looking up the full pedigree in the church registers. EDITOR.]

[2] The account of "Tom Thumb" is taken from Hastings Gilford, see Bibl. No. 403.

anaemia. (This feature is, however, much more obvious in his brother and his sister-in-law, III. 13 and 14, who have the colour of wax models in a show.) He is a very intelligent man. He writes a clear hand showing considerable character and though a foreigner writes English idiomatically and speaks it almost without an accent. His manner is that of an adult. He seems of somewhat timid disposition. III. 13, Primo Magri, known as "Count" Magri, aged 60 years, music hall artist. Resembles his brother in height and other particulars almost exactly but has no hair about the face, and the infantile appearance is more marked. *Note.* III. 11, 13 and 14 are, as nearly as possible, of the same height and proportions (3′ 9″). The "wax-like" complexion is more marked in 13 and 14 than in 11. In all three the teeth are still present and normal. None of these three individuals have any resemblance to cretinism, myxoedema, achondroplasia, rickets, or other of the more usual varieties of dwarf growth. No family history of syphilis, alcoholism, tuberculosis, in the Magri family could be obtained. None of these have taken thyroid extract. Unfortunately no physical examination, measurements or radiograms could be made of these dwarfs and though they look "anaemic" no blood examination could be made. An account of Mrs Tom Thumb is given by Gould and Pyle (see Bibl. No. 332) and by Hastings Gilford (see Bibl. No. 403). We owe a new photograph of Baron Magri taken especially for this work to his kindness: see Plate CC (48) and also Plate AA (43). A photograph of "Le comte, la comtesse, le Baron M..." is given in *Le Bulletin médical*, 23e Année, p. 961.

[The earliest accounts of the Magri family are due to Veratti Verardini and Taruffi and as the two latter give an earlier and in some respects a fuller account of the family, abstracts are included here. Taruffi says that "The Magri family" were natives of the province of Ferrara. I. 1 and I. 2, were robust and of a good height. They had thirteen children (Rischbieth gives only twelve) of whom eight survived. Of these eight, three were dwarfs, III. 9, III. 11 and III. 13, and five were normal. III. 9, the seventh child, was a perfectly symmetrical dwarf. At the age of 19 she had not menstruated, her height was 102 cm., she weighed 45 pounds (libbre) and had considerable discernment. In 1865, at the age of 27, she was exhibited before the Medical Society of Bologna and then measured 105 cm. and weighed 21 kilos. The circumference of her head was 48 cm. and the distance from the top of the head to the chin 52 cm. (dal sincipite al mento). She died aged 33 of angina diphtherica and her height was then 110 cm. Ernesto was the 12th child. He was also well formed, with the exception of his head which was rather large. He had an active mind and aggressive character. At the age of 11 in 1858 he was 89·0 cm. in height, at 18 in 1865, 104 cm. The circumference of his head was 53 cm., and the distance of the top of the head from the chin 57 cm. He weighed 19 kilos. With his arms perpendicular the wrists (carpi) reached the trochanters. He had no hair on his face and little on the pubis, the testicles were but slightly developed and frequently withdrawn into the inguinal canal. At the age of 26 he married III. 12, a girl 156 cm. in height. They had two children; the first was a son who in 1878 was aged 4, and was 95 cm. in height and well developed. The second, a daughter, born 1876, was well formed at 15 months old, and 60 cm. in height, that is 12 cm. below normal, so probably she would be a dwarf (according to Rischbieth she was the *third* child and *not* a dwarf). Ernesto in 1877 was aged 30 and was 110 cm. in height. Primo was the 13th and last child (according to Rischbieth the 12th child). At 8 years of age he had well-formed limbs and measured 82 cm. In 1865, when 15 years of age, he was 91 cm. high, the circumference of his head was 48 cm., and the distance from the top of the head to the chin 53 cm. With hanging arms his wrists reached the great trochanters. The testicles were small and partly concealed in the inguinal canal; there was no hair on the pubis. He weighed 14 kilos. He was sharp and intelligent. At the age of 28 he measured 109 cm. (Bibl. No. 248, p. 446.) Verardini also gives (Bibl. No. 133b) the report of a medical commission on the Magri family, 1865. Primo Magri aged 15, Ernesto aged 18, Amalia aged 27. All had a slight degree of abnormality in the conformation of the head, otherwise they were harmoniously proportioned, they were intelligent but had not the mental power of their age and seemed more like children.

	Amalia	Primo	Ernesto
Horizontal circumference	48 cm.	48 cm.	53 cm.
Vertex to chin	52 „	53 „	57 „
Sagittal circumference	45 „	45 „	47 „
Thoracic circumference under axillae	61 „	53 „	58 „
„ „ at nipples ...	57 „	54 „	60 „
Pelvic circumference	68 „	56 „	60 „
Bi-acromial diameter	26 „	23 „	26 „
Bi-iliac distance	25 „	24 „	23 „
Stature	105 „	91 „	104 „
Weight	21 kilos.	14 „	19 „

Amalia menstruated regularly. Ernesto had little hair on face or pubes, and Primo less. In the case of these brothers the testicles were largely in the inguinal canal. These facts were ascertained from the doctor to the family, Giuseppe Veratti. EDITOR.]

Fig. 691. *Rischbieth's Case.* Adolf Gehrler from Innsbruck, Tyrol. II. 1, was an only child. His parents and all their known relations were of ordinary size. II. 2, and her only brother II. 3, were of ordinary size, as were their parents and all known relations of these. Of the children of II. 1 and 2, there were six; of these, III. 1, 2, 3, 4 and 6, were of ordinary size. All known descendants of these are also of ordinary size. III. 5, however, is a dwarf now aged 30 years. No measurements, radiographic examination or physical examination were permitted, so that the account is so incomplete as to be almost valueless. His height[1] is approximately 3′ 6″. He has the proportions, facial expression, and general appearance of a very small man, and not that of a child, as is usual in most dwarfs of his kind. There is a fairly strong moustache. The bridge of the nose is not depressed or broadened. The nose is not tip-tilted. The frontal and parietal eminences are not markedly prominent and the cranium, when compared with the face, is not proportionally larger than in the ordinary adult. The teeth are all present and normal. The skin is not rough, nor dry, but perfectly normal. The hair of the scalp is neither thin, nor dry, nor brittle. The hands show no difference from those of an ordinary adult except smallness in size (*i.e.* they are not spatulate, show no main-en-trident or other peculiarity in the shape of the segments of the fingers; the shape and mode of insertion of the nails are in no way peculiar). Their size is in the usual proportion for an adult to the other segments of the limbs, the trunk and the head. The limbs show no curvatures. They are rather thin but otherwise fairly well formed. Their length both in the proportion of one segment to another and of the whole to the trunk and head is that of the ordinary adult. The complexion as in all dwarfs of this variety is "waxy" and yellow and would suggest, almost exactly, that of pernicious anaemia if seen in other individuals. No examination of the blood being permitted it was not possible to determine whether anaemia was present or not, but apart from his colour, which is that of disease, this individual has no symptoms and feels in good health. The voice has the timbre of an adult male, though somewhat squeaky and high pitched probably owing to the smallness of the chest and consequently defective phonation. The intelligence is good. He answers questions quickly and clearly, can read, and writes a clear well formed hand. All movements are of the average celerity and precision. He seems to be in no way lacking in independence. No information as to sexual characters could be obtained. This dwarf has not the least resemblance in any way to that dwarf growth seen in cretinism or infantile myxoedema, or that of achondroplasia. No family history of syphilis, alcohol, tuberculosis. or other known cause of dwarf growth could be obtained. No descendants. No history of collaterals obtainable.

Another German dwarf, resembling the above in height, growth of hair about the face, shape of head and of hands, colour of face and general appearance (namely that of a very small man rather than of a child as is the usual condition), is also known to me. Like the above the pedigree is negative and it has not been considered worth recording. These two individuals, however, are mentioned here because they present features pointed out above not usual in this condition known as "true dwarf growth," "echten Zwergwuchs" or "nanisme vrai." Thus of some 20 male dwarfs of this variety over 20 years of age that I have seen, only three showed any growth of hair about the face—the above two and E. Magri whose photograph is shown—(see Fig. 690). The last, however, in the shape of cranium, with its prominent frontal and parietal bosses, is more like the usual type, as also is his voice character, etc.

Fig. 692. *Jacobsen's Case.* In generations I. and II. all known individuals were of normal growth. II. 7, height 4′ 10″, is now dead (?should not II. 7 be noted as a semi-dwarf). She had three brothers and two sisters, all of average stature, all married, and all having children of average growth. The number of these is not stated. She had one sister, II. 6, of average stature, unmarried. II. 7, was aged 24 or 25 years when she married II. 8, a well developed man of 5′ 4″. There were three children of this marriage, III. 6, 7 and 8, all dwarfs. III. 6, aged $14\frac{1}{2}$ years; height 3′ 11″. Generally dwarfed. No pubic or axillary hairs. Backward in every way except mentally. Ideas rather childish but very intelligent. Passed the sixth standard at the age of $13\frac{1}{2}$ years. "Had something wrong with his left ankle, when younger, which necessitated his spending three years in a hospital." III. 7, aged 13 years. Height 3′ $8\frac{1}{2}$″. No signs of puberty and has never menstruated. Slight genu valgum. Mentally acute. Has passed the fourth standard. III. 8, aged 11 years. Height 3′ $0\frac{1}{4}$″. Very puny and delicate. Has to be kept from school in the winter months. Mentally acute. Slight genu valgum. III. 6, 7 and 8, were all born at full term and no comment was made at birth as to their size. Had not grown for two years at least before observation. When seen all had large heads and weak limbs, but the latter were in normal proportion to their height (*i.e.* there was no micromelia). II. 9, is the second wife of II. 8, and is of about the same height as was II. 7. She has two children, III. 9 and 10. III. 9, is aged 8 years. Height 3′ $8\frac{1}{2}$″, *i.e.* this is as great as that of III. 2, aged 13 years, the second child of the first wife. III. 10, is aged 4 years. Height 3′ 4″, or $3\frac{3}{4}$″ greater than that of III. 8, aged 11 years, the third child of the first wife.

[1] This had to be inferred by comparison with surrounding objects such as tables.

III. 9 and 10, are growing yearly, whereas the dwarfs have not grown for the last two years. II. 10, was the first husband of II. 9, the second wife of II. 8. By him there was one child, III. 11, aged 14 years. Height 5′ 2″, or 15″ taller than III. 6, the dwarf boy, though she is 6 months younger than he. She has axillary and pubic hairs and mammae and has menstruated regularly for 3 months. (See Bibl. No. 265.)

Fig. 693. *Boruwlaski Family.* The account of this family is taken from the Memoirs of Josef Boruwlaski, II. 4, and from the edition published in 1792. He says "I was born in the environs of Chaliez, capital of Pokucia in Russian Poland, November, 1739. My parents were of average height, they had six children, five boys and a girl, three of these children reached a height above the average, whilst I and two others remained of less stature than ordinary children of 4 to 5 years old. What seems equally remarkable is that this difference in height alternated in our births, I insist upon this point because it is singular and to correct an error in the *Encyclopedia.*" II. 1, the eldest brother, aged about 60 when Boruwlaski wrote, was nearly 3″ taller than II. 4. He always enjoyed robust health and lived with a Russian lady who found he had enough capacity to be entrusted with the management and direction of her affairs. II. 2, the second brother, was of weak and delicate constitution, he died aged 26, being then 5′ 6″ in height. II. 6, was 7 years younger than II. 4, she died aged 22 of small-pox and was then only 26″ high. Boruwlaski says that she could easily walk under his arm, and that she had a lovely face and was so beautifully proportioned that a sculptor could find nothing to criticize. The heights of II. 4 at different ages are given in English measure; he continued to grow till he was 30 years of age, and was then 3′ 3″ in height. His brother, II. 1, also continued to grow till he was 30. Boruwlaski was very intelligent, he is described in the *Encyclopédie* article "Nains," from which an extract is given in the preface of the "Memoirs" as having "a figure well-formed, head well-proportioned, fine eyes, gentle expression, knees and feet all in proportion." He lived for many years with the Countess Humieska and left her because she refused to sanction his marriage with a protegée of hers who lived with her as companion. He married this lady, II. 3, later. They travelled about a great deal, apparently living on what he got from the Royal Princes and Nobles who entertained him. He mentions the birth of his first child, III. 1, a daughter, in his memoirs, but the Preface states that at the age of 53 he had four children, the eldest aged 11. The latter part of his life was spent in England at Durham where he died, aged 98. There is a statue of him by Bonomi in the Museum of Durham University (see Plate II (67) and (68)), there are several portraits of him in existence. See Iconography, 144[a]—6[b], 163, 165. (Bibl. Nos. 43 and 47.)

Fig. 694. *Neumann's Case.* No details of the parents or collaterals of I. 1 or I. 2. I. 1, Christian Goerke, aged 54 years, "Eigenköthner," in the village of Gross Ksionsken, in the Strassburg region of West Prussia. Height 5′ 6″ (Prussian measure). I. 2, wife of I. 1; aged 58 years. Height 4′ 11″ (Prussian measure). "Thus this married pair show nothing remarkable in their stature, which is neither very great nor very small." They have had six children, II. 1 to II. 6. II. 1, died aged 1 year. It was of ordinary proportions for age. II. 2, aged 30 years. Of average height for a woman, somewhat over 5′ (Prussian measure). II. 3, Christine, aged 26 years, height 3′ 6″. II. 4, aged 24 years, height 3′ 8″. II. 5, Johann, aged 21 years, height 3′ 10″. II. 6, Jakob, aged 18 years, height 3′ 4″. "Thus these four individuals have the height of children of the age of 5 or 6 years, and such an age their bodily proportions suggest. Their heads are in proportion to the rest of the body. The faces are those of children, and indeed of very well-formed and for the most part charming children. Neither of the males show any growth of hair about the face. The breasts of the females are undeveloped. Their external genitals are infantile, as are those of the males, and show no growth of hair. No menstruation or other sex characteristics have been observed by the parents in any of these children. Wisdom teeth lacking in all. In II. 6 the second molar teeth are also lacking. Their strength corresponds to their childlike proportions and is so feeble that they can only perform such muscular work as any child of 5 or 6 years of age would be capable of performing. Their mental qualities have, however, in no way remained undeveloped as their physical qualities have done, and are in every way much more accurately expressive of their real age. They are, for this reason, welcomed by their larger fellows of their own age, and abstain from all purely childish amusements and occupations." (See Bibl. No. 84, p. 705.)

Fig. 695. *Levi's Case II.* III. 8, Domenico Gazzano, aged 33, was born at Garescio, province of Cuneo, Piedmont. His height was 111 cm. and his weight 23 kilogs. His father, II. 2, was very strong and normally intelligent, but very small; III. 8, said II. 2 was exactly the same height as himself and had the same malformation of the nose and the last phalanges of the fingers. His mother, II. 3, and all his grandparents, I. 1—4, were normal. II. 2 and II. 3, had eight children, III. 1—8, of whom two, III. 6—7, were twins, who died aged 3, and the five other brothers, III. 1—5, were normal. II. 8, was normal at birth, and in early years developed normally. He learnt to read and write. His growth stopped between 8 and 10 years of age, but otherwise he developed normally. At 15 he was sexually adult, and had sexual intercourse at 16. Five years ago he had married

III. 9, a normal woman of medium height. She had a miscarriage, IV. 1, at five months. A second pregnancy resulted in a healthy girl, IV. 2, aged nearly 2 years, who exactly resembles her father; there was a second girl, IV. 3, aged 6 months, apparently normal, who was not seen. For some years Domenico had taken to drink, otherwise he led a regular life. He was perfectly proportioned and completely developed. His nose was similar to that of Magro Santo (see Pedigree 742), tip-tilted and with wide nostrils. He had bilateral inguinal hernia. The genital organs were those of an adult, the limbs normally proportioned, and the last phalanges of the fingers malformed. The measurements were: Height of pubis from ground 55 cm. Maximum thoracic circumference 68 cm. Maximum abdominal circumference 62·5 cm. Length of sternum 13 cm. From acromion to tip of medius 48 cm. Acromion to tip of olecranon 18·5 cm. Tip of olecranon to radial epiphysis 17 cm. Maximum length of hand 12·5 cm. Length from antero-superior iliac spine to heel 64 cm. Great trochanter to inter-articular cleft of knee 26 cm.; this point to external malleolus 28 cm. Maximum length of foot 17 cm. Maximum circumference of cranium 48 cm. Maximum bi-parietal diameter 170 mm. Bi-temporal diameter 143 mm. Bi-zygomatic diameter 120 mm. Cephalic index 85. IV. 2, Alessandrina Gazzano, aged 22 months, was born at term. She was very small at birth and only weighed 2 kilogs. She was always smaller than children of her age, was very lively and intelligent, had eight teeth, could walk alone and was well proportioned. The measurements were: maximum thoracic circumference 36·5 cm. Maximum abdominal circumference 35 cm. Length of sternum 7 cm. Height of pubis from ground 24 cm. Total length of upper limb 24 cm. (? 24·5). Length of upper arm 10·5 cm. Fore-arm 8 cm. Hand 6 cm. Total length of lower limb from antero-superior iliac spine to heel 26·5 cm. Length of thigh 13 cm.; of leg 11·5 cm.; of foot 8·5 cm. Maximum circumference of cranium 43·3 cm. Bi-parietal diameter 150 mm. Bi-temporal diameter 120 mm. Cephalic Index 80. (Bibl. No 640, p. 534.)

Fig. 696. *Rischbieth's Case.* The account of this family is given by III. 1. He is perfectly clear as to the number of his brethren, and their order of birth and of his parents and their brethren as shown. But he knows nothing about the descendants, if any, of any of the latter. He knows all the individuals shown in generation I., but nothing of their brethren, if any, or descendants of these, if any. His brother, III. 4, agreed with his statements, after discussion and suggested amendments. All the members shown of generations I. and II. are still living and are of ordinary size. III. 2, 3, 5, 6 and 7 are of ordinary size for their age, at least not small like himself and his brother. III. 1, Heinrich Glauer, aged 24 years. Height between 3′ and 3′ 6″. He had the general appearance of a child of about 4 years of age, but his face, in a vague way, looks much older. No hair about the face. Expression childish. Face yellowish, waxy looking. Suggestion of pernicious anaemia. Skin normal. Not dry nor thickened. Hair of the scalp not thin and in no way abnormal. Frontal and parietal bosses not very prominent. Bridge of nose not depressed, broadened or flattened. All teeth except third molars present, sound. Voice squeaky, infantile. The limbs show no curvatures; they are somewhat massive, but the muscles are rounded, as of a child, rather than prominent as of a muscular man. The limbs show no shortening, of segments or as a whole. The hands are not spatulate nor "en trident"; they are those, as it were, of an infant grown old; short, broad and thick, with short and thick fingers, but these differ in length from one another as in the ordinary infant or adult and show no approximation to equality as in the achondroplasic. Manner somewhat childish, but it is difficult to be sure to how great an extent this is due to surroundings (those of a "show" dwarf who is usually treated either as a child or a curiosity). Intelligence good for his age (24 years); he is particularly quick and definite in his answers to questions, and took an intelligent interest in the formation of his "Stammbaum," as he described it. He can read well and writes a well-formed hand. Active and quick in all movements. The general intelligence of this dwarf would appear to be about that of the average of small shopkeepers or innkeepers. III. 4, Bruno Glauer, brother of the last, aged 20 years. He presents all the features shown by his brother, but is smaller and looks younger. His stature is approximately 3′ and he presents the general appearance of a child of about three years of age. On a more careful examination, however, his face gives the impression of a very much greater age, which might be almost anything from 12 to 25 years. He has the same "thick-set" appearance as his brother, and the description of the latter would, with the above exceptions, also apply to him. He is quite intelligent and quick and can read and write well. But his manner is childish. Most dwarfs of this variety are markedly infantile in their facial aspect; but in the case of these two it is more accurate to say that this is childish. Neither of these two know at what period of their lives they ceased to grow, never having been told. The above account is probably of little value, for no physical examination, measurements, nor radiograms were permitted. Hence even their heights had to be roughly inferred from a comparison with surrounding objects such as tables. Again, they look anaemic, but no examination of the blood was permitted. They have no real resemblance to cretinism, infantile myxoedema, achondroplasia or other common varieties of dwarf growth, and are markedly intelligent. There is no history of syphilis, alcoholism, tuberculosis or rickets in the family. They have never taken thyroid extract and never suffer from general ill-health. Photographs of these two dwarfs in their native dress are given on Plate CC (50). (Unpublished.)

Fig. 697. *Gould and Pyle's Case.* (The Rossow brothers.) II. 1, Franz Rossow, aged 20 years,

height 21″, weight 24 lbs.; II. 2, Carl Rossow, aged 18 years, height 29″, weight less than that of his brother; they are the two eldest of 16 children, and clever gymnasts. No note of I. 1 or 2 or of any collaterals or ancestors. See Plate AA (43). (Bibl. No. 332[1].)

Fig. 698. *Virey's Case I.* I. 1, was 5 "pieds" 5 "pouces" in height. The height of I. 2 was 5 "pieds." They were Germans. Virey examined their daughter, II. 2, in 1818. She was aged 8 or 9 years and was only 18 "pouces" in height. She was active and bright with the intelligence of a child of 3 or 4 years old. She began to get her teeth at age of 2 and only began to walk and speak at 4. The mother said she had had a dwarf child before, a boy, II. 1, who did not live and was only a few inches long. (Bibl. No. 64, p. 270.)

Fig. 699. *The Gibson Family.* Richard Gibson, II. 2, the dwarf artist, is an historical character. Accounts of him are given in biographical dictionaries, and in Walpole's *Anecdotes of Painting*. Born in 1615, he is said to have been a native of Cumberland, but no statement is made with regard to his parents or brothers and sisters. Walpole however states that the artist Wm. Gibson was his nephew, so in that case he must have had a brother. He became page to a lady who discovered his talent for painting and had him instructed by Francis Clein, manager of the tapestry works at Mortlake. He perfected himself by copying Lely's paintings. He became page to Charles I and obtained considerable success as an artist. He drew Cromwell's portrait several times, and under Charles II was appointed instructor in drawing and painting to the Princesses Mary and Anne. His height was 3′ 10″. He married Anne Shepherd, II. 3, a dwarf of the same height who was in the service of Mary Duchess of Richmond. No note is given of her parents, I. 3 and I. 4, or any other relatives. From their portraits these dwarfs appear to have been true dwarfs. They had nine children, III. 1—9, five of whom lived to maturity and were of normal size. A daughter, III. 9, was a water-colour artist, and Walpole says Edward Gibson the artist was probably a son. Gibson died in 1690 in his 75th year and his wife in 1709, aged 89. They were buried in Saint Paul's, Covent Garden. (See Iconography, Nos. 66—7, 185—6, Bibl. No. 42, p. 116, and *Dict. Nat. Biog.* Vol. XXI. p. 283, London, 1890, where there are doubtful statements as to pictures.)

Fig. 700. *Trevisani's Case.* (Antonio Toselli.) I. 1 and I. 2, were both robust; they had three daughters and two sons, II. 1—5, all of normal stature and build. I. 2, was in her 40th year and in the first month of her pregnancy with II. 6 had a fall. She suffered no serious injury, but menorrhagia followed and after this endometritis. She had such pain in the lumbar region that it was impossible to leave her bed until her confinement, which, though difficult, was followed by normal delivery. II. 6, was born January 16, 1808, at Penzale, and was very small at birth. At the end of 9 months his body as a whole was well proportioned, with the exception that his head was a little too large. He was weaned at 14 months. Before this he had whooping cough. He began to walk in his first year and articulated the first words well, but this development did not continue at the same rate, and after his 4th year he grew very little. At 10 years of age attempts were made to instruct him, but he could not be taught the rudiments of learning. At puberty his intellect appeared to improve. At 20 years of age he seemed to be approaching old age. He frequented churches and had no other occupation. He had fair eyebrows, large blue eyes, a large flat nose depressed at the bridge, normal teeth and a large mouth. He had a downy chin and a white flaccid wrinkled skin covered with freckles like other members of his family. His voice was nasal. *Measurements.* Total height 103 cm. Height from soles of feet to top of shoulders 84 cm. Circumference of head around temples 51·5 cm. Arc from the root of the nose to the occipital protuberance 51·5 cm. Distance from the chin to the bridge of the nose 10 cm. Bi-temporal diameter 9·9 cm. Height of the forehead 5 cm. Length of the neck 5 cm. Circumference of the neck 36·5 cm. Width of the shoulders 24 cm.; chest at the level of the floating ribs 25 cm.; chest in the upper part 19 cm. Measurement between the two iliac crests 30 cm. Height from the sacrum to the first cervical vertebra 37 cm.; pubis to the upper extremity of the same 37 cm. Length of the upper arm 16·5 cm.; of forearm 13·5; of hand with fingers 11 cm.; of thigh 28 cm.; of leg 19 cm.; of feet 16 cm. Height of the foot from the malleolus to ground 9 cm. Width of extended arms from the middle of the left hand to the middle of the right hand 101 cm. In Merlin's balance he weighed "libbre centesi 60 ed once 6.' (Bibl. No. 86, p. 60.)

Fig. 701. *Browning's Case.* II. 2, was the second son of Louis Hopkin, I. 1, who lived near Bristol. I. 1 and I. 2, were normal and had six children, II. 1—6, of whom four were normal. II. 1, is thus described by Browning:—"This surprising but melancholy subject was a young man entering the 15th year of his age though his stature was no more than 2′ 7″ and his weight 13 pounds, labouring under all the miseries and infirmities of very old age: weak and emaciated, his eyes dim, his hearing very bad, his countenance fallen, his voice very low and hollow; a dry husky inward cough; his head hanging down before so that his chin touched his breast, consequently his shoulders were raised and his back rounded,

[1 Gould and Pyle give another case: "In the middle of the 17th century a woman brought forth four dwarfs." Nothing more is said about it and there is no reference given! It is possibly Clauder's Case, but such unverifiable statements deserve the severest censure. EDITOR.]

not unlike a hump-back. His teeth were all decayed and rotten except one front tooth below. He was so weak, he could not stand without a support. The father and mother both said that he was naturally sprightly, though weakly until 7 years old, would attempt to sing and play about, and then weighed 19 lbs. and was as tall if not taller, naturally straight, well grown and in due proportion, but from that period he gradually declined and grew weaker, losing his teeth by degrees. They said also that this lad had a sister (II. 3), about 10 years of age, in the same declining state." ? not ateleiotic. (Bibl. No. 22, p. 278.)

Fig. 702. *Virey's Case II.* This case is that of the Souvray sisters whom Virey appears to have been the first to describe, at least no earlier description has been found. The Souvray sisters, II. 1—2, were born in the Vosges district of parents of ordinary size. No note is given of any other relatives. II. 2, Anne Thérèse Souvray was aged 73 when seen by Virey in Paris; her height was 33 "pouces." Garnier gives it as 86·4 cm. She was neither scrofulous nor rachitic, but was healthy and active and danced with her sister Barbe, II. 1, who was two years older and 8 "pouces" taller. Garnier gives her height as 105·3 cm. Virey gives an engraving of Thérèse Souvray (see our Plate II (70)), Garnier has an engraving of her and her sister, neither of whom shows any sign of achondroplasia. II. 2, had formerly been the fiancée of Bébé, who was a few years older, but his death prevented the marriage. She, however, called herself Mme Bébé. See Iconography, Nos. 140—141. (Bibl. No. 64, p. 153.)

Fig. 703. *Béclard's Case.* Béclard describes a dwarf, Anna Barbara Schreyerin, II. 5. It appears more than probable that this dwarf is the one described by Dornier (Bibl. No. 60) and by Chaussier and Adelon (Bibl. No. 63) under the name of Babet Schreier. The number in the family is the same as that given by Chaussier and Adelon, but the particulars differ somewhat. Béclard's account is followed here as he apparently saw the dwarf, and it seems doubtful if Chaussier and Adelon were not quoting from a previous account. I. 1, was of average height, no statement is made with regard to I. 2. They had five children, of whom four II. 1—4, three girls and a boy, were of ordinary height. Chaussier and Adelon state the first child was rather small and only lived 5 months. II. 5, was born at term, she was 8 "pouces" long at birth and weighed $1\frac{1}{2}$ "livres." She was born October 31st, 1813 (?1810) and was aged 7, October 31st, 1817. Her teeth showed she was aged 7. Chaussier and Adelon state her growth was rapid until the age of 2 years and then gradually ceased. The bones were well-formed, the muscles firm and the senses normal except for the eyes, which were myopic; the left eye was turned in. Her measurements were:—Weight $8\frac{1}{2}$ "livres." Height $21\frac{1}{2}$ "pouces." Distance from soles of feet to the pubic eminence 9 "pouces." From the soles of feet to umbilicus $11\frac{1}{2}$ "pouces." From the vertex to the umbilicus 10 "pouces." From the vertex to the pubic eminence 9 "pouces" (?12·5). (Bibl. No. 62, p. 486.)

Fig. 704. *Drysdale and Herringham's Case.* II. 2 and 3, and their antecedents and collaterals showed no dwarfing of growth[1]. III. 1, 2 and 3, were normal in all respects and were above the average height. III. 4 and 5, were born dead. No peculiarity known. III. 6, 7 and 8, aged 13, 11 and 7 years respectively, were brought to St Bartholomew's Hospital for "bending of the legs"; in the eldest osteotomy was performed on both sides. Both legs had been quite straight in all three at birth, but began to bend at about the age of 5 or 6 years. Respective heights: 44″, $38\frac{1}{4}$″ and $34\frac{1}{4}$″. Average heights for age are 57″, $53\frac{1}{2}$″ and 44″ respectively. In each the head is large. The cranial vault is well developed, the forehead prominent and parietal eminences marked. The nose is flattened, but the bridge is not deeply depressed in the manner seen in cases of achondroplasia. The upper limbs are of normal proportional length. Arm and forearm, thigh and leg show a normal relation to each other. (Compare with achondroplasia, in which rhizomelic shortening occurs: see our p. 374.) The lower limbs are not shortened as in achondroplasia, but they are curved so as to lessen the distance of the trunk from the ground, so that the umbilicus is below the mid-point of a vertical line from vertex to the soles of feet. (In achondroplasia this mid-point is displaced as high, or nearly as high, as the ensiform cartilage, owing to the shortness of the lower limbs.) The hands are short, broad and fleshy, but the fingers are not conical in shape; the hand is not "en trident." The hands are like those of myxoedematous rather than of achondroplasic dwarfs. The pulp of the fingers projects beyond the nails. The nails are attached at an angle to the dorsal surface of the distal phalanx. The feet show similar changes. There is enlargement at the epiphyseal junctions of wrists and ankles and at the junction of the ribs and costal cartilages. Radiograms of the hands show marked delay in the appearance of the centres of ossification. Those present in the child aged 7 years, correspond to those present in a normal child at the age of 2 years or 3 years, or are even less advanced. In the child of 13 they correspond to those of a normal child of between 7 and 10 years. (The epiphyses of metacarpal bones normally begin to ossify between the 3rd and 5th years. In the case of the child of 7 no epiphyseal ossification can be seen except the centres for the heads of the metacarpal bones of the fingers.) In all three children the intelligence was normal. They were all three treated with thyroid extract, thymus extract, mercury and potassium iodide and by anti-rachitic measures for long periods without any appreciable result. III. 8, the youngest, was seen subsequently, at the age of 19 years. Her height was then 3′ 6″, and circumference of head $22\frac{1}{8}$″. Her face was childlike, the hair was fine, but not thin, the skin natural, so that cretinism was excluded. She was very sensitive

[1] Owing to a slip of the draughtsman I. 1 and 2, which should be normals, are given by wrong symbols on Plate LIV.

as to her condition and complete examination was impossible. III. 7, was then dead. Died aged 20 years of cerebral haemorrhage. A post-mortem examination was made and an inquest held, but no further facts were obtainable. III. 6, was still alive and still a dwarf, but was not seen. The sexual functions were normal in all three. "The condition seems to be due to delayed appearance of the ossificatory process. All the bones were affected except those of the vault of the skull." These authors also give an account of another case, of the same kind, in a woman aged 42 years, but she had no known relatives similarly affected. (See Bibl. No. 575, p. 193.)

Fig. 705. *Schaaffhausen's Case.* Schaaffhausen gives two accounts of this case. The first just after the death of II. 4, in 1868, the second after the exhumation of his skeleton in 1882. The family who were called Lehnen were induced to permit the exhumation by a medical student Peter Lehnen. Similarity of name suggests relationship, but nothing is said on that head. I. 1 and I. 2 were tall. They had nine children, eight boys and one girl, II. 1—9. II. 1, was tall, II. 2—3, were about 5 "Fuss" high. Of the others four, II. 4—7, were dwarfs, and the two others, II. 8—9, "sind zwar schon mit 6 Jahren gestorben, aber man sah dass auch sie klein bleiben worden." II. 5, was same height as II. 4, II. 6, was 5 "Zoll" higher. II. 7, also a dwarf, who was dead, had like the others a high voice and no beard. II. 4, who died aged 61, was seen by Schaaffhausen and there was an autopsy. His height was 94 cm., and the size of his head was not remarkable. The circumference of his head was 520 mm., length of head between forehead and occiput 170 mm. Maximum breadth of head 150 mm. Weight of brain 1183·33 gms. Weight of body 45 "Zoll Pfund." His mental faculties were good. His milk teeth had fallen out at the age of 22. As an old man he had lost most of his front teeth, but had no gray hair or sign of baldness. He had a high voice, no hair on his face or elsewhere except on head ; his nose was undeveloped, the under lip broad and chin weak. The skull was of a child-like type which showed itself especially in the projecting protuberances of the skull. All the sutures were open. The brain showed numerous convolutions and deep sulci. The internal organs were like those of a child aged 6 years. He had bi-lateral cryptorchism. After exhumation the following measurements were given :—Length of femur 22 cm., of tibia 16·20 cm. Length of skull 164 mm., breadth 147 mm., height 121 mm. Schaaffhausen accounts for the difference in length of skull at autopsy and after exhumation by assuming it became shortened in the grave (?allowance for flesh). Almost all the epiphyses of the long bones were ununited, many of them separated easily from the diaphyses. The only parts of the skeleton which were of normal size were the teeth and ossicles of the ear. This dwarf died at Coblenz and is generally known as the Coblenz dwarf. It may be noted that the dwarf artist Jacob Lehnen was born near Trier and the two places are not so far apart as to render a family connection improbable. (Bibl. No. 136, S. 26.)

Fig. 706. *Tissié's Case.* I. 1, a carter living in the vicinity of Bordeaux. I. 2, his wife, occupation housework. They are cousins "germans." Both are of average height and both are in good health. No alcoholism or syphilis. "No heredity of dwarfism." II. 1, aged 25 years, is of height 1·64 m. II. 2, aged 23 years, is of height 1·75 m. Both have performed their military service. II. 3, aged 21 years. Height 0·98 m. II. 4, aged 20 years. Height 1·11 m. II. 5, child died young. Age and sex not stated. II. 6 and 7, aged respectively 12 years and 5 years, are developing normally. In the case of II. 3, aged 21 years, the pregnancy began three months after the birth of the second child. A normal pregnancy followed. No nervous or moral shock or trauma occurred during it. An easy labour occurred; breech presentation. Child very small at birth. Its growth was very slow. Weaned five or six months after birth, then bottle fed. "Favus and eczema" at the age of 5 or 6 months. Did not begin to walk until 1 year and 10 months of age. Body well proportioned. Torso developed. Thighs and arms normally proportioned to the height. Hands spatulate, the second and third phalanges being arrested in development. Persistence of cartilage of ossification in various bones. An exostosis on the lower end of left humerus. The toes present peculiarities similar to those of the fingers. The great toes are relatively enormous. Menstruation regular since 16 years of age. Marriage in view. She is intelligent. She occasionally falls out of bed at night under the influence of dreams but has no active somnambulism. II. 4, shows the same kind of arrest and development as his sister. At birth, head presentation and easy labour after a normal pregnancy. He is normally proportioned and is intelligent. He has been to school but has not learned to read or to write. He shows complete development of the sexual instinct. Neither of these dwarfs has any enlargement of the thyroid gland. (See Bibl. No. 321, p. 408.)

Fig. 707. *Schreier's Case.* I. 1 and 2, peasants living in Stromholz in Holstein, were both healthy and both of strong constitution. They had 13 children by their marriage. Of these, ten were from $6\frac{1}{4}'$ to $6\frac{1}{2}'$ (German) in height, but three were of dwarf stature, II. 2, 3 and 5. II. 3, Johanna Green, at the age of 9 years, measured 16″ in height ; at the age of 18 years she was 23″ (German) in height and weighed 20 pounds (German) ; at the age of 34 years her height was 2′ $6\frac{1}{2}$″ (German). She was normally proportioned in every way. At her confinement it was proved that the pelvis was of normal size relatively to the rest of the body. It was broad and normally formed, with a conjugate of 3″; the sacral promontory could scarcely be felt. "It was obviously not a rickety pelvis." On the death of her parents in her 9th year she left her birthplace and went to Eckernförde. Here she had to contribute to

her maintenance in part, by her work, as her foster-parents were poor. Having lived with these people, from whom she did not receive the kindliest of treatment, for $2\frac{1}{2}$ years, she went to a widow, an innkeeper. She remained with this woman $3\frac{1}{2}$ years, during which time she learned the essential handicrafts such as sewing, dressmaking, etc. When she returned to her brethren, these had determined that she should join a troupe of travelling players; but in this project they were forestalled, for just then Herr Hawlitschek came to that place with three dwarfs on show. Johanna and her sister, Catharine, II. 2, now 40 years of age, determined to join this show. Herr Hawlitschek observed that she had a talent for entertaining by singing and declamation. By the time she was 18 years of age she was an accomplished singer, harpist and reciter of small pieces. She visited, in this way, the chief cities of Germany, Switzerland and France, was presented to many notable persons and received many presents. She then returned to Holstein and subsequently went to Hamburg, where, during an illness, she came under the care of Dr Schreier, who attended her during a confinement. The child, III. 1, a male, was delivered dead (having died several days before birth) after perforation of the head. The child was normally developed except for an "external hydrocephalus," and was of almost normal size; it was certainly viable except for the "external hydrocephalus." II. 4, the father of this child, was of normal size. Four weeks after the confinement she went away, taking the child, preserved in spirit, to Sweden, Russia and Austria, where she gave demonstrations and showed the child. She amassed sufficient money to set up as an innkeeper near Altona, where she had been for two years. By this means she supported her whole family. (See Bibl. No. 110, p. 116.)

Fig. 708. *Levy's Case.* In 1858, III. 1, an unmarried female dwarf, aged 39, came to the Hospital as she was enceinte. Her father, II. 1, was a dwarf, her mother, II. 2, of average height, her paternal grandfather, I. 1, was also a dwarf, most likely a court dwarf, as there was a picture of him in the portrait gallery at Frederiksburg. Accurately measured her height was 44″ and she was well proportioned. From her 14th year she had acted as her father's clerk, then she had the advantage of being taken into a noble family for some years and later set up house for herself in town. She was lively, gay and fond of dress. She finally started a pension for men, with the result that she became enceinte. She looked like a girl of 8 or 9 years of age. At first the doctors thought Caesarian section would be necessary, but closer examination led them to adopt other methods. The child, IV. 1, was born and was a well-made thin slender-limbed girl. All attempts to resuscitate it failed. It weighed $4\frac{1}{4}$ lbs. and measured $17\frac{1}{2}$″. The mother died the second day after birth. Measurements of mother:—Length of head $6\frac{1}{8}$″, minimum breadth of head $4\frac{1}{2}$″, maximum breadth 5″. Cephalic index = 81·08, *i.e.* the skull was brachycephalic. Length from vertex to sole of foot 44″; vertex to umbilicus 18″; umbilicus to sole 26″; acromion to capitulum ulnae $14\frac{1}{4}$″; of hand from the styloid process of the radius to the end of the index finger $4\frac{1}{4}$″; lower limbs from the great trochanter to the external malleolus $20\frac{3}{4}$″. (Bibl. No. 121, p. 304.)

Fig. 709. *Virchow and Maass's Case.* (The Burmese Dwarfs.) These dwarfs were in Castan's Panopticum in 1896, and remained in Berlin three years. They were exhibited by Maass before the Society for Anthropology and Ethnology in Berlin, but Virchow appears to have also had details of them. In 1910, II. 6, was exhibited at Olympia, London, and was seen by Dr Rischbieth, who got further details.—Virchow stated that the Burmese company with which they were came from Mergui, Upper Burma, and according to the district official of Mergui, the father, I., Mong Sein Bu was dead, no statement was made about I. 2. The three children, II. 4—6, were with the company. II. 4, and II. 6, were dwarfs, and II. 5, was normal. II. 4, and II. 6, were charming little things with well-proportioned bodies, bronze-coloured, with well developed teeth, long smooth hair and black eyes. II. 6, had a slight convergent squint. The adults with them, two males and a female, who were probably relatives were well grown and of medium height. The measurements given do not agree. The district official of Mergui on July 27, 1896, described them thus: Phatama, II. 4, age 14, height 31″. Kyn Lui, II. 5, age 11, height 41″. Samar Arm, II. 6, age 10, height 28″. Maass' measurements in the same month and year are: Fatma, II. 4, age 16, height 65 cm., Smaûm (alias Smaûl), II. 6, age 14, height 60 cm. Virchow's measurements were II. 4, height 74·6 cm. II. 6, height 68·2 cm. II. 5, Kyn Lui or Julei, age 11, height 125·9 cm. Head measurements are also given. Length of head, II. 6, 127 mm. II. 5, 171 mm. II. 4, 126 mm. Breadth of head, II. 6, 111 mm. II. 5, 141 mm. II. 4, 102 mm. Cephalic index, II. 6, 87·4. II. 5, 82·4. II. 6, 80·9. Further measurements are given but it is stated they were not altogether trustworthy owing to the uneasiness of the children and the indefinite limit of their hair. Rischbieth states that I. 1, and I. 2, had four normal sons, three other normal sons, II. 1—3, have therefore been entered in the pedigree in addition to Kyn Lui, II. 5. He also was told that II. 4, was a dwarf and "died young." He thus describes II. 6, "Smaûn Sing H'poo, aged 26, is a 'Burmese Ring Performer' or 'Music Hall Artist.' No physical examination, measurements or radiographs were permitted, so that this account is of little value. His height is approximately 2′ 9″ to 3′, being inferred from comparison with tables and other surrounding objects. He has marked internal strabismus of the L. eye, and no hair about the face. The facial expression is that of an adolescent of his race. The bridge of the nose is not depressed, the frontal and parietal eminences are not prominent and indeed the cranium appears relatively small and round, so much so as to suggest

microcephaly. The chin is somewhat receding, but he has no symptoms of idiocy. The length of the imbs relatively to trunk and head and the proportions of one segment to another are those of an ordinary adult. The limbs are rather slight but otherwise well formed, they show no curvatures and are not shortened. The hands show no peculiarities. The skin is normal, the hair thick and not dry or brittle. The voice is squeaky and childlike. He is intelligent, vivacious, quick of movement and in no way defective mentally. His self reliance is shown by the fact that he roller-skates on a crowded rink at Olympia amongst individuals of ordinary size and of many times his own weight (many of whom are not expert, judging by the number of downfalls that occur) without being crushed and killed. A photograph of this individual is shown on Plate DD (54). His condition presents no resemblance to cretinism or infantile myxoedema and appears to be an example of true dwarf growth or ateleiosis of the type shown by Caroline Crachami in which the cranium takes part in the general hypoplasia, in contrast to the usual condition in which its capacity is proportionately greater than usual. He does not appear to be an example of microcephalic dwarfism, as he is in no way mentally defective, but possesses considerable intelligence, though possibly somewhat childish for his age. This is however difficult to judge owing to his environment [those of a 'show' dwarf in a large collection]. He hugs and kisses a small female dwarf in this collection, to whom he appears to be much attached." (Bibl. No. 323, p. 524.)

PLATE LV. Fig. 710. *Wood's Case I.* III. 6, Calvin Philips was born at Bridgewater, Massachusetts, Jan. 14, 1791. When born he weighed scarcely two pounds and his thigh was no thicker than a man's thumb. He had none of the ordinary ailments of childhood except whooping cough. He was weaned at seven months, walked at 18 months, but did not speak till he was four years old and ceased to grow at five years. His teeth came at 10 or 11 months, and he had the usual number. At the age of 8, his height was $26\frac{1}{2}''$ and his weight 12 pounds including clothes. He was active, playful, sprightly and much devoted to childish sports, but his mental attainments were not up to those of normal boys of his age. His figure was well proportioned and his face though thin and delicate was regular and agreeable and much matured beyond his years. He had five brothers and sisters, III. 1—5, all of ordinary height. His parents, II. 2—3, were normal, II. 2, being aged 24, and II. 3, aged 26 at his birth. His grandparents, I. 1 and I. 2, exhibited him in New York in 1810. I. 1, was a big robust man aged 56. No measurements are given. Garnier (Bibl. No. 205), who also gives an account of this dwarf under the name Philippe Calvin, says he died aged 20, of "old age." Wood says nothing about his death. (Bibl. No. 138, p. 385.)

Fig. 711. *Kirby's Case I.* II. 1, Nanette Stocker was born at Kammer in Upper Austria. Her parents, I. 1 and I. 2, were of ordinary stature as was also a younger brother, II. 2. She was a 10 months and 24 days child and larger when born than children usually are. She did not exhibit anything remarkable, except that she was rather unwieldy till the age of 4, when she ceased to grow. She was then 33″ in height. She was well formed, well proportioned and never ill. She had a talent for music and was exhibited by her tutor with John Hauptmann aged 20, and four years older than herself. He was born at Ringendorff near Bousvillers, Dept of Lower Rhine; his height was 36″ 2‴, and he had an extraordinary talent for music. In 1815, II. 1, was aged 33, weighed 33 pounds, and measured 33″. She died in Birmingham in 1819. Her portrait and that of John Hauptmann, reproduced from Kirby's *Wonderful Museum*, are given on Plate 11 (69). (Bibl. No. 53, p. 228.)

Fig. 712. *Wood's Case II.* II. 2, Robert Skinner was born at Ripon, Yorkshire. No note of his parents or other relatives is given. He was 2′ 1″ in height and married II. 3, who was an inch taller. She was Welsh and her Christian name was Judith. They were married at St Martin's Church and lived together 23 years and had fourteen children, III. 1—14, all well grown and healthy. They were exhibited in London in 1742. II. 2, was then aged 44. They only exhibited themselves for two years. II. 3, died in 1763, and II. 2, in 1765. He is said to have left £22,000. (Bibl. No. 138, p. 350.)

Fig. 713. *Gilford's Case I.* II. 6, aged 28, was born in Layer Breton, Essex, Feb. 23rd, 1874. Photographs of I. 1 and I. 2, and some of their other children showed they were of ordinary size. I. 1 and I. 2, had eight children II. 1—8, six sons and two daughters, one of whom had died of pneumonia. II. 6, had measles in childhood and had influenza twice since 1899. He was of average size when born and cut his teeth at the usual time. It was first noticed he was not growing when between 1 and 2 years of age, though there was nothing to account for it. He went to school at 10 years old and left at 16 after passing the sixth standard. He was first seen when aged 23. His size and general configuration were those of a child; the head was large in proportion to the body and the outlines of the muscles were hidden by fat. The head was broad and of great depth from the sagittal suture to the ears. The face was broad and the bridge of the nose sunken. Sexual hair was absent, but there was plenty of hair on the head. The mental development and tastes were somewhat childish. He was fond of reading and capable of steady application to his work and earned 3*s.* a week as under-gardener. The organs of special sense appeared normal. The external organs of generation were in size and appearance

like those of a child of 3, the testicles were undescended. All the bones were slightly formed, but there was no irregularity of ossification anywhere:—*Measurements.* Height when just over 23, 107·8 cm.; 1 year later 108·6 cm.; 3 years later 109·6 cm. Circumference of head 49·3 cm.; maximum length of head 17 cm.; breadth 13·9 cm. Round chest in nipple line (inspiration) 62·6 cm.; (expiration) 61 cm. Round abdomen 56 cm. From acromion to elbow 20·75 cm. External condyle to styloid process 17·3 cm. Length of hand 11·75 cm. Great trochanter to external condyle 28·5 cm. Lower end of femur to internal malleolus 24 cm. Length of foot 17 cm. Weight 25 kilogs. The middle point of the total length was 1 cm. above pubis. (Bibl. No. 403, p. 316.)

Fig. 714. *Gilford's Case II.* This is the case of a French dwarf. I. 2, came to Gilford to seek advice for obesity, she was of ordinary stature and said her husband I. 1, and two of her children II. 1—2, were of normal stature and she did not know any member of the family who had ever had any abnormality of growth. II. 3, aged 18 was born in Paris, she was small at first but grew at the ordinary rate to a little over 2 years of age. After this she continued to grow at a uniform but diminished rate. She had attacks of bronchitis in childhood but was otherwise healthy. She showed no signs of approaching puberty and in almost every respect resembled a child, but her intelligence seemed more mature than is usual in a child of her height. She was lively but not restless, her hair was fine, eyes full and the nose was depressed at the bridge. Her teething began in the 8th month, and the teeth formed two irregular rows much crowded and displaced. Ossification was little more advanced than in a child of 6. There was no hair on her body, the breasts and sexual organs were undeveloped, the pelvis that of a child. The mother said she could walk 3 kilometres (2 miles), and had walked 6 kilometres ($3\frac{3}{4}$ miles) without undue fatigue. She was an expert dancer and gained her living on the stage: *Measurements.* Total height 85·0 cm. Circumference of head 44·5 cm. Round chest in nipple line between inspiration and expiration 47·5 cm. Round abdomen at umbilicus 46·0 cm. Length of arm 15·3 cm.; forearm 14·0 cm.; hand to extremity of middle finger 10·75 cm.; thigh 23·0 cm.; leg 25·0 cm.; foot 12·5 cm. The photograph of this dwarf is reproduced on Plate AA (42). (Bibl. No. 403, p. 320.)

Fig. 715. *Kirby's Case II.* II. 5, Simon Paap was born at Zandvoort in Holland in 1789. His father I. 1, a fisherman and his mother I. 2, were normal. They had four other normal children II. 1—4, two sons and two daughters. II. 5, exhibited no sign of anything remarkable till he was 3 years of age, when he stopped growing. He was exhibited at Bartholomew Fair, Smithfield, in 1818. His height was then 28″ and his weight 27 pounds. He was handsome and well proportioned in his limbs and body, but his head was disproportioned being rather large. He spoke Dutch, French and English with fluency and correctness. Garnier states that he died at Dendermond, Dec. 2nd, 1828. His portrait is reproduced from Kirby on Plate RR (97). (Bibl. No. 53, p. 147.)

Fig. 716. *Wood's Case III.* I. 1, a little German woman, the "Dwarf of the World," in July, 1700 was "at the brandy shop over against the Eagle and Child, in Stocks' Market," where the Mansion House now stands. She was only 2′ 8″ in height, the mother of two children and was "carried in a little box to any gentleman's house if desir'd." Her handbill runs as follows: "At the Brandy Shop, over against the Eagle and Child in Stocks' Market is to be seen any hour of the day, from eight in the morning till nine at night, a Little German Woman, the Dwarf of the World, being but 2 foot 8 inches in height, and the mother of 2 children, as straight as any woman in England, she sings and dances incomparable well, she has had the honour to be shown before Kings and princes, and most of the nobility of the land; she is carried in a little box to any gentleman's house, if desir'd." Another handbill of the same person and period, states that she was 49 years of age. (Bibl. No. 138, p. 307.)

Fig. 717. *Home's Case.* This is the case of Caroline Crachami. Her mother I. 2, an Italian or Sicilian woman aged 20, was travelling in a caravan with the baggage of the Duke of Wellington's Army in the Peninsular War and was frightened by a monkey which got under her clothes while she slept, she being then three months gone with child. She did not miscarry, but the child II. 2, when born weighed 1 pound and was 7″ long. II. 2, was brought to London and shown as a curiosity and died after completing her ninth year. The *Times* of June 17th, 1824, gives an account of how the body was stolen and of the distress of the father I. 1. The skeleton is now in the Royal College of Surgeons, London. When seen by Home she could walk alone but with no confidence, her sight was very quick, her voice shrill and she had some taste for music. I. 2, had a fifth child II. 3, in Ireland, who like her three first, II. 1, was naturally formed. The following measurements and description of the skeleton are given by Hastings Gilford (Bibl. No. 403.) Height 49 cm. Middle point of total height 1·3 cm. above symphysis pubis. Length of spine 5·8 cm. (? 15·8), of clavicle 4·8 cm. Acromion to elbow 8·9 cm. External condyle to lower end of radius 7 cm. Femur 12 cm. Tibia 9 cm. Total length of lower extremity 23 cm. Foot 6·18 cm. Hand 6 cm. Maximum length of skull, 12·6 cm. Maximum breadth 9·4 cm. Vertex to base 8·55 cm. Circumference 33 cm. The skull is very thin and delicately formed. The anterior fontanelle is open for a length of 1·6 cm. and width of 1·2 cm. The lower half of the frontal suture is closed, but there is a small unossified oval area 6 mm. long near the superior angle of the occipital

bone close to the lambdoid suture on the L. Another is present at the bottom of each occipital groove. The ear bones are of adult size[1], but the tympanic bone is a mere ring, like that met with in infants. Ossification throughout is generally delayed. This is shown in the backward condition of the epiphyses and the smallness and delicacy of the shafts. The long bones possess hardly any curve. The ribs have very abrupt curves at their angles and are almost straight from thence onwards. See our p. 368 and Plate Z (38). (Bibl. No. 58, p. 191.)

Fig. 718. *Gilford's Case III.* I. 1, was a "small-made" man rather below medium height. I. 2, was of average height and so were her children, II. 2, aged 28, excepted. II. 3, aged 13, was 1·34 m. in height, and I. 2, said she believed II. 2, was about the same height at the same age, he seemed to stop growing at that time, but she could not account for the circumstance. He was healthy at the time and had no illness before or since; his proportions and appearance were those of a boy of 14. His occupation was that of a farm boy, but he was too stupid to do better work than minding sheep. He was capable of working all day and then taking a four miles' walk without being tired. His hands and feet were rather large and there was slight kyphosis and lordosis of the spine. He was also knock-kneed and flat-footed to the same degree so that his gait was somewhat awkward and shambling. The hair of his head was fine and thin and he had plenty of lanugo on the body and limbs. His teeth were sound and dentition regular, there was no sign of syphilis. The ossification was that of a boy of 14 or 15. The special senses were of ordinary acuteness. The external genital organs were of the size and appearance of those of a child of 8 or 9. There was no pubic hair, the R. testicle was undescended, the L. could be felt half-way down the inguinal canal. He was unusually timid and modest. Three months later he got ill, pneumonia set in, he had a series of eclampsic attacks and died in one of them. There was a post mortem. *Measurements.* Weight 35·6 kilos; brain 1275 grms. Height 146 cm. Circumference of head 52 cm.; length 174 mm.; breadth 142 mm. Circumference of chest round nipple line 73 to 75 cm. Circumference of abdomen at umbilicus 69 cm. Length from acromion to elbow 24·5 cm.; external condyle to styloid process of radius 22 cm. Length of hand 17·5 cm.; from great trochanter to external condyle 39 cm.; external condyle to external malleolus 36·3 cm.; of foot 25·3 cm. Middle point of total height 2 cm. above pubis. (Bibl. No. 403, p. 335.)

Fig. 719. *Garnier's Case.* II. 1, Lucia Zarate a female dwarf was born at San Carlos near Vera Cruz, Mexico, of Spanish parents I. 1, and I. 2, who had other normal children II. 2. She could speak Spanish and a little English. She weighed 2½ pounds (livres) at birth. Her height is not given but a picture of her and General Mite with whom she was exhibited is shown. From this she appears to be a true dwarf. The cranium is proportionally small and she is probably an example of Hastings Gilford's Group I as were Caroline Crachami and Bébé. (Bibl. No. 205, p. 230.)

Fig. 720. *Illustrated London News. Case I. The Illustrated London News* of May 24th, 1851, gives a picture of this dwarf II. 1, and her mother, and also a short account of her. Wood (Bibl. No. 138) also gives an account of her and thinks she is identical with a Miss Gibbs, daughter of a farmer named Gibbs, of Blean near Canterbury, who was exhibited. Her parents I. 1, and I. 2, were of normal stature and no note is made of any other children. When exhibited at the age of 14 months, her height was 16″ and her weight 5 pounds. Her feet were 2″ long and she possessed the utmost regularity of limb and feature. She was exhibited at University College, London, to more than 500 doctors. (Bibl. No. 101, p. 450.)

Fig. 721. *Illustrated London News. Case II.* The three dwarfs in this case were exhibited in the Cosmorama, Regent Street, and a picture of them appeared in the *Illustrated London News* of May 30th, 1846. They were natives of the county of Ross and were born in the province of Lochcarron; their father, I. 1, was a shepherd of the district, but nothing is said about either his stature or that of his wife I. 2, nor does it state whether there were other children besides the three dwarfs. II. 1, the eldest (the central figure in the engraving), was 23 years old, 45″ in height and weighed 5 stone 11 lbs. II. 2, aged 21, was 44″ in height and weighed 5 stone 3 lbs. II. 3, aged 19, was 44″ in height and weighed 5 stone 10 lbs. In the picture their limbs look quite in proportion to their height, but the heads look a little large. (Bibl. No. 90, p. 357.)

Fig. 722. *Magitot's Case.* This case is also described by Larrey. I. 2 and I. 3, were both well formed. They had three children, II. 1—3. II. 1, aged 17, was quite normal. II. 2, who died a few days after birth, was also normal. II. 3, the third child, born when his parents were both 27 years old, was a dwarf. As far as the parents knew no such anomaly had occurred before in their families. The mother said she got a great fright in the fifth month of her pregnancy with this child. He was so small when born that he was wrapped in a pocket handkerchief, but he was neither measured nor weighed. Nothing particular happened during his infancy except a fall which caused incomplete dislocation of the right knee outwards; this dislocation had never been reduced, which accounted for the limp which he had. When seen, at the age of 14, he was in general well proportioned, one might almost say there was complete equilibrium between the different parts of his body; but the height of his head relative to the height of

[1] This of course is the normal case, the bones being full-sized at birth.

his body was not quite normal according to the canons of sculpture, and another disproportion existed in the extraordinary size of his nose and its projection from the middle of the face and of the intermaxillary, without however any trace of hare lip. His nose measured 4·5 cm. in total length and was 2 cm. broad at the base. I. 1, a paternal uncle, had the same kind of nose. His skin was rather dark, his hair dark chestnut, smooth and abundant. The limbs, hands and feet were regularly proportioned, but the feet were flat and the hand only showed a weak development of the thenar eminence. He weighed 9 kilogs. His intelligence was moderate, about that of a child of 10. He could read and write, had a good memory and sang various romances of his country. He had no hair in the axillae or on the pubis. *Measurements.* Height 93 cm.; of external auditory meatus from ground 87 cm.; the acromion 75 cm.; epicondyle 56 cm.; styloid process of the radius 44 cm.; medius 33 cm.; umbilicus 57 cm.; symphysis pubis 45 cm.; antero-superior iliac spine 51 cm.; knee 27 cm.; external malleolus (flat foot) 2 cm. Width of shoulders posteriorly (back slightly curved) 20 cm.; anteriorly 15 cm. Head measurements: Maximum ant. post. diameter of the skull 14 cm. "Diamètre iniaque" 14·5 cm. (?). Maximum transverse diameter 12 cm. Bi-auricular diameter 10 cm. Bi-temporal diameter 10 cm. Horizontal circumference of skull 39 cm. "Courbe iniaque" 35 cm. (?). Height of face 14 cm. Bi-zygomatic diameter 9 cm. Bi-malar diameter 7 cm. Maximum vertical diameter of the head 14 cm. The length of the little finger relative to the ring finger was such that its extremity did not quite reach the level of the last inter-phalangeal joint like that of the normal individual. The lower limbs exhibited nothing particular except complete absence of calf. His legs were thin, but he was agile and adroit in spite of his limp. The genital organs were normal. His dentition was not so advanced as is normal at his age; his teeth were free from caries, and his milk teeth, of which he had lost four or five, were according to his parents equally healthy, but those which remained were deformed and atrophied. (Bibl. No. 186, p. 692.)

Fig. 723. *Zagorski's Case.* I. 1, aged 25, came to Basel Hospital for her confinement. Her physical and mental development was very backward and she would answer few or no questions. Her people stated she had always been healthy and had done her share of housework well. Caesarian section was performed and the child, a female, was delivered alive; it was normally proportioned, showed no abnormality and weighed 5 Pfund (civil weight). The pelvic measurements of mother were:—True conjugate 4″ $2\frac{1}{2}$‴ (Swiss), 4″ 8‴ (Paris). Anterior transverse diameter 2″ 8‴ (Swiss, 3″ 1‴ Paris). Posterior transverse diameter 3″ 4‴ (Swiss, 3″ $9\frac{1}{2}$‴ Paris). Diagonal conjugate 4″ 8‴ (Swiss, 5″ 4‴ Paris). Antero-posterior diameter of true pelvic cavity 4″ (Swiss, 4″ 6‴ Paris). Depth of pelvis (vertically) 2″ 8‴ (Swiss, 3″ 1‴ Paris). I. 1, died. On autopsy: The body was very small; length 139 cm.; length of humerus 20 cm.; of forearm 20 cm. Two series of measurements of the pelvis are given, one taken during life, one after death; it was of that unusual variety, pelvis nana or dwarf's pelvis. In size and shape it was that of a child of 6 or 7 years of age. (Bibl. No. 137, p. 57.)

Fig. 724. *Ludwig's Case.* This case is quoted by Lawrence from Ludwig. Ludwig himself says that II. 1, Catharina Helena Stöberin, of Nürnberg, was about 3″ (3 Fuss) high in her 20th year, well proportioned and intelligent. Her pulse was weak like that of a child. Her parents, I. 1, and I. 2, and her brothers and sisters, II. 2, were dwarfs. Ludwig refers to Lavater and Wünsch, neither of whom, however, mentions any relatives. Garnier states he could find out no particulars with regard to C. H. Stöberin. There are particulars of her in various books (see Iconography, No. 138). She was certainly a true dwarf, but for the details of her family we can depend only on Ludwig's statement. (Bibl. No. 49, p. 154.)

Fig. 725. *Michaelis' Case.* I. 1, was a small Jewess about 4′ high and quite normally proportioned. Measurements of pelvis were:—External conjugate 6″; interspinous 6″ 9‴; intercristal 8″ 3‴; intertrochanteric 9″ 9‴; diagonal conjugate 3″ 11‴. She bore the two children, II. 1—2, without difficulty in labour. They only weighed 5 lbs. (Bibl. No. 102, p. 163.)

Fig. 726. *Naegele's Case.* I. 1, was a strong man rather above average height. I. 2, who had been dead 23 years, was said to have been of small stature. II. 3, aged 29, was of medium height, healthy and of good appearance. II. 2, aged 31, was only 3′ 6″ in height but otherwise well proportioned, her head and limbs were in normal proportion to the size of her body and except for her features she looked like a child of 7. The joints of her extremities showed no trace of enlargement. Her mental powers and inclinations resembled those of a child. She had been healthy from infancy, was good-humoured, and on this account much beloved by her father; she was diligent and active in any household work suited to her powers. She menstruated at 18. Her first intercourse with a strong man was very painful and accompanied by great loss of blood; she had been with him altogether 10 times. Throughout her pregnancy she was quite well. She quite understood her position, had thought about it and spoken to others and absolutely refused to allow Caesarian section; and her father only permitted her to be under Naegele's care when she was six months pregnant on condition no such operation should be attempted. Delivery was by forceps; the child, a boy (sex omitted on Plate), weighed 3 Pfund 6 Loth (civil weight), and was born dead. The mother died on the 10th day after birth of "indigestion caused by eating dainties" of which she was very fond. The pelvis appeared to be a perfectly well-formed female pelvis on a small scale. Measurements of

pelvis:—From the sacral promontory to the subpubic angle 3″ 3‴. From tuber ischii to crista ossis ilii 5″ 5‴. From tuber ischii to linea innominata ossis ilii 2″ 7‴. Height of the symphysis pubis 11‴. Antero-posterior diameter of pelvic inlet 3″. Transverse diameter of pelvic inlet 3″ 7‴. Transverse diameter of pelvic outlet 3″. Antero-posterior diameter of the pelvic cavity 3″ 3½‴. Transverse diameter of the pelvic cavity 3″. The vertebral column, the pelvic bones and in short the whole skeleton did not exhibit the soft thin conformation which one observes in rickets: the innominate bones in their iliac portions did not appear short in mass or circumference as in rickets; neither these bones nor those of any other parts showed any trace of any rickety tendency having existed; on the contrary in volume and size they corresponded in every respect to the size of the body. It would be hardly possible to see a better proportioned frame than the skeleton of this individual. (Bibl. No. 81, p. 181.)

Fig. 727. *Paltauf's Case.* II. 4, came to Hospital at age of 49. He had worked for some years as private servant to a Colonel and later as a gardener and had got rheumatic affections in his knees. Some years after he had general oedema and three weeks before entering the Hospital he developed dropsy. He died 12 days after entrance. His parents, I. 2 and I. 3, and his sister, II. 3, were of normal stature and he had two half-brothers (whether paternal or maternal not stated), II. 1—2, who were big strong men, one of them, II. 1, died after suffering for a year from cough and chest affection. II. 4, was 112·5 cm. in height with thin and light bones and moderately developed muscles. *Measurements.* Head: Circumference of head 54 cm.; length of face 18 cm.; mento-occipital diameter 22½ cm.; bi-temporal diameter 14 cm.; bi-parietal diameter 15 cm. Thorax: Breadth at highest point of axillae 15·5 cm.; antero-posterior diameter at height of second rib 15·5 cm., at height of nipple 16·5 cm.; length from clavicle to arch of ribs at nipple line 19·5; circumference at the level of the nipples 67 cm. Pelvis: Distance between the antero-superior iliac spines 18·5 cm.; between the iliac crests 19·5 cm. Extremities: From acromion to distal extremity of middle finger 52 cm.; styloid process of ulna to olecranon 18·5 cm.; styloid process of radius to deepest point of the fossa cubitalis 16 cm.; great trochanter to external malleolus 56 cm.; internal condyle of femur to internal malleolus 28 cm.; great trochanter to external condyle of femur 26 cm. Abdomen: Circumference at a level two fingers' breadths below the extremity of the xiphoid process of the sternum 73 cm.; midway between the extremity of the xiphoid process and the symphysis pubis 79 cm. When dissected the body measured 111 cm. The head appeared relatively large; the face broad and short. The neck was short, the abdomen prominent and rotund, the hands and feet very small. (Bibl. No. 262, p. 6.)

Fig. 728. *Virchow and Nagel's Case.* II. 9, Pauline Musters has been described many times. Virchow described her in 1882, Bouchard in 1884, Mortillet in 1885, Manouvrier also wrote of her though apparently he had not personally seen and measured her; the last description is that of Nagel who was present at her death. II. 9, was born at Ossendrecht, Holland, Feb. 26th, 1878 (Nagel gives date 1876). Dr van der Moolen, in a letter to Bouchard, says that the doctor who attended her mother, I. 2, at her confinement had given him particulars and that the uterus with its contents had remained nine months in the pelvic cavity, to this fact he attributed the small size of the child. I. 1 and I. 2, were Dutch. Van der Moolen, who knew the family, states I. 2 was well made but thick-set and I. 1, a cobbler, was a drunkard. This couple had 12 children, II. 1—12, of whom II. 9 was the seventh. II. 10, a seven months child, was still-born and was said to have been even smaller than II. 9, but Bouchard expresses some doubt on this point. When Nagel wrote there were six sisters, II. 1—6, and two brothers, II. 7—8, all rather above average height and all alive. Mortillet mentions seven sisters. All accounts agree in stating II. 9 was well-made; three weeks after birth her length was 30 cm. and her head, trunk and limbs were all in proportion. She was of average intelligence. Bouchard, Virchow and Nagel all give tables of measurements, Virchow's and Nagel's measurements are given below. Nagel says she was nearly perfect in bodily development, of rather pleasant features, graceful in all her movements and of good general education, speaking Dutch, French, German and a little English. Nagel attended her in her last illness, which originated in a simple cold taken during her performances at the theatre where she performed all sorts of acrobatic feats. Before her illness she was said to have been in perfectly good health and free from physical defects of every kind. She had menstruated at 16. Her body had all the characteristics of a fully developed woman. Her breasts were round and prominent and the pubes was covered with hair. Her vitality was lowered by her constant performances and her relatives tried to replenish it by means of stimulants administered after every performance. She consumed in this way large quantities of alcoholic beverages. Her cold developed into bronchitis, bronchitis into pneumonia complicated with meningitis and she died in 10 days. Nagel states her body had become considerably elongated during her illness. *Virchow's Measurements.* Total height 53·8 cm. Measurement of extended arms 53·6 cm. Length of head 134 mm. Breadth of head 107 mm. Height of head from ear 8·2 cm. Circumference of head 36·3 cm.; forearm 6·0 cm.; calf 10·5 cm. Length of hand 6·3 cm.; foot 7·8 cm. Weight about 8 lbs. *Nagel's Measurements.* Height at birth 12″; 19 years old when alive in stockinged feet 19″. Length of body when dead 24″; arm to tip of fingers when dead 12″; leg from hip to tip of toe when dead 12″. Circumference of head 16″. Length from chin to forehead 5½″; chin to ear 3½″. Circumference of chest across breasts 18½″; higher up under axillae 19″; of abdomen 19″; around hips 18″; across waist 17″. Length of foot 4″; hand 3½″. Distance from shoulder to shoulder in front 7½″. Circumference of

thigh 7″; of knee 6″; of calf 4½″. Weight in normal health from 7½ to 9 lbs. Mortillet (Bibl. 215) gives her height as 59 cm. and weight 9 lbs. According to Manouvrier the measurements given by Testut and Bouchard in 1883, when she was 5 years old, were as follows :—Height 55 cm. Head: Maximum antero-posterior diameter 129 mm.; transverse diameter 105 mm.; minimum frontal diameter 51 mm.; bi-zygomatic diameter 75 mm.; maximum horizontal circumference 39 cm. On another page he states her height at age of 5 was 65 cm. and weight 1·5 kilogs. (Bibl. No. 196, p. 215, Bibl. No. 322, p. 369, and Bibl. No. 214, p. 276.)

Fig. 729. *Moreno's Case.* This case appears to be the same as that described by José de Antelo in *La Rivista d'Antropologia*, T. II. 1875, but there is no reference to Antelo in Moreno's paper. I. 1 and 1. 2, were natives of Pilas, near Seville, they were robust and healthy but were the descendants of many consanguineous marriages and had many consanguineous marriages amongst their relatives. Amongst their collateral relatives there appear to have been four females, two sisters in one branch of the family and two sisters in another branch who were either dwarfs or nearly dwarfs. Moreno mentions them and had apparently seen them, but he does not describe them in this paper, nor does he define the relationship between them and the two dwarfs described below. He says they were natives of the same village and members of the same family. He also states that in all the cases there was either a normal birth or births between the dwarfs. I. 1 and I. 2, had 11 children, II. 1—11, of whom nine were tall and healthy and the other two, II. 9 and II. 11, Gabriel and Pedro Benitez Campos, were dwarfs. II. 9, Gabriel, was aged 28. When born he was more robust than the average child, he was breast-fed for a year and had no illness during this time. When seen his skin was dry and flaccid, his complexion pale brown, and his face though animated was like that of an old man. His hair was dark chestnut; he was not handsome either in face or body, but the whole body was developed in proportion except the hands and feet, which appeared a little large. His voice was hoarse as when in a state of change. The testicles were undescended, the penis small. He had no sexual instincts, was gentle and affectionate in character and devoted to his brother Pedro. His intelligence was puerile and very limited and he occupied himself looking after pigs and smoking and drinking brandy. II. 11, aged 25, had a sadder and older looking face. His voice was hoarse, his genital organs in a similar condition to those of his brother, II. 9, his character was reserved and taciturn. He had been breast-fed for only eight months, for his mother became pregnant again and for four or five months he had insufficient nourishment and according to the father suffered from intestinal disorder, ascites, at this time. Moreno calls it "hidropesia ascitis." Neither dwarf had any trouble in dentition and both had retained some of their milk teeth.

	Gabriel	Pedro
Measurements:		
Weight	39½ lbs.[1]	34 lbs.[1]
Height	97 cm.	94 cm.
Head: Antero-posterior diameter ...	17·07 ,,	17 ,,
Occipito-mental ,, ...	19 ,,	18 ,,
Bi-parietal ,, ...	15 ,,	13 ,,
Bi-temporal ,, ...	10 ,,	10 ,,
Fronto-mental ,, ...	12 ,,	12 ,,
Sub-occipital bregmatic diameter	15 ,,	10·05 ,,
Chest: Bi-acromial	20 ,,	20 ,,
Dorso-external	13 ,,	13 ,,

Moreno says these dwarfs were neither cretins nor imbeciles, and the cases are probably ateleiotic. (Bibl. No. 157, p. 157, etc.)

Fig. 730. *Hecker's Case.* I. 2, born at Villmar, was a very strong, short and thick but well-proportioned man, with a broad chest, short neck and thick head. He had been always healthy except for having once had intermittent fever. He was in comfortable circumstances and diligent and clever at his business. He had been twice married. Hecker says that II. 13, Margaret Leonhard, was the eleventh living child of her father and the fifth living child of her mother. Therefore there were apparently six children of the first marriage alive, II. 1—6, and perhaps others had died. Nothing is said about these six children, so presumably they were normal. I. 3, aged 40, the second wife, was well built and flourishing and had never had any serious illness. She was an excellent mother and housewife. She was slightly hysterical. Not related to her husband. She evidently had five children alive, but II. 8 was dead, so that II. 9—11, were alive and possibly she had had other children who had died and others

[1] Spanish.

again who were alive and younger than II. 13. All the living children of this marriage except II. 13 were healthy and well proportioned. II. 8, the first child of the second marriage, a boy who died soon after birth was according to the account of the parents much the same as II. 13 at birth but not quite so small. I. 3, noticed nothing peculiar during her pregnancy with II. 13, the confinement was very easy. The exact measurements of the child at birth were not known, as the parents were ashamed of her and would not show her, but the father said he could cover the whole body with his two thumbs placed together, so she was probably about 9″ (Zoll) long and may have weighed about ¾ of a pound (Pfund). She was born May 16, 1840, and was breast-fed, but got too much milk and for 18 months had almost constant diarrhoea. Hecker saw her first when about 6 months old. She was about as long as a shoe and 1¼ pounds (Pfund) in weight. Her head was about the thickness of a hen's egg and both fontanelles were closed, they were said to have been closed at birth. Hecker saw her constantly after this. Her mental development was rather backward, but her senses were acute. In 1846 (?) the following measurements were made, in Parisian 'Zoll':—Antero-posterior diameter of head 4″. Transverse diameter of head 3¼″. Distance from chin to external occipital protuberance 5¼″. Distance between shoulders 5¼″. Diameter from middle of chest to the corresponding point of the spinal column 3¾″. Distance between the iliac crests 5″. From pubis to the first vertebra of the sacrum 3¼″. Length of arm from the armpit to the tip of the middle finger 8½″; lower limb from the great trochanter to the heel 13″. Distance between trochanters 5″. Weight of body without clothes (bürgerliches Gewicht, das Pfund zu 32 Loth) 11 lbs. She was a well-proportioned dwarf. (Bibl. No. 89, p. 48.)

Fig. 731 *a*. *Maass' Case II.* I. 1, was a railway station assistant, and he and his wife, I. 2, were normal, as were also five of their children, II. 1, and II. 3—6. II. 2, Helene Gabler, was born Nov. 15, 1874, and so was nearly 20 years of age when seen, she was 106 cm. in height. She had a normally proportioned body but looked like a child of 6. Her face was pretty and intelligent, with blue eyes. She was the second of the family, and, according to her mother, never grew after 6 years of age. The mother said her sexual development was that of a child of 6. Her voice was childish. (Bibl. No. 297, p. 364.)

Fig. 731 *b*. *Gilford's Case IV.* I. 2, who died aged 78, was only about 120 cm. in height. He was exhibited as a dwarf in shows for 21 years, part of the time being with Robert Hales, the well-known Norfolk giant. His brother, I. 1, was a tall man, who was in the police force in London. I. 2, married I. 3, of ordinary stature, and had two children, II. 1—2. II. 1, aged 38, unmarried, is said to have been a small child, "like a doll" at birth. She continued growing until she was 13 or 14, and definitely ceased growing at 16, about a year after menstruation began. She is a lively woman of good intelligence, the mistress of a country post and telegraph office. She wears gloves of 00 size and shoes of "children's sevens." She looks healthy and shows a tendency to obesity. Her features are small; her face is inclined to flatness and the type is childish. She resembles II. 2 in appearance. The hair is abundant, and the nails delicate and well formed. The teeth are crowded; the lower incisors seem smaller than usual; many teeth are absent or decayed, and the last molars have not erupted. The knuckles of the hand are prominent and the lobules of the ear almost absent. The head is flattened at the top and almost cone-shaped, with the rounded apex of the cone in front. The thyroid gland is of fair size in proportion to the body. Pubic and sexual hair are abundant and the breasts of fair virgin development. As far as one can judge the digestive, urinary and generative organs are normal. II. 2, brother of II. 1, aged 36, is a boot rivetter, earning 15*s*. a week. He was of the usual size at birth, but nearly bled to death from some defect in the tying of the umbilical cord. It was first noticed he was not growing when he was about a year old, but he thinks he grew more quickly at 14 or 15 and ceased altogether at 17 or 18. He had inflammation of the lungs when 11 years old, but has had good health since. Puberty set in when he was between 16 and 17, but he says his facial hair did not grow thickly until he had turned 20. He married at 25, II. 3, and has had seven children, III. 1—7. Four died when about 3 months old, one of whom was a male, and three were females. All four are said to have been dwarfs, but of this one cannot be positive. Of the three now living: III. 5, is a dwarf boy aged 10, and III. 6—7, are well-developed girls. II. 2, has, like his sister, a cone or pyramid shaped head with the angles worn down and rather flat at the top. Its circumference is that which is usual at about 12, while his height is that of a boy of 10. He is of average intelligence, talkative, and of good disposition. His teeth are all of the permanent set, and are crowded and irregular, many of them being decayed or absent. Of the molars only the first have erupted. His digestive, urinary, circulatory and reproductive organs seem to be normal. The thyroid gland is of proportionate size. He is of fair muscular development and can work all day without getting tired. A radiograph of his hand shows that ossification is completed and that the bones are thicker than is usual in those of his age. III. 5, aged 10, was like other babies at birth and it is not known when growth became abnormal. The father thinks he noticed that his boy was not so big as he should be when he was about a year old. Since then he has grown very slowly, but no measurements have been taken. A normal sister of 6 years is taller than he. The head is shaped

like that of II. 1 and II. 2, its circumference is that which is usual at 2. The face and the proportions of the whole body are like those of a child of about 4. He has passed the second standard at school and seems to be well behaved and of good intelligence. The thyroid gland is present and apparently normal. The teeth are of the first dentition, with the exception that the two mesial lower incisors are just appearing through the gums, one milk incisor being absent and the other loose. The organs are normal with the exception of the reproductive, the scrotum being like that of an infant, while the testes cannot be felt; but the penis does not seem to be quite so backward in development as the testes and scrotum. He is a cryptorchid. A radiograph shows that his epiphysial and carpal ossification resembles that which is customary at 5. Gilford calls II. 1 and II. 2, cases of sexual ateleiosis, and III. 5, a case of asexual ateleiosis. Measurements of the three cases are as follows:

	II. 1 Age 38	II. 2 Age 37¾	III. 5 Age 12		II. 1 Age 38	II. 2 Age 37¾	III. 5 Age 12
					cm.	cm.	cm.
Measurements:				*Measurements:*			
Weight (kilogs.)	—	33·11	16·32	*Upper limb:* Across extended arms	—	132	95·5
				Acromion to external condyle	16·1	22	15·5
	cm.	cm.	cm.	External condyle to styloid			
Height	129·5	132	95·3	process	15·5	20·5	13·8
Sole of foot to fork	—	57·8	42·5	Styloid process to knuckle of			
,, ,, pubes	—	62·8	46·4	middle finger	5·6	7·5	5·3
,, ,, navel	—	72·4	54·2	Middle finger	6	8·7	7
,, ,, top of				*Lower limb:* Trochanter to lower			
sternum	—	1·06	80·3	edge of patella	29·4	33	24·5
Face: Vertex to base of				Lower edge of patella to in-			
nose	—	15·4	12·7	ternal malleolus	25	27·7	20·2
Vertex to chin	—	17·5	16·8	Internal malleolus to ground	4	5·3	4·5
Head: Diameter	13·7	14·8	13·8	Heel to first metatarsal joint	11	14·8	10·7
Length	17·1	18·4	16·7	Great toe	4	5	3·7
Circumference	49·5	53·2	51	Between antero-superior iliac			
Chest: Circumference	67	75	—	spines	18·6	—	—

Ages given in this table of measurements differ from ages in text. Photographs of two of the cases are given in original. (See Bibl. No. 664, p. 596, where a good photograph of II. 2 and III. 5 is given. The measurements are from the *Brit. Med. Journal*, 1904, Vol. II. p. 914 except the height of II. 1 which is from No. 664.)

Fig. 732. *Hitschmann's Case.* I. 1, and I. 2, were normal and not related, they did not remember any deformities or dwarfs in their families. They had three normal children, II. 1—3, of whom II. 3, a son, died aged 35. II. 4, aged 35, was healthy as a child, but the mobility of his right shoulder was limited. He grew like other children till the age of 5, then much more slowly, but he continued to grow till his 25th or 26th year. He attended school and learnt like other children, then joined a Lilliputian troupe of actors and had since been a variety singer. For the last half year he had suffered from violent headache in the occiput and temples. His height was 118 cm., he was regularly built, his head being in proportion to his body; the circumference of the head was 52 cm. The length of the upper arm was 22 cm., that of the forearm 19 cm. His figure was slender [gracil], his gait slow, but not tottering. He was beardless but there was a little hair in the axillae. His voice was like that of a child. A long account is given of his right arm which was defective in function. Probably a case of ateleiosis. (Bibl. No. 340, p. 663.)

Fig. 733. *Aldrovandi's Case.* Ferdinando Cospe of Bologna had two dwarfs, brother and sister, II. 1—2. They were the children of needy parents, I. 1 and I. 2, who were of ordinary height, and were peasants living in the Commune of Bagnarola. The son II. 1, was 26 years of age, and height about 31″. The daughter II. 2, was aged 23, and her height was about 29″. Both were of elegant proportions and much admired. Taruffi translates the heights thus: II. 1, 32 oncie = 101·1 cm. in height. II. 2, 30 oncie = 94·8 cm. in height. The Latin is as follows "Nanus nomine Sebastianus jam annum sextum supra viginti natus altitudinem trium dodrantium cum dimidio vix superat. Nana alterius soror nomine Angelica jam vigesimum tertium agit annum et tamen ejus altitudo tres tantum dodrantes et duas uncias adequat." (Bibl. No. 8, p. 602.)

Fig. 734. *Virchow's Case*: "Dobos Janos." This case was described by Virchow on April 27, 1892, before the Medicinische Gesellschaft of Berlin. The same dwarf was then described by Daniel and

Philippe (*Extrait de Clinique*, August 2, 1902, Brussels), by Lardennois, Bibl. No. 454, and also by von Hansemann. It is the account of the last which is given here. I. 1 and I. 2, were presumably normal as no statement to the contrary is made. The number of their children is given somewhat indefinitely. The account states that II. 5, aged 22 nearly, had still 12 brothers and sisters in 1902, therefore apparently 13 children were then alive, and II. 7, II. 10 and II. 12, were dead, which would make 16 children, but possibly there may have been others. II. 5, was born in March, 1881, and was 11 years of age when seen by Virchow, who states he weighed 500 grammes at birth. He was the fifth child and the first dwarf in the family, and was born at full term after a normal pregnancy and was unusually small at birth. The mother, I. 2, afterwards bore three other dwarfs of the same kind, II. 7, II. 10 and II. 12, after normal pregnancies. These were all unusually small at birth. II. 7, died aged 2 years, II. 10, died aged 10 months, and II. 12, died aged 10 weeks. Between these births were some of the normal children. With the exception of his small size and swollen knotty finger-tips, II. 5 had no deformity. Though his head appeared extraordinarily small it was in proportion to the rest of his body. Rieger (*Sitzungsbericht der Würzburger phys. med. Gesellschaft*, 1895), calculated the weight of his brain at about 600 grammes, he then weighed 13·2 kilos. The head could not be said to be too small for his body, he was not microcephalic. He spoke Hungarian and some French and German, but had never been taught to read and write; he showed no trace of idiocy and conducted himself as a man. He had a sprouting if not a luxuriant beard, and the pubic hair was well developed. He had bi-lateral cryptorchism. His voice had become deeper than formerly and the thyroid cartilage was as prominent in the neck as in an adult man. The cephalic index was not much altered, it was a little over 81, it had formerly been nearly 81.

	von Hansemann Age 22	Virchow Age 11		von Hansemann Age 22	Virchow Age 11
Measurements:			*Measurements:*		
Height of crown of head	114·5 cm.	92·6 cm.	Head: Maximum horizontal length	135 mm.	129 mm.
„ „ shoulder	95·0 „	75·9 „	Maximum breadth	110 „	104 „
„ „ elbow	75·0 „	58·4 „	Height of ear	87 „	86 „
„ „ wrist	57·5 „	43·6 „	Horizontal circumference	395 „	374 „
„ „ middle finger	45·5 „	33·2 „	Height of nose	47 „	39 „
Length of right hand	12·0 „	10·0 „	Length „	45 „	38 „
Breadth „ „	7·0 „	5·0 „	Breadth „	23 „	20 „
Length of right foot	15·2 „	12·5 „			
Breadth „ „	6·0 „	5·1 „			

The glabella-inion line was 139 mm. the vertical height (Calottenhöhe) 75 mm. which gave a height index of 53·8 the index of a well-formed man. (Bibl. No. 401, p. 1209.) Lardennois says Dobos Janos was the fifth of nine children, that his parents and five brothers and sisters were normal, and that three brothers were dwarfs and died young, so presumably II. 7, II. 9 and II. 12 were males. Dobos Janos is stated to be aged 22, height 115 cm. and weight 18 kilos. The rest of Lardennois' description corresponds on the whole with that of von Hansemann. (Bibl. No. 277 b, p. 517, No. 401, p. 1209 and No. 454, p. 121.)

Fig. 735. *Quetelet's and Bellefroid's Case.* This case was communicated to Quetelet by Dr Bellefroid. Quetelet has compared the measurements with those of the celebrated dwarfs, Tom Thumb and Admiral Tromp: see Table, p. 519. I. 2, normal, of medium height, married twice, by her first husband, I. 1, she had two normal sons, II. 1—2; by her second marriage, with I. 3, she had three children, II. 3—5, of whom two, II. 3—4, were dwarfs, and the third, II. 5, was too young to know whether she would be a dwarf or not. II. 3, aged $11\frac{1}{2}$, was born 1838, at the village of Kerkum near Binckum in the neighbourhood of Tirlemont. He was born at term, and stopped growing at age of $2\frac{1}{2}$ years, and had developed little since then. He never had any serious illness but frequently had colds. He appeared well built with limbs normally proportioned to size of body, the head alone was a little large. He was restless with sharp eyes and rather sallow complexion. II. 4, was born five years later than II. 3. II. 5, aged 17 months, appeared to be perfectly formed, but as her brothers did not stop growing till 3, a decided opinion with regard to her normality could not be formed. (Bibl. No. 99, p. 344.)

	Tom Thumb	Admiral Tromp	Kerkum Dwarf	Normal Child
Measurements:				
Age	11 years (14 years?)	11 years	11½ years	From 13—14 months
Total height	71·0 cm.	72·8 cm.	78·6 cm.	71·0 cm.
Span	66·0 „	70·4 „	62·0 „	69·8 „
Height of head	15·3 „	14·8 „	19·0 „	15·6 „
Circumference of head over eyebrows ...	44·2 „	50·5 „	49·0 „	43·8 „
From crown of head to clavicles ...	17·5 „	18·0 „	19·8 „	16·8 „
Distance of shoulders between the acromion processes	20·2 „	19·5 „	18·0 „	17·4 „
Circumference of shoulders at the acromion processes	50·0 „	48·5 „	45·0 „	43·0 „
Circumference of hips	47·8 „	52·0 „	50·0 „	44·7 „
Length of upper limb from the acromion process	24·5 „	27·2 „	34·3 „	28·4 „
Length of hand	7·5 „	8·8 „	8·6 „	8·5 „
Length of foot	10·5 „	10·5 „	11·0 „	11·1 „
Breadth of hand	4·4 „	4·7 „	5·2 „	4·3 „
Breadth of foot	4·2 „	4·4 „	6·3 „	4·6 „
Length of the lower limb from the patella	17·5 „	17·8 „	20·3 „	17·3 „
Length of the lower limb from the bifurcation to ground	26·5 „	27·4 „	31·7 „	25·4 „
Length of the lower limb from the trochanter to ground	30·0 „	29·6 „	36·0 „	30·0 „
Circumference of calf	15·7 „	16·8 „	17·0 „	15·2 „
Length of ear	4·7 „	4·7 „	5·2 „	4·5 „

Fig. 736. *Manouvrier's Case I.* I. 1, was robust, well formed and non-alcoholic. I. 2, was small but well formed. The account does not state how many other children she had. II. 1, aged 23, Auguste Tuaillon, was born in the village of Esmoulières, Canton de Faucogney (Haute-Saone), March 28, 1873. His mother was never ill during her pregnancy with him. He was born at term, was in good health and of normal size at birth. He walked at age of 13 months, at age of 1½ years he had small white pimples over his hands and feet so thick one could not put the head of a pin between them and the nails on his hands and feet fell off (Cheiropompholyx). At 3, he fell down a flight of stairs from the first story to the ground floor, but apparently was not injured. His mother noticed that he stopped growing at the age of 4½, and then had "le carreau" *i.e.* a very large and very hard abdomen and was much constipated. Manouvrier calls this illness "intestinal tuberculosis." He went to the primary school regularly and got his certificate for reading, writing, elementary arithmetic, the decimal system and elementary history and geography. At the age of 20 he was called as a conscript, his height was then 97 cm. and weight 17 kilos. In a later paper Manouvrier stated his height at this time was 99·7 cm.—there is apparently some discrepancy. His general formation resembled that of a child. He had a large skull and a small face; his limbs were thin and short relatively to the trunk, the abdomen was prominent and the skin without hair everywhere, even the pubes. At a distance he looked like a child, but when close wrinkles were visible. No part of his body was deformed, if the weakness and smallness of his muscular system be left out of account. The hands, feet and ears were normal in form. He had no wisdom teeth, the size of his teeth was proportional to the gums. He walked easily and could walk several kilometres without fatigue, but walked with legs rather far apart, unless he was thinking about it and then he walked correctly. He had no trace of rickets. The fingers were in normal proportion to the size of the hands. *Measurements.* Sitting height 56·8 cm. Bi-acromial breadth 21·4 cm. Bi-trochanteric breadth 19 cm. Length of ear 5·8 cm. Width of ear 3·2 cm. Head: maximum antero-posterior diameter 178 mm. Metopic antero-posterior diameter 178 mm. Maximum transverse diameter 148 mm. Vertical super-auricular diameter 127 mm. Minimum frontal diameter 102 mm. Horizontal circumference 53 cm. Manouvrier saw Tuaillon again at the age of 24½ and said his height was then 103 cm. (Bibl. No. 324, p. 265, Bibl. No. 330, p. 655.)

Fig. 737. *Manouvrier's Case II.* I. 1 and I. 2, were normal. II. 1, aged 23, was their first child born at term, her weight was normal, viz. 3·70 kilos, the head and fontanelles were also normal

at birth. The mother had noticed retardation in growth since birth. II. 1, at 23, was 124 cm. in height, her proportions were regular, she had large breasts and apparently was a fully developed woman, except for a pubes without hair, but her mother I. 2, whom she closely resembled, differed little from her in that respect. Her head was very large relatively to her body and her intelligence was normal. She was known as "Princess Blanche." *Measurements.* Height above ground of crown of head 124 cm.; point of chin 106·7 cm.; of jugular notch 100·6 cm.; of nipple 85·3 cm.; of umbilicus 75·1 cm.; of upper margin of symphysis pubis 63·9 cm.; of antero-superior iliac spine 69·0 cm. Sitting height 64·3 cm. Limbs: height above ground of external extremity of acromion 100·6 cm.; of elbow joint 77·2 cm.; of styloid process of radius 63·0 cm.; of distal extremity of middle finger 47·8 cm.; upper edge of great trochanter 65·0 cm.; of knee-joint 33·1 cm.; of apex of external malleolus 3·7 cm. Breadths: Bi-acromial 25 cm. Bi-humeral 27·1 cm. Bi-nipple 16·6 cm. Minimum waist 19·1 cm. Internal bi-iliac (iliac spines) 20·8 cm. External bi-iliac (iliac crests) 27·4 cm. Maximum bi-trochanteric 27·4 cm. Hand: total length 13·8 cm. Maximum width (heads of metacarpals) 6·25 cm. Free length of middle finger 6·05 cm. The length of the index finger differed little from that of the middle finger, but was 10 mm. longer than the ring finger which appeared to be shortened as was also the little finger. Foot: total length 18·1 cm. Post malleolar length 4·35 cm. Maximum width (heads of metatarsals) 6·5 cm. The great toe was the longest, the others decreased in size regularly. The form of the foot was normal. Circumferences: neck 24·7 cm.; thorax above breast 64·6 cm.; waist 54·5 cm.; thigh at the gluteal fold 36·2 cm.; thigh above the knee 26·6 cm.; calf maximum 22·2 cm.; supra-malleolar 15·8 cm.; middle part of upper arm 16·4 cm.; maximum of forearm 16·8 cm.; minimum of forearm at wrist 11·2 cm. Head: maximum antero-posterior diameter 16·55 cm.; metopic antero-posterior diameter 16·6 cm.; maximum transverse diameter 14·45 cm.; maximum vertical diameter 11·5 cm.; minimum frontal diameter 9·0 cm. Cephalic index 87·3; vertical index 74·2. Face: bi-zygomatic width 10·4 cm.; internal bi-ocular width 2·7 cm.; external bi-ocular width 8·6 cm. Forehead (hair to eyebrows) 4·2 cm. Height of nose 3·65 cm.; width of nose 2·8 cm. Mouth (oral cleft) 3·7 cm.; length of upper lip 1·8 cm., of lower lip 1·3 cm.; bi-labial mucus 1·2 cm. Length of ear 4·95 cm.; width of ear 3·35 cm. (Bibl. No. 330, p. 659 and No. 345, p. 111.)

Fig. 738. *Chavlovsky's Case.* In the discussion on Manouvrier's paper on Auguste Tuaillon, M. Th. Volkov mentioned some Russian dwarfs, among them a pair of dwarfs exhibited before the Society of Anthropology at St Petersburg by Prof. Chavlovsky. No date was given. Nothing was said about the parents, I. 1 and I. 2, except that they were normal. II. 1, aged 26, was normal until the age of 6, but his growth stopped (?) after an injury to his head. His height was 104 cm. II. 2, his sister, aged 18, was 96 cm. in height, and had been very small from birth. Both were developed in good proportion, and all their physiological and psychological faculties were completely normal. (Bibl. No. 324, p. 288.)

Fig. 739. *Volkov's Case I.* Practically no details are given of these dwarfs. Nothing is stated with regard to their parents. II. 2, Serge Bachirov, aged 34, was from the province of Riazan and was 74 cm. in height. His development was quite normal, he was a petty clerk and had married another dwarf, II. 3, aged 23 and 70 cm. in height, who was daughter of a noble landowner. She was also completely developed, clever, and a good housekeeper and worker. (Bibl. No. 324, p. 288.)

Fig. 740. *Ranke and Voit's Case.* This is an account, in 1884, of Frank Flynn (General Mite) and Millie Edwards. I. 1 and I. 2, were healthy and of average size. I. 2 was aged 17 when II. 1, her first child was born. She afterwards had five normal children, II. 2—6, of whom the eldest, II. 2, was aged 14, and II. 6 was born October, 1883. II. 1, aged 16, according to his father weighed 2 lbs. at birth and his mother nursed him for 18 months. At 15 months old he began to walk and at 2 years to talk. His father said that in September, 1876, he measured 62·23 cm. (24½″) and weighed 4812 gms. (10½ lbs.). He could neither read nor write, except his name, but he had a good memory and was what Americans call "smart." He had bad teeth and no pubic hair. He had inflammation of the lungs twice and in 1883 he had whooping cough. *Measurements.* Total height 82·4 cm. Height when sitting 45·2 cm. Height of trunk to 7th cervical vertebra 31·5 cm. Breadth of shoulders from acromion to acromion 17·5 cm.; of hips 13·3 cm. Length of head 145 mm. Breadth of head 115 mm.; face (Jochbreite) 100 mm. Height from upper edge of auditory meatus to crown of head 93 mm. Maximum circumference of skull 42 cm.; chest in inspiration 48 cm. Minimum circumference of chest in expiration 46 cm. Total length of upper limb from acromion to tips of fingers 32 cm. Length of the upper arm 11 cm.; forearm 11 cm.; hand 10 cm. Total length of lower limb measured from trochanter 41·5 cm. Length of thigh 21·5 cm.; leg 15·8 cm. Height of foot from malleolus to sole 4·2 cm. Length of foot 11·5 cm. In 1884, II. 1 married II. 8, Millie Edwards. She was born in the State of Michigan, September 1st, 1871, so was aged 13 in 1884. Her mother, I. 4, aged 36 in 1883, was first married at the age of 19 to I. 5, and had four normal children, II. 15—18. She remained a widow two years and then married I. 3. II. 8, was the first child of this marriage, afterwards I. 4 had a miscarriage in the third month, II. 9, and since that had four healthy normal children, II. 10—13, the youngest, II. 13, was aged 8 months in 1883. II. 8, in October, 1883, measured 72 cm. in her stockinged feet, and weighed with her clothes 6601 gms.

She had bad teeth, the front incisors were almost entirely decayed. She showed no trace of rickets. Her attendant stated she had no pubic hair. No measurements are given. Gilford (Bibl. No. 664, pp. 578—9) gives instructive pictures of General Mite and Millie Edwards. (Bibl. No. 216, p. 229.)

Fig. 741. *Löhlein's Case.* No statement is made with regard to I. 1. I. 2, was a small dwarf person 136 cm. in height, of regular build and with slightly curved extremities. She was healthy in youth, but much addicted to self-abuse; had done much hard work. At the age of 29, in the 7th month of her pregnancy, she had a sanguinolent foetus. She came into the Institution again for a second confinement. The following measurements were taken at the second reception :—External conjugate 160 mm. Diagonal conjugate 85 mm. Interspinous 180 mm. Intercristal 210 mm. Intertrochanteric 245 mm. External oblique diameter 170 mm. At this second confinement she bore a female 47 cm. long who weighed 1835 gms. and died during birth. The mother recovered and left in 10 days. (Bibl. No. 163, p. 41.)

Fig. 742. *Levi's Case I.* II. 5, Santo Magro, aged 49, was born at Randazzo, Sicily, near Etna. His height was 106 cm. Weight 23·5 kilos. He and his son, III. 1, were exhibited in Paris, 1909. His parents, I. 1 and I. 2, were normal and died of old age. I. 1, was neither alcoholic nor syphilitic. I. 2, had four normal children, II. 1—4, two died when adult of acute diseases, two were still alive. No case of dwarfism was known in the family. II. 5, born at term, was extremely small at birth, he was breast-fed and his physical development was normal. He was lively and intelligent but uneducated. His sexual development was normal. At the age of 14 he had hair on pubes and axillae, at 16 had sexual intercourse and at 20 married a normal woman, II. 6, aged then 18. She had died two years ago of pneumonia. After 18 months of married life a boy, III. 1, was born, who was extremely small at birth; two years later a girl, III. 2, was born even smaller than her brother, but like him normal except for size. She died, aged 10, of croupy pneumonia. For about 15 years II. 5, had been inclined to drink too much. His body was well proportioned, the cranium too large relatively to the body, and the last phalanges of the fingers were deformed. There was no trace of rickets, the genital organs were those of a normal adult, the muscular system was perfect. The nose was depressed. He had bi-lateral inguinal hernia. A long description of him is given, but there appears to be no abnormality except his size and the malformation of the last phalanges of his fingers. The measurements were :—Thoracic circumference at nipples 68 cm. Abdominal circumference at umbilicus 69 cm. Length of sternum 12 cm. Distance from the superior limit of the pubis to the ground 47 cm.; antero-superior iliac spine to the ground 48 cm.; great trochanter to the articular line of the knee 20 cm.; this point to the external malleolus 24 cm. Maximum length of foot 15 cm. Total length of upper limb from acromion to tip of medius 39 cm. Distance from acromion to tip of olecranon 15·5 cm.; olecranon to radial styloid process 16 cm. Maximum length of hand 11 cm. Maximum circumference of cranium 50 cm. Bi-temporal diameter 140 mm. Maximum bi-parietal diameter 152 mm. Bi-zygomatic diameter 139 mm. Cephalic index 90. III. 1, Giuseppe Magro, aged 12½, height 77 cm., weight 9·1 kilos, was extremely small at birth. He was breast-fed; his development was regular and gradual and always in proportion to his small size. He had no education but was intelligent. He had no sign of puberty and was perfectly proportioned. Penis that of a child of 4, he had bi-lateral cryptorchism. There was no psychical deficiency and nothing abnormal was shown by radiographic examination. There is a long description of him. The measurements were :—Height of pubis from ground 35 cm. Maximum thoracic circumference 45 cm. Maximum abdominal circumference 42 cm. Length of sternum 8 cm.; from tip of acromion to tip of medius 31 cm.; from acromion to tip of olecranon 12·5 cm.; from olecranon to radial styloid process 11 cm. Maximum length of hand 7·5 cm. Length of lower limb from antero-superior iliac spine to heel 38 cm.; from great trochanter to articular line of knee 17 cm.; from this point to external malleolus 17·5 cm. Maximum length of foot 11·5 cm. Maximum circumference of skull 46 cm. Bi-parietal diameter 143 mm. Bi-temporal diameter 143 mm. Bi-zygomatic diameter 134 mm. Cephalic index 91. (Bibl. No. 640, p. 542.)

Fig. 743. *H. D. Smith's Case.* II. 1 and II. 2, both normal, had four children, all of whom were normal, as were also their descendants except III. 7, a dwarf, type uncertain, who married a normal woman, II. 8, and had 14 children, IV. 3—4, of whom only one, IV. 4, was a girl; no note is made of their size, so presumably they were normal. II. 3, sister of II. 2, had at least three sons and three daughters, all rather tall. One son, III. 9, married III. 10, a normal woman, and had several children, IV. 5—6, of whom IV. 5 was an ateleiotic dwarf, a medical man of ability and character (unpublished).

Fig. 744. *Benzenger's Case.* The Kostesky Family. II. 1, was tall and strong; he died, aged 44, of "fièvre chaude." His wife, II. 2, aged 80, was alive. It is stated that the grandfather of III. 8 was tall and strong, but does not specify which grandfather. It also states III. 8 remembered three of his six uncles, but again does not specify whether they were maternal or paternal uncles, therefore they have not been entered in the pedigree. II. 1 and II. 2, had seven children. III. 1—4, were living, all tall, and their children, IV. 1, were of medium height. III. 5—6, were also tall and alive, III. 6 had 11 children, IV. 2, all tall. III. 8, aged 45, height 1·79 metres, was robust, healthy and of great muscular power. He was born at Kanef, province of Kiev, had, according to his mother, cholera at 8 years of age, of which he

nearly died, and had no serious illness since, but suffered from a species of aphasia the result of a fall on the ice two months before. His wife, III. 9, was not related to him, she was of medium height, 1·54 metres. Height of her father, II. 4, was 1·55 metres. Her mother, II. 5, was tall, and according to her the grandparents, I. 1, I. 2, I. 3 and I. 4, were healthy and of average height. II. 4 and II. 5, had had eight children, III. 9—16, of whom only two were alive. No dwarf had ever been known in the family. III. 8 and III. 9, had nine children, IV. 3—11, all breast-fed. IV. 3 and IV. 10—11, died in their first year. IV. 8, a girl, aged 6 years and 10 months, was normal, her height was 1·16 metres. IV. 9, aged 4 years and 2 months, could not be called a dwarf. Her height was 1 metre and she was still growing. IV. 4—7, were dwarfs. IV. 4, was aged $16\frac{1}{2}$, height 97·1 cm.; IV. 5, aged 14, height 102 cm.; IV. 6, aged 11 years and 10 months, height 95·5 cm.; IV. 7, aged 9, height 92 cm. All these children had ceased to grow in their 4th year, all were of the dwarf type, had large heads, large abdomens, short limbs, flaccid muscles, and were prematurely aged with some facial wrinkles. IV. 4, had some grey hairs. Their skin was fresh and rosy, their hair dark chestnut and eyes light chestnut. Their normal sister, IV. 8, was blonde with light eyes. The intellectual abilities of these dwarfs were remarkable. They had never attended school, but could read and write Russian and read Slavonic. They knew sacred history and a number of prayers, psalms, fables and songs. IV. 7, was an agile dancer, and the girls, IV. 5—6, excelled in manual work, sewing and embroidery. They had also learnt French and German, the violin and dancing. IV. 4, was the most apathetic, but at the age of 10 or 12 he had been as lively as IV. 7. They appeared to have no wills of their own. The original account of this family was published by Dr Benzenger at Moscow, but he had lost sight of them for more than a year. M. Th. Volkov, in the discussion on Manouvrier's paper, Bibl. No. 324, mentioned the family Kostesky, and said they formed a troupe of dramatic artists who gave representations in all the towns of S. Russia. He gave no family details, but gave the heights and ages of the four dwarfs, and evidently either he or Brongiart has made mistakes with regard to height. Judging from the ages given, Volkov's measurements were made about 10 years later than Brongiart's.

	Brongiart		Volkov	
	Age	Height	Age	Height
IV. 4	$16\frac{1}{2}$	97·1 cm.	28	90 cm.
IV. 5	14	102 „	25	92 „
IV. 6	$11\frac{10}{12}$	95·5 „	20	89 „
IV. 7	9	92 „	19	90 „

For portraits of these dwarfs see our Plate KK (75). (Bibl. No. 232, p. 179, and Bibl. No. 324, p. 288.)

Fig. 745. *Geoffroi's Case.* No statement is made with regard to I. 1 or I. 2, except that I. 2 was aged 35 and had three children, II. 1—3, of whom II. 1, Nicolas Ferry, otherwise Bébé, was the eldest. When born he was about 8″ or 9″ (pouces) long and weighed 12 ounces (12 onces) or $\frac{3}{4}$ lb. (3 quarterons). He was born Nov. 13, 1741, after a labour which lasted for 48 hours. On July 25th, 1746, he was carefully measured by M. Kast, physician to the Queen of Poland. He was then made in miniature like a man of 20, which made M. Kast conjecture he would grow no more. He was 22″ (22 pouces) long and weighed when stripped 9 lbs. 7 oz. (9 livres 7 onces). All parts of his body were well proportioned, he had a pretty face, nose well made and aquiline, dark brown eyes and fair hair. He had had small-pox at 3 years of age. He was extraordinarily vivacious, never still for a moment, feared nothing and could never be dissuaded from the object he had in view. He appears to have had some memory, but not as much as a normal child of his age. His voice was the voice of a child a year old. His knees, especially the R. one, were bent out a little (genu varum), which diminished his height by $\frac{1}{2}$″ ($\frac{1}{2}$ pouce). Manouvrier (*Mémoires de la Soc. d'Anth. de Paris*, T. 4, pp. 347—402) gives the following measurements for Bébé, whose skeleton is in the Musée d'Histoire Naturelle in Paris: see our Plates Z (39—40) and RR (98):—Height 100 cm. Length of femur 24·52 cm.; tibia 17·61 cm.; humerus 20·38 cm.; radius 12·17 cm. Buffon, Bibl. No. 33, gives a very complete table of measurements. (Bibl. No. 20, p. 44.)

Fig. 746. *Taruffi's Case I.* Taruffi says he does not know where the original of this case is to be found. No statement is made with regard to I. 1 and I. 2. II. 2, had several normal brothers and sisters, II. 1. II. 2, Caterina Pospoel, was born 1820 at Isembeck, in the neighbourhood of Brussels. She always had good health. At the age of 33 she was 91·8 cm. in height with limbs in proportion. She was thin and looked like a miniature woman of 45. She was of a bright disposition with well developed intelligence and busied herself in house and field work. (Bibl. No. 248, p. 442.)

SECTION III. DWARFS OF UNCERTAIN TYPE.

THE information given about the dwarfs in Figs. 747—841 is, in most cases, insufficient to permit of any attempt at definite classification under achondroplasia, ateleiosis, or other type. In some cases where the statures or other measurements are given, a fair guess at the nature of the dwarfism may be made.

PLATE LVI. Fig. 747. *Luigi Frank's Case.* This is the case of the Leporati family. I. 1, and I. 2, were people of tall stature, natives of Varano de' Melegari, a mountain about 24 "miglia" distant from Parma, they had five children, II. 1—5, all of whom were tall except II. 5. All their relatives were also tall. II. 5, Francesco Leporati, was born in 1730, and was a dwarf well proportioned in all parts of his body. His height was 113 cm. In his 19th year he went as page to Parma and learnt to be a watchmaker. At the age of 36 he married a normal woman, II. 6, and they had nine children, of these, three, two girls and a boy, III. 3—5, died in infancy. There were also two miscarriages, III. 1—2. II. 5, died aged 83. Of the six surviving children five were dwarfs. III. 6, aged 50, was 113 cm. in height. She had been a nun, but had left the convent. III. 8, aged 44, height 135 cm., was a watchmaker. He married twice, by his first wife, III. 7, he had four children, IV. 1—4. IV. 1—3, died young, IV. 4, who survived, was fairly tall and married. By his second wife, III. 9, aged 33 and also normal, he had four sons, IV. 6—9. IV. 6, died young. The other three were young but apparently dwarfs. IV. 7, aged 14, height 94·5 cm.; IV. 8, aged 9, height 97 cm.; IV. 9, aged 7, height 91 cm.; III. 10, aged 42, height 130 cm., was also a watchmaker, he married III. 11, aged 32, a normal woman, and had five children, IV. 10—14. IV. 10, died young. IV. 11, aged 13, was normal. IV. 12, aged 6, height 82·5 cm. IV. 13, aged 3, height 65 cm., and IV. 14, aged $1\frac{1}{2}$, height 61·6 cm. were said to be dwarfs. Frank says IV. 14 looked as if he would be a dwarf. III. 12, aged 41, was 98 cm. in height. III. 13, aged 34, was normal and married. III. 15, aged 31, was 115·5 cm. in height. Scarcely ateleiosis. (Bibl. No. 67, p. xcvii.)

Fig. 748. *Mussot Arnould's Case.* It is stated that this family appeared at a fair in Saint Germain, 1779. They were Lapps. I. 1, aged 30, was only 31″ (31 pouces) high (0·97 m.). I. 2, was 28″ (28 pouces) high (0·756 m.). They had been married in France and their child, II. 1, was only 18″ (18 pouces) high (0·446 m.). They were well made and of interesting countenances, speaking French well enough to answer questions. Quoted by Garnier, not seen in original. Possibly ateleiosis. (Bibl. No. 35, and No. 205, p. 187.)

Fig. 749. *Taruffi's Case II.* I. 1, and I. 2, were agricultural labourers in the province of Cuneo, of a good height. Taruffi says they had 13 children, seven sons and four daughters. There is evidently a mistake here of some kind, so only the eleven children have been entered. All of them attained normal height except the third and seventh sons. These were unable to follow the occupation of their parents, the third, II. 3, became a tailor and the seventh, II. 7, aged 36, travelled round with a showman. He was born at term and at age of 31 married a woman of normal stature, II. 8, and had two children, III. 1—2, who both died before they were 12 months old. He was well formed and had no trace of rickets, but his head was very large in proportion to his stature. He had a dolichocephalic skull. *Measurements.* Height 115·5 cm. Circumference of head 52 cm. Antero-posterior diameter of head 18 cm. Maximum transverse diameter of head 14·4 cm. Vertical diameter from the auditory passage to the vertex 11·1 cm. Cephalic index 80. Face from the glabella to the chin 10·5 cm.; whole height of face 17·4 cm. Length of hand 12·0 cm. (Bibl. No. 248, p. 452.)

Fig. 750. *Taruffi's Case III.* No statement is made with regard to I. 1. I. 2, aged 35, had had four confinements, II. 1—4. After the fourth, her hair fell out, she had a cutaneous eruption of her hands and arms and afterwards suffered several times from affections of the mouth and throat. These symptoms were considered syphilitic. When she recovered she became again pregnant, and during pregnancy was subject to nocturnal pains in the lumbar and crural regions so went to the Maternity Hospital in Bologna in 1884. Of her children II. 1, and II. 3, appeared healthy and robust. II. 2, showed no external defect but would not take the mother's breast and died of hunger on the fifth day. II. 4, was born dead and emaciated. II. 5, came at term like the others and was born without trouble but died half hour later. The head measurements were: Occipito-mental diameter 10 cm.; occipito-frontal diameter 10 cm.; bi-parietal diameter 9 cm.; bi-temporal diameter 7 cm.; sub-occipito-bregmatic diameter 9 cm.; fronto-mental diameter 8 cm.; total length 38 cm.; weight 2000 grammes. The cranial vault showed no defect but had a fine down on the skin, the face had no eyebrows, the nose was depressed at the root and a little flattened at the point. The philtrum of the upper lip was wanting in its middle and lower part, and the lower lip was not in the same plane as the upper because the mandible was shorter than usual. The edge of the lip did not turn over because the mucous membrane of the floor of the mouth extended to the alveolar margin and adhered to the middle part of the edge of the lip. The parotid and masseteric regions were

very prominent. The hard palate was cleft and the alveolar arches very deficient. The upper limbs were relatively short, the L. measured 9 cm., the R. 8 cm., and the humeral part was longer than the radial, the joints were not very mobile though the epiphyses were not enlarged. The forearms were held at R. angles to the upper arms. The L. hand had seven fingers spread out like a fan and none of them resembled a thumb or little finger. The R. hand was rather narrower and divided by a longitudinal sulcus; it had only two fingers which were relatively rather long. The lower limbs were thick and deformed, the knees bent at a right angle so that they seemed shorter than they were. The thick thighs were much abducted, the legs on the contrary were adducted and twisted in such a manner that the fibulae were found behind. The feet were in the equino-varus position, the toes webbed to the middle, the R. foot had six toes the L. five with a space of 4 mm. between the third and fourth. There were several deformities in the internal organs. There was slight scoliosis of the vertebral column with dorsal convexity to the L. and lumbar to the R. The pelvis was symmetrical. The humeri measured 45 mm. The right forearm had only a radius which resembled the letter S, in the L. both bones were present. The femora measured 47 mm., the tibiae 32 mm. The legs had the normal number of bones but the fibulae were still in a cartilaginous condition. Possibly achondroplasia with multiple abnormalities. (Bibl. No. 209, p. 663.)

Fig. 751. *Bayon's Case I.* I. 1, was abnormally small and mentally defective but no measurements of him are given. He was a shoemaker in Laudenbach, near Karlstadt. No statement is made with regard to I. 2, neither is total number of offspring stated. II. 1, was well formed. II. 2, was even smaller than II. 4, and was married. II. 4, was brought to hospital in 1888, she had always been weak-minded but was showing signs of increasing mental defectiveness. She had never worked but supported herself by begging. She died in 1888 aged about 66. Bayon says this was certainly not a case of cretinism, he calls it rickets. *Measurements of* II. 4. Total height 120 cm. Span 125 cm. Humerus 23 cm. Radius 16 cm. Femur 26 cm. Tibia 22·5 cm. (Bibl. No. 436, p. 46.)

Fig. 752. *Dufour's Case.* I. 1, died of tuberculosis. I. 2, of heart disease and hemiplegia, both were normally formed. Of their five children, the three elder, II. 1—3, and the youngest, II. 5, were normal. II. 4, aged $14\frac{1}{2}$, was born at 8 months. At birth it was noticed that her hands and feet were badly formed. When four years old her small size was noticeable. When seen in 1906 she had a relatively big head, the cranium being very large; the lower limbs were too short. She had very pronounced lumbar curvature and prominent abdomen. Her muscles were on the whole well developed. When the arms were extended by the sides the extremities of the fingers passed slightly beyond the limit which separates the upper third from the middle third of the thigh. She was merry but backward, could read and write but her intelligence was equal to that of a child aged 7. Possibly achondroplasia. *Measurements.* Height 124 cm. Trunk, from episternal notch to symphysis pubis upper border 52 cm. Total length of upper limb 52 cm. Upper arm 20 cm.; forearm 18 cm.; hand 14 cm. Total length of lower limb 62 cm. Thigh 33·5 cm.; leg 28·5 cm. The fourth finger of each hand was almost as short as the fifth. Of the forearm the radius alone was incurved. (Bibl. No. 519, p. 133.)

Fig. 753. *Morse's Case.* I. 1, and I. 2, were Italians who were healthy and of fairly good habits. There was no history of any similar deformities and no miscarriages. Their first child, II. 1, weighed 12 lbs. and was born dead after severe instrumental delivery. II. 2, was born at full term and except for deformities seemed normal. He was brought to the hospital for inguinal hernia. The limbs were very short, especially the legs. The bridge of the nose was depressed. He had slight exophthalmus. No abnormality of the thyroid gland could be made out. The humerus felt $2\frac{1}{2}$ cm. thick at least, the rotation of the radii was normal. The thighs and legs were held in almost the position of a circle. He died of some enteric disease aged 4 months and no autopsy was obtained. Measurements are given, but the description of them is vague in several cases. (Bibl. No. 416, p. 561.)

Fig. 754. *Heiman's Case.* There was no history of dwarfism in this family. The parents, I. 1, and I. 2, were of at least average intelligence and physique. Of their two children, II. 1 was of normal growth. II. 2, was brought to hospital at the age of 6 months for penile hypoplasia. He was well nourished but had an almost imbecile expression; he seemed, however, able to see and hear. The head was about normal in size, the anterior fontanelle patent, the posterior closed. The hair was thin, the ears deformed, the nose short, flat, and *retroussé*, with broad and depressed bridge. The gums were enormously hypertrophied and irregularly thickened; two months later, *i.e.* at the age of 8 months, teeth began to erupt. The hard palate was high-arched, the soft palate thickened, the neck short and the thyroid gland not palpable. The abdomen was protuberant. The long bones of the extremities were short, curved and thickened. The hands were squat and showed the trident deformity. The fingers were nearly all of the same length. The third toe on each foot was deformed and without a nail. Most of the nails were ill-developed. *Measurements.* Length of body 56 cm. Vertex to umbilicus 30 cm. Umbilicus to sole of foot 26·5 cm. From acromial angle to distal extremity of middle finger 19 cm. Upper arms 8 cm.; forearms 5·5 cm.; hands 5·7 cm.[1] Antero-superior iliac spine to internal malleolus 21 cm. Length of

[1] No points of measurement stated for "arms."

thigh from great trochanter to "knee" 11·5 cm.; from "knee" to internal malleolus 9 cm. Circumference of neck 20 cm.; chest 38·5 cm.; abdomen 40·5 cm. Sub-occipito-bregmatic diameter of skull 12·6 cm.; sub-occipito-frontal ditto 13·6 cm.; occipito-frontal ditto 14 cm.; occipito-mental ditto 14·5 cm.; bi-parietal ditto 11 cm.; bi-malar ditto 9·15 cm.; occipito-frontal circumference of skull 41 cm. (Bibl. No. 487, p. 842.)

Fig. 755. *Chambrelent's Case.* There is very little said about this case. During a discussion on Auché's case, M. Chambrelent stated he knew a young achondroplasic woman, II. 2, whose twin sister, II. 3, was also achondroplasic. The same case is said to be reported in the *Journal de Médecine de Bordeaux*, 1906, Année 36, p. 67, with the addition that a third sister, II. 1, was also achondroplasic. Nothing is said of the parents. (Bibl. No. 517, p. 117.)

Fig. 756. *Dupuytren's Case.* I. 1, and I. 2, were healthy and of average height. They had five children, II. 1—5. Two boys, II. 1—2, born before II. 5, died, II. 1, aged 1½, II. 2, aged 8 months, both of normal size for their years. II. 3—4, were healthy with a development superior to that of most children of their age. II. 5, aged 26 months, when born was only the size of a foetus of 5 months, but his development was that of an ordinary child at birth, there was nothing remarkable about him except the extreme smallness of his body especially the head. He was breast-fed and at the end of a year had only grown a few inches, and his weight had increased just in proportion to the increase in his size. When seen by Dupuytren at the age of 18 months, he had very long hair, and a very small skull with the fontanelles closed. Proportionately to the skull the face was much developed, the neck was thin, the chest and abdomen of moderate size, the lower limbs longer than is usual at this age. He was merry and restless and enjoyed good health. When seen again 8 months later his weight was 6 lbs. 2 oz. (6 livres 2 onces) and height 1′ 5″ (1 pied 5 pouces). The eyes were very small, the senses well developed, the sense of smell, however, least so. He did not walk or speak any word distinctly. The intellectual faculties were fairly well developed and apparently he could combine ideas as he acted in a way which presupposed reasoning. Perhaps ateleiosis, possibly microcephalic dwarfism. (Bibl. No. 54, p. 146.)

Fig. 757. *von Franqué's Case II.* No statement is made with regard to I. 1, and I. 2. II. 2, aged 28, was a weakly anaemic person of dwarfish stature. Height 44″ 8‴. She said she had learnt to walk in her third year. She suffered from violent pains in the lumbar vertebrae and sacral region. The vertebral column was scoliotic, there was a great curve to the L. in the upper and middle dorsal region and in the lumbar region a lesser deviation to the R., which ended in the sacrum. The conjugata of the pelvis was 3″ 3‴ so premature delivery was brought on. The child was very small, weighed "2 Pfund 13 Loth" and died three days later. The mother recovered. Possibly riekets. (Bibl. No. 141, p. 120.)

Fig. 758. *Michel's Case.* I. 1, was epileptic. I. 2, was healthy and in 12 years had borne six girls, II. 1—6, all of whom were alive but weakly and suffering from slight rachitis and scrofula. Syphilis was out of the question. II. 7, a boy, was stillborn. He had a small body, very short limbs, and a head like a ball covered with long brown hair. The neck was short and swollen, the abdomen prominent, the arms thick and swollen and flexed at the elbows. The metacarpus was thick and the fingers badly formed. The legs, which were in varus position with the soles of the feet turned up from behind, were like sausages, with no sign of a knee. The feet were thick but well formed. *Measurements.* Length of body 37 cm.; trunk 29 cm. Distance of the neck from the umbilicus 14 cm.; navel from the symphysis 3 cm. Circumference of head 29 cm.; chest 21 cm.; pelvis 19 cm. Length of the arms from the axillae 9·5 cm.; distance from the axillae to the elbows 3 cm.; elbows to the wrist 3·5 cm.; wrist to the tips of the fingers 3 cm. Circumference of upper arm 8 cm.; of forearm in upper part 7·3 cm., in lower part 7 cm. Length of the legs from the groin to the tips of the toes 9 cm.; distance from the groin to the ankle 6 cm.; to the knee 4 cm.; knee to the ankle 2 cm. Circumference of the thigh 11·5 cm.; of knee 8 cm.; of leg in the upper part 6·5 cm.; of lower part 5·5 cm. Length of great toe 1·1 cm.; L. humerus 3·7 cm.; L. ulna 3·5 cm.; L. radius 2·7 cm.; L. femur 4·5 cm.; L. tibia 3·1 cm.; L. fibula 2·5 cm. The diaphysis of the humerus was much curved the convexity being to the front; it was fractured lengthways and transversely. The femur was bent at an obtuse angle in the middle, the convexity being behind, it was fractured at the bend and abnormally flexible. (Bibl. No. 449, p. 1.)

Fig. 759. *Johannessen's Case.* I. 2, had been a driver three years before, and had then drunk somewhat. He still took a glass now and then but was "nie bei Zechgelegen." He had bronchitis about three years before, and afterwards coughed more or less. Some years later he had inflammation of the lungs and was ill for three weeks at home and for six weeks in hospital. I. 3, had had bronchitis seven years before and was weak and had little strength. One of her sisters, I. 4, aged 11, died of dropsy, another, I. 5, of inflammation of the stomach. A sister of I. 2, namely I. 1, died of a purulent disease of the lungs. Neither I. 2, nor I. 3, ever had syphilis, and no tuberculosis, insanity, convulsions, haemophilia or struma could be ascertained to exist in the family. I. 3, had had to stand a good deal during her last pregnancy nursing her husband, and had twin girls II. 6—7, who got measles at the age of 5 months

and died. She also had to look after her five eldest children, II. 1—5, who were healthy, and she worked as an ironer as well. II. 8, weighed 3000 grammes at birth with wrappings. Her spine was very long, and she had lumbar dorsal kyphosis. She was taken to hospital when one month old. The pelvis was extremely deformed. She had a peculiar appearance and looked rather like a skinned hare. She died and there was an autopsy. *Measurements.* Circumference of head 34 cm.; bi-parietal diameter 10 cm.; fronto-occipital diameter 11 cm. Chest circumference under axillae 26·5 cm.; over nipples 26 cm.; over the costal arch 26·5 cm. Total length of body 52 cm. Height when sitting 37 cm. Span 45 cm. Length of upper arm from acromion: R. 6 cm.; L. 7 cm.; forearm 6 cm.; hand through third finger 8 cm. Distance between the iliac crests 9 cm.; antero-superior iliac spines 8 cm.; postero-superior iliac spines 2·5 cm.; great trochanters 12 cm.; ischial tuberosities 2·5 cm. Length of thigh from end of trochanter to knee-joint R. 8·5 cm., L. 9 cm. Length from knee-joint to end of internal malleolus 8·5 cm., to end of external malleolus 8 cm. Length of foot from heel to second toe 9·5 cm.; neck from the hyoid bone to upper limit of manubrium 2 cm. Circumference of neck 15 cm. Length of humerus about 6·7 cm.; radius 5·3 cm.; ulna 6 cm.; femur 8·7 cm.; tibia, about 8 cm.; fibula 7 cm. (Bibl. No. 351, p. 351.)

Fig. 760. *Du Plessis' (James Paris) Case.* I. 1, John Grimes, aged 57, height 3′ 8″, was born at Newcastle on Tyne, he married I. 2, a normal woman, and had four children, II. 1—4. He was a short and very thick man, "he was as broad as he was long from hand to hand stretched" (meaning obscure). He sold himself to a surgeon some years before his death for 6*d.* a week—to be dissected after death. Paris gives a water-colour picture of him. He is represented with a stick and a long coat to the knees. His legs look straight, and he does not appear deformed—he has no beard and looks like a boy. Garnier (Bibl. No. 205), however, states that he was deformed and a small Hercules, and Paris that he could between 30 and 40 years easily lift upon his two hands two ordinary men. Paris saw his skeleton after dissection. (Bibl. No. 18[b].)

Fig. 761. *Maygrier's Case.* I. 1, was alive and healthy. I. 2, had been very nervous, subject to convulsive attacks followed by loss of consciousness. She died aged 44 of cerebral haemorrhage. She had 13 children, II. 1—13, of whom four, II. 9—12, were pairs of twins. II. 13, aged 23, primipara came for her confinement, five of her brothers and sisters were alive and healthy, of whom several had healthy children, III. 1. II. 13, had been breast-fed. She was healthy but nervous. She married at age of 21, and for about four months afterwards suffered from bad headaches. Her husband, II. 14, was aged about 35, he was rather delicate and suffered from chronic bronchitis. He had been ten years in the Colonies and had had intermittent fever. He never had syphilis, but drank a good deal, and was of only mediocre intelligence. He could give no particulars of his family, and had the face of a rachitic person. The pregnancy was normal, labour was difficult, instruments were used, and the child died during labour. It was a boy and weighed 2640 grammes, length 38 cm. He had the characteristic appearance of an achondroplasic infant. The trunk was normal, the limbs short, deformed and thick. The skin lay in rolls on the limbs. Head diameters were: occipito-mental 13·5 cm.; occipito-frontal 12·3 cm.; sub-occipito-bregmatic 9·6 cm.; bi-parietal 10 cm.; bi-temporal 8·2 cm. No other measurements are given. (Bibl. No. 346, p. 249.)

Fig. 762. *Boissard's Case.* II. 2, was member of a family which was healthy and rather above the average in height. She had a younger sister, II. 3, who was tall and healthy. She herself had never been ill, but had not walked till she was 3½ years of age. Her height was 114 cm., but she was fairly well proportioned and her whole skeleton appeared reduced. Her head was large with a bulging forehead. The upper and lower limbs were very short, but only slightly incurved. The vertebral column was normal. No measurements are given. She became pregnant; Caesarian section was performed, and a male child extracted who weighed 2760 grammes, and had both feet in equino-varus position. Mother and child lived. (Bibl. No 347, p. 33.)

Fig. 763. *Lecadre's Case.* I. 1, was alive, but no statement is made with regard to his health. I. 2, died of cancer of the womb. II. 3, was of frail constitution, she had had a normal child, a strong and healthy boy[1], five years before marriage. She married at age of 36, and had two miscarriages at four months, neither she nor her husband, II. 4, showed any trace of rickets, syphilis or scrofula. She came to be confined in 1858, then aged 38. The confinement was natural, a female stillborn child, III. 4, being born. The child had a very large head, like a hydrocephalic head with the sutures widely separated, she had dark hair 1·5 cm. in length, a bulging forehead, flat nose, thorax depressed laterally and an enlarged abdomen. The genital organs were normal. The upper arms were represented by two thick and short appendages, 9 cm. in circumference at the middle, the forearms were equally deformed and rudimentary and were flexed on the upper arms. The hands were normal. The lower limbs were also thick and short. The thighs were very short, the legs being represented by two small fleshy stumps, the whole forming an irregular arc with the concavity turned inwards and backwards. The knees were far apart, the soles of the feet turned in, the skin in general thick. The humerus was thick and short, with enlarged epiphyses, it was concave in front and convex behind and was

[1] Erroneously marked on pedigree as offspring of a first *marriage.*

covered like all the bones with a reddish periosteum. The radius was shaped like an S, it and the ulna formed an obtuse angle with the humerus. The fibula was curved similarly to the radius and was about the same length. The hand was bent at a right angle on the ulnar side. The femur was shaped like an hour-glass. Relatively to the coxal bone and the leg, it was directed horizontally outwards and forwards from the cotyloid cavity. The tibia and fibula were also shaped like a double cone or hour-glass, but the curves were less pronounced. *Measurements.* Length from the sinciput to the calcaneum 33 cm. Bi-acromial diameter 9 cm. Bi-iliac diameter 8 cm. Occipito-frontal circumference 33 cm. Mento-bregmatic circumference 33 cm. Sub-occipito-bregmatic circumference 31 cm. Circumference of abdomen at umbilicus 27 cm. Distance of the umbilicus from the pubis 5 cm.; from the xiphoid process 6 cm. Length of the humerus 2·7 cm.; radius 2 cm.; femur 3 cm.; diaphysis of the femur 2 cm. Breadth of the femur at middle point 0·5 cm.; at the extremities 1·4 cm. Length of foot 4·5 cm. Breadth of foot 2·5 cm. (Bibl. No. 108, p. 8.)

Fig. 764. *Townsend's Case.* No statement is made with regard to I. 1 and I. 2. II. 2, aged 32, height 3′ 9″, and with legs and arms much deformed by rickets, came to the hospital for her confinement. She had convulsions during delivery, the cephalotribe was used and the child extracted. The mother died, and there was an autopsy. The antero-posterior diameter of the pelvis only measured $1\frac{9}{16}$″ across, the transverse diameter was $3\frac{1}{2}$″. A large fibrinous polypus occupied almost the whole of the R. auricle, being attached to the heart near the tricuspid valve. (Bibl. No. 166, p. 90[1].)

Fig. 765. *Quatrefages' Case.* "Prince Balthazar or Balthazar Zimmermann." I. 1, and I. 2, were robust and well formed. They had nine children, II. 1—9, eight of whom were normal. II. 9, the dwarf, was born at Glaris in Switzerland. The man who was exhibiting him said he had been of normal proportions at birth, but his growth ceased later, he also said he was aged 16, measured 76 cm. and weighed 9 kilos. Quatrefages was unable to measure or weigh him, but judged from seeing him that the measurements were not far wrong. There appeared to be nothing deformed in his body, the shoulders were broad, the torso rather thick set and the chest well developed. The abdomen was rather over developed. The limbs appeared to be in harmony with the trunk, the hands were plump (potelées), the head much too large for the body, its height being rather more than a fifth of the total height. The forehead was high and arched, the parietal bosses being very pronounced. He had not much hair. The cheeks were large and fleshy, the eyes relatively large and the mouth small. The nose was too little developed being depressed and narrow at the bridge. He did not look like a child. He could read and write German well and knew some Italian. He could sing songs, his voice was thin and rather cracked, but true. Towards his family he was very affectionate. Regnault has classified the case as achondroplasic, but pictures (see our Plate JJ (71[a])) do not wholly confirm this view. (Bibl. No. 187, p. 703.)

Fig. 766. *Stilling's Case.* No statement is made with regard to I. 1 and I. 2. II. 5, was the fifth child of II. 2, the four others, II. 1—4, were normal. II. 5, was stillborn, born without help but so precipitately that the head and neck received injuries. There was dark hair on the head, the skin was pale and very oedematous, forming thick rolls on the limbs, which became circular ridges at the joints. The extremities were remarkably short in proportion to the body, and the abdomen large relative to the extremities. The face was quite normal, as were also the external genital organs. The most remarkable abnormality was the roof of the skull which was almost wholly membranous. When the brain was taken out the soft parts fell together and formed a thick wrinkled membrane which covered the base of the skull. There was slight scoliosis in the upper part of the dorsal spine with convexity to the right, otherwise the spine was normal. The thorax was in general normal and the pelvis was normal. The L. humerus was short and thick with the cartilaginous epiphyses well developed, the radius and ulna were much curved, the curvature being outwards. The R. upper extremity was like the L., except for a fresh fracture in the lower third of the humerus. A fracture was noticeable in the upper third of the L. thigh. The tibia was bent in below the middle, the bend being like an obtuse angle with the opening behind. The fibula was much bent, the R. thigh was broken in the middle. The cartilaginous epiphyses of both femora were large in proportion to the diaphyses, but yet were normal. The diaphysis of the R. fibula was detached from the upper epiphysis, the bone itself showed a worse bend than the L. and the curvature of the R. fibula was greater than the L. *Measurements.* Total length 35 cm. Length of R. arm 7 cm. Length of hand 3·5 cm.; index finger 1·6 cm.; middle finger 1·8 cm. Transverse circumference of hand 3·5 cm. Length of R. lower extremity 8 cm.; foot 4·5 cm.; second toe 1 cm. Transverse circumference of foot 4 cm. Total length of tibia 3 cm. Upper epiphysis of tibia 0·8 cm. Lower epiphysis of tibia 0·6 cm. (Bibl. No. 245, p. 357.)

Fig. 767. *Schieb's Case.* I. 2, looked rather imbecile, she had five children, the first, II. 1, by a different father, suffered immediately after birth from "Augenfluss" ("ein Auge soll ausgelaufen sein"). I. 2, had heartburn (Brennen) then, with incontinence of urine (Wasserlassen) and falling of the vagina.

[1] The reference to this pedigree in Bibl. No. 293 is an error.

Four months after the birth of the first child, she had a miscarriage, II. 2. The account does not state whether the miscarriage was due to pregnancy by I. 1 also. The other children, II. 3—6, by II. 3, came into the world in six years, the children were healthy, had no deformity and learnt to walk soon. At three years of age all had suffered from skin ulcers which soon disappeared. II. 3, the father, was alcoholic and suffered from chest affection. No goitre or deformity was known in the family. II. 7, was being exhibited by his parents in the annual market as a "Maulwurfmensch" and was taken to the hospital. He had been born after a normal pregnancy, and the doctor who had attended gave details. The birth was normal, but the small size, curved limbs, extraordinary growth of hair and almost coal black colour of the skin were remarkable. The mother could not nurse him, not having milk enough. He was extraordinarily small and thin, with dark brown skin over the whole body, nearly black on the forehead and cheeks. There was long black hair on the head, black lanugo over the whole body, especially on the forehead and on the exterior-surfaces of the limbs and back. The head was very large relative to the body and the fontanelles were wide open. The extremities were curved and thickened at the angles. The greatest curvature in the lower extremities was in the middle of the thigh. The testicles and epididymis were greatly developed. The child died in about two months. *Measurements.* Length from vertex to umbilicus 28 cm.; vertex to buttocks 34 cm.; heel to umbilicus 9 cm.; heel to buttocks 8 cm.; the xiphoid process to navel 5 cm. Circumference of chest at the nipples 26·5 cm.; at lower aperture 25 cm. Maximum abdominal circumference 24 cm. Abdominal circumference at umbilicus 22 cm.; at the height of the antero-superior iliac spine 22 cm. Distance from the symphysis pubis to the umbilicus 3 cm. Length of humerus from the great tuberosity to the external condyle 6·5 cm.; radius from the capitellum to the styloid process 6·1 cm.; ulna from the olecranon to the styloid process 6·5 cm.; the hand from the styloid process of the radius to the end of the third phalanx of the index finger 6·2 cm.; femur from tip of great trochanter to the external condyle 8·4 cm.; tibia from the internal condyle to the internal malleolus 6·3 cm.; fibula from the capitulum to the external malleolus 5·8 cm.; foot from the posterior calcaneal process to end of first toe 6·5 cm.; left clavicle 4·1 cm.; right clavicle 3·5 cm. Head: Fronto-occipital diameter (from the glabella to the furthest projecting point of the occiput) 10 cm.; bi-parietal larger transverse diameter 11·5 cm.; mento-occipital largest oblique diameter (from chin to most distant point of the skull on the lower edge of the small fontanelle) 12 cm.; sub-occipito-bregmatic, smaller oblique diameter (from the limit between the occiput and neck to the middle of the large fontanelle) 10 cm.; vertical diameter (from vertex to base of skull) 8 cm. Further measurements of hand, pelvis and foot as well as particulars of the microscopical examination are given. (Bibl. No. 367[b], p. 93.)

Fig. 768. *Temple's Case.* I. 1, a dwarf, aged 45, height somewhat under 3′, was seen by Temple in the retinue of the Bey of Tunis at a place called Tozer. He was called Aboo Zadek and had a family, II. 1—6, consisting of four boys and two girls. He had been married four times, and his fourth wife was said to be extremely pretty. (Bibl. No. 77, p. 180.)

Fig. 769. *Bode's Case.* No statement is made with regard to I. 1. I. 2, was healthy till she had chicken-pox in childhood. She was fairly strongly built and showed no signs of rickets or any disease, except a moderate degree of struma. She had twins, II. 1—2, at her first confinement, no statement is made regarding them. At her second confinement there was born a boy, stillborn, II. 3, who weighed 3100 grammes. The middle of the length of the body was about at the base of the xiphoid process, the head was hydrocephalic, the nose flat and depressed at the root, the skin of the trunk swollen and in some places in folds, the chest depressed and short, the abdomen prominent. The extremities were much too short in proportion to the trunk. In a vertical position the upper arms did not reach the iliac crests. The diaphyses were ossified and shorter and thicker than in normal cases, the epiphyses enlarged. The diaphyses were curved, the humerus was concave towards the front and inwards, the ulna concave towards the front, the radius concave outwards and towards the front. In the femur, the head and great trochanter were to outward appearance fully ossified; "die Epiphysenenden nach hinten stark umgewälzt, so dass eine stark vorn convexe Krümmung zu Stande kommt, welche am rechten Femur zugleich stärker als links nach aussen gerichtet ist, so dass die Patella mehr auf der äusseren Seite aufsitzt." The hand was greatly developed in proportion to the arm. The thorax was shortened from above downwards and the lower aperture enlarged towards the sides. Full measurements are given both of body and skeleton but some appear to be rather vaguely defined. (See Bibl. No. 203, p. 421.)

Fig. 770. *Mansfeld's Case.* No statement is made with regard to I. 1 and I. 2, except that I. 1 was a ladies' tailor and II. 1 was his illegitimate son. II. 1, was a soldier for a long time; in 1815 he squandered his little property in drink, became a complete drunkard and in 1816 married II. 2, a woman several years older than himself. They had five children, III. 1—5. III. 1, died of a scrofulous affection of the abdomen. III. 2—3, with the exception of scrofulous eruptions and inflammation of the eyes, were fairly healthy and well grown. Three years before the birth of III. 5, II. 1 had inflammation of the lungs and vomiting of blood, and for a still longer time he had suffered from cough and contraction of the chest. II. 2, during her pregnancy with III. 5, had suffered from gouty and hysterical affections. Both parents were over 40 years of age at time of birth and were

in the direst poverty. The confinement was normal. The child II. 5, a girl, showed no deformity except that her feet were drawn up to the body. The L. foot was turned towards the genital organs and covered them, both knees were turned out and it was impossible to extend the legs. The head was round like a ball and covered with long black hair; its size, not over large, was proportionate to the size of the rest of the body. All the ribs looked as if they had been fractured *at least* once and grown together again. The upper arms showed apparent fracture in the middle and grown together again. The ulnae and radii of both arms were much thicker than normal and were so close together that there was no recognisable interstitial space. They also showed signs of healed fractures as did the femora and tibiae. *Measurements.* From the vertex to the tips of the toes 1′ 1″ 3‴; to the end of the sacrum 9″ 8½‴. Length of spine to the end of the sacrum 6″ 9‴; dorsal spine 2″ 8‴; sternum with the xiphoid process 2″ 2‴; the xiphoid process 6‴; head from posterior end of sagittal suture to chin 3″ 3½‴. Maximum height of head in region of vertex 2″ 11‴; breadth of head close under both parietal protuberances 2″ 11‴. Length of R. humerus without cartilaginous extremities 1″ 3‴; L. humerus without cartilaginous extremities 1″ 4½‴; R. ulna 1″ 5½‴; L. ulna 1″ 6½‴; R. radius 1″ 2½‴; L. radius 1″ 3½‴; middle metacarpal bone 4½‴; middle finger of each hand 10‴; R. thigh 1″ 4½‴; L. thigh 1″ 5½‴. Maximum thickness of thigh at upper end 7‴; in middle 6‴. Length of R. tibia 1″ 1‴; L. tibia 1″ 1‴; each foot from the heel to the second toe 1″ 5‴, etc. (Bibl. No. 76, p. 552.)

Fig. 771. *Dyes's Case.* Of I. 1 and I. 2 nothing could be ascertained. II. 3, was a twin brother of II. 2, and it is stated that he, II. 4 and II. 5 were all remarkably small, not much taller than II. 2. II. 2, primipara, aged 40, was unmarried. She had been well taken care of by her foster-parents and as a child had never been ill. She came to the hospital for her confinement. She was well nourished, her muscles were well developed but flabby. All parts of the body appeared retarded in growth, especially the upper extremities. The root of the nose was sunken; the tubera parietalia were projecting. Her expression was not that of a cretin. The teeth were regular and showed no abnormality. The spine was straight, the epiphyses of the long bones were not thickened and the diaphyses were straight but noticeably short. Her gait was slightly waddling; she gave good answers to questions. The head was rather too large for the trunk. On account of the small pelvis, it was decided to bring on a premature confinement in the 32rd week of pregnancy. A female child, III. 1, was born without any deformity. Weight 4¼ "Pfund." Length 45·5 cm. The head measurements were: Horizontal diameter 102 mm.; greater transverse diameter 80 mm.; lesser transverse diameter 70 mm.; greater oblique diameter 115 mm.; lesser oblique 80 mm.; circumference 30 cm. It is not stated whether the child lived or not, apparently the mother survived. *Measurements of mother.* Total height 120 cm. Head measurements: Diameter between glabella and external occipital protuberance 16 cm.; anterior transverse diameter between the alae magnae 11 cm.; posterior transverse diameter between the tubera parietalia 13·5 cm.; height of head from foramen magnum to vertex = external auditory meatus to vertex 13·5 cm.; vertical distance of chin from vertex 19·2 cm.; distance from the root of the nose to vertex 12·5 cm.; circumference over glabella and external occipital protuberance 48 cm.; circumference (through ends of greater oblique diameter) 55·5 cm.; transverse arc (Topinard), *i.e.* from one cavity under the auditory meatus to the other 35 cm. Length of trunk = vertical distance of the cavity under the external auditory meatus to the horizontal line of the symphysis pubis 53·5 cm. Suprasternal notch to the xiphoid process 16 cm. Sagittal diameter of the thorax, at the height of the suprasternal notch 11·5 cm.; at the middle of the sternum 15 cm.; at the level of the xiphoid process 17 cm. Circumference (tape measurement): at the level of the manubrium 73 cm.; at the level of the middle of the sternum 75·2 cm.; at the level of the xiphoid process 69 cm. Breadth of chest at axillae 26·5 cm. Upper limb: from acromion to tip of middle finger 52 cm.; from acromion to lateral condyle on the lower end of the humerus 21·5 cm.; from the lateral condyle to the styloid process of the ulna 17·5 cm.; length of hand from semilunar bone to tip of middle finger 13 cm.; distance from the tip of the middle finger to the sole 48·5 cm.; distance from acromion to acromion 26·5 cm. Lower limb: from upper edge of symphysis pubis to the sole 57 cm.; from the tip of the great trochanter to the tibio-femoral articulation 28 cm.; from the external malleolus to the tibio-femoral articulation 26·2 cm.; length of foot 19·5 cm.; distance from the knee joint to the sole 31 cm.; "cavity of Baudelocque" (? locus) to the sole 60·5 cm.; lower edge of symphysis pubis to the sole 53 cm. (Bibl. No. 225, p. 14.)

Fig. 772. *Kehrer's Case.* I. 1, died of tuberculosis. I. 2, was healthy. II. 2, aged 19, was pale, but appeared to be healthy. Nothing is said about II. 3. III. 1, was born in the 8th month and died at birth. Its head was large, the ribs bent in on both sides, the abdomen large, and the extremities relatively short, with short, curved, thick diaphyses and enlarged epiphyses. It was the first child. No measurements of the skeleton are given. (Bibl. No. 145, p. 61.)

Fig. 773. *Kaufmann's Case.* No statement is made with regard to I. 1 and I. 2, but II. 1, aged 20, and II. 2, aged 16, were brother and sister. Neither of them ever had syphilis, but II. 2 was weakly. A premature confinement was brought on and description of the infant, III. 1, a female, is given. The expression was senile, the nose flattened and rather depressed at the bridge, the

upper lip and eyelids very thick, and the soft parts of the pelvic region greatly developed. One could not distinguish where the forearms ended and upper arms began, and there was a circular groove at the wrists. The hands were flexed at an acute angle on the forearms. In the lower limbs the heels touched the inner surface of the thighs, the outer edge of the foot was turned very much out and the soles were very concave. The spinal column was very flexible; the clavicles were hard and ossified throughout but slender. The scapulae were short and thick and measured 2·2 cm. at the inner edge. The humeri were bent at an angle inwards and their upper epiphyses were disproportionately thick. The short thighs were bent at an angle on the inner side and the bones of the lower part of the leg were much shortened and bent at an acute angle. The feet were in equino-varus position, the end of the calcaneum being about 1·2 cm. from the external condyle of the femur. *Measurements.* Skull: circumference 23 cm.; smaller transverse diameter 6·4 cm.; greater transverse diameter 7 cm.; longitudinal diameter 8·2 cm.; perpendicular diameter 5·5 cm.; smaller oblique diameter 7·5 cm.; greater oblique diameter 8 cm.; length of large fontanelle 3·2 cm.; breadth of large fontanelle 2·2 cm. Length of body 24 cm. Distance of vertex from umbilicus 17 cm.; from sole to umbilicus 7 cm.; xiphoid process to umbilicus 5 cm. Circumference of chest at nipples 20 cm.; at axillae 18 cm.; at abdominal ring 21 cm. Maximum abdominal circumference 18 cm. Circumference at umbilicus 17 cm.; of pelvis at antero-superior iliac spines 13 cm. Distance from the symphysis pubis to umbilicus 3·5 cm. Length of arm 6·5 cm.; hand 3 cm.; index finger 1·2 cm.; hand without fingers 1·1 cm. Circumference of hand 5 cm. Length of lower limb 8 cm.; foot 4 cm. Circumference of calf 6·5 cm. Maximum thickness of thigh 11 cm. Width of neck 12 cm. Length of clavicle 2·6 cm.; femur 4 cm.; diaphysis of femur 2·5 cm.; transverse diameter of diaphysis of femur 0·5 cm.; upper epiphysis of femur 1·9 cm.; lower epiphysis of femur 2·1 cm. Length of tibia 2·2 cm.; diaphysis of tibia 1·8 cm.; fibula 1·5 cm.; humerus 3·8 cm.; radius 2 cm.; ulna 2 cm. (Bibl. No. 275, pp. 16 and 25.)

Fig. 774. *Reyher's Case I.* I. 2, and I. 3, were siblings. This is the only thing of note in the family history. II. 2, and II. 3, had nine children, of whom seven were alive. The account states further that II. 2 had miscarried four times, twice, III. 1—2, before the first child, once, III. 6, before the fourth child, and once, III. 12, before the ninth child[1]. Of the children that died, III. 3 died aged 14 months of inflammation of the chest and lungs, the other, the fourth child, III. 7, died aged 4 days; this child had a similar appearance to that of III. 13. The doctor who was present said it measured 40 cm., the limbs were extremely short and deformed, the head large relative to the body, the nose flat and depressed and it also had cleft palate. Five of the surviving children, III. 5 and III. 8—11, were healthy and well grown. III. 4, aged 14, was retarded in growth but showed no sign of chondrodystrophia foetalis, being well proportioned. The mother said she had carried the achondroplasic children longer than the others. III. 13, was brought to hospital at the age of 4 months; the mother said that from birth she had a large head, short neck, protuberant abdomen and too short limbs, and had suffered from shortness of breath. The trunk was normal, the head appeared too large, the limbs were too short. With hanging arms, the finger tips scarcely reached to the trochanters, the hands were short, square and trident shaped. The neck was very short, the tongue appeared rather large, the skin lay in folds on the lower limbs. The hair was dry; she had slight umbilical hernia and genu valgum on both sides. The knee-joints exhibited a considerable amount of super-extensibility. In the elbow joints perfect extension was impossible. *Measurements.* Total length at age of 4 months 51 cm. Length from vertex to umbilicus at age of 4 months 26 cm. Distance from umbilicus to sole of feet, at age of 4 months, not quite 25 cm. Total length of body at age of 8½ months 56·5 cm. Length of trunk at age of 8½ months 33 cm.; lower part of body at age of 8½ months 23·5. Circumference of head at age of 8½ months 42 cm. Weight at age of 8½ months 5660 grammes. (Bibl. No. 545, p. 130.)

Fig. 775. *Volkov's Case I.* Very little is said of this case. I. 1, and I. 2, were normal. Their son, II. 2, aged 24, a peasant of Tartar origin, in the district of Tetiushi, in the province of Kazan, was 90 cm. in height, very well made and fairly robust. (Bibl. No. 324, p. 288.)

Fig. 776. *Reyher's Case II.* I. 1, and I. 2, were healthy. No case of dwarf growth or deformity was known in their families. Of their children, II. 1, aged 16, was perfectly healthy. II. 2 and II. 4, were stillborn, the result of strangulation by the umbilical cord, but they were well formed. II. 3, died aged 1½ years of diphtheria. After birth of II. 5 there were two miscarriages, II. 6—7. II. 5, aged 3½ years, weighed 9½ "Pfund" at birth. It was noticed then her head was too large and limbs too short. The birth was normal, she was breast-fed for three weeks and began to walk at the end of the second year. Her head was abnormally large with projecting tubera frontalia and parietalia on one side. The large fontanelle was not closed. The limbs were short, the fingers scarcely reached to the trochanters. She had lumbar lordosis. In peculiarities of skin, knee and elbow joints and genu valgum this case resembled Reyher's Case I. The humerus and femur were very short. A radiographic plate of the forearm is given. She weighed 23½ "Pfund." *Measurements.* Circumference of head 53·5 cm. Length of body 78·5 cm.; part above umbilicus 40·5 cm.; part below umbilicus 38 cm.; radius 7·9 cm.; ulna 8·9 cm.;

[1] Owing to a misreading III. 12 on the plate is marked as *four* miscarriages instead of as the fourth miscarriage.

metacarpus I. 1·6 cm.; metacarpus II. 2·3 cm.; metacarpus III. 2·3 cm.; metacarpus IV. 1·9 cm.; metacarpus V. 1·9 cm. (Bibl. No. 542, p. 134.)

Fig. 777. *Schwenderer's Case.* Of I. 1 no statement is made. I. 2, was never rachitic but in youth she had suffered from goitre, which had disappeared for a time under treatment, but later reappeared. Her sister, I. 3, was a deaf mute. She had eight children and a miscarriage, II. 1—9. One child, a boy, II. 1, aged 13, was rather retarded in intellect but did not suffer from goitre or rachitis. II. 2, was a miscarriage. II. 3, died young of inflammation of the lungs. II. 4—8, had neither goitre nor rachitis. II. 9, the eighth child, came into the world alive but died soon after. The extremely large head made extraction difficult. The nose was flattened, the neck short and thick, the abdomen large, the scrotum large and oedematous, reaching to the soles of the feet. The extremities were short. The whole arm was curved so that there was posterior and external convexity. No articulation was visible between the upper arm and the forearm or between the thigh and the leg. The feet were in decided equino-varus position, so that the soles of the feet touched the scrotum. The spine was flexible and not deformed. The muscles well developed. *Measurements.* Total length 35 cm. Circumference of head 37·8 cm.; of skull 35·5 cm. Bi-temporal diameter of head 9 cm.; of skull 8 cm. Bi-parietal diameter of head 11 cm.; of skull 10·3 cm. Abdominal circumference at umbilicus 33·5 cm. Distance from vertex to umbilicus 25 cm.; umbilicus to sole of feet 10 cm. Circumference of hips at antero-superior iliac spine 31 cm. Length of upper limb 10 cm.; hand 4·7 cm.; extended middle finger 2·7 cm. Circumference of upper arm 12 cm. Length of lower limb 6·5 cm.; of foot 5·5 cm. Maximum circumference of thigh 14·5 cm. Circumference of calf 11·7 cm. Length of humerus 4·7 cm.; radius 2·9 cm.; ulna from tip of olecranon 3·6 cm. Length of diaphysis of femur 4·7 cm.; upper epiphysis 1·3 cm.; lower epiphysis 1·2 cm.; tibia 3·8 cm.; fibula 3·4 cm. A picture of the child is given and an account of the microscopical examination. (Bibl. No. 357, p. 14.)

Fig. 778. *West and Piper's Case.* I. 1, aged 40, was tall and slender. I. 2, aged 25, was slightly below medium height. Their parents were alive and well. They had three children. II. 1, died of pneumonia at 6 months as far as could be ascertained. She was healthy. II. 2, aged $3\frac{1}{2}$, was alive and healthy. II. 3, aged 14 months, was born normally. Two things were noticed at birth, the large round head and the shortness and thickness of the fingers. She was breast-fed but very cross and cut her teeth at the eighth month. When 5 months old slight angular curvature of the spine was noticed. The intelligence was normal. The head was very large with a full growth of soft hair, the anterior fontanelle was open, and the frontal and parietal eminences prominent. The bridge of the nose was depressed and the end turned up. She had slight exophthalmus. The neck was very short and the hands trident shaped. *Measurements.* Weight $13\frac{3}{4}$ lbs. Length $24\frac{1}{2}''$. Circumference of neck 8″; chest at nipples 14″; abdomen at umbilicus 15″; pelvis at crests $12\frac{1}{2}''$; head $19\frac{3}{8}''$; head from ear to ear $11\frac{1}{4}''$; head from glabella to occiput 14″. Head diameters: bi-temporal $4\frac{1}{2}''$; bi-parietal $5\frac{1}{4}''$; antero-posterior $6\frac{1}{2}''$; glabella to occiput 6″. Length from the vertex to the antero-superior spinous process $15\frac{1}{2}''$; antero-superior spinous process to the sole 9″. Distance from sternum to pubis $10\frac{1}{2}''$; sternum to umbilicus 8″; umbilicus to pubis $2\frac{1}{2}''$; trochanter to condyle of femur $3\frac{3}{4}''$. Length of fibula $3\frac{1}{4}''$; tibia 3″; foot (instep $4\frac{1}{2}''$ to $4\frac{3}{4}''$) $3\frac{1}{2}''$. Distance from acromion to wrist $6\frac{1}{2}''$. Length of humerus 3″; ulna 3″; hand and wrist $1\frac{1}{4}''$. (Bibl. No. 453, p. 730.)

Fig. 779. *Thomson's Case.* I. 1, and I. 2, were healthy respectable people. They had 10 children, II. 1—10, four of whom, II. 1—4, died of acute illnesses. One of these, II. 4, the fourth in order, was said never to have grown properly and to have been always dull. The tenth child, II. 10, was a well-marked sporadic cretin. The others, with the exception of the eighth child, II. 8, were all well-grown and normal. Neither I. 1 nor I. 2 knew of any idiots or dwarfs among their relatives. II. 8, aged 4 years and 8 months, had at birth been considered a big baby and was very fat. She was breast-fed for 11 months and at first seemed to thrive well, but when 6 months old the mother noticed she was not as large as she should be, though in other respects she seemed normal and was bright and lively. She began to get her teeth at 7 months old and cut them rapidly and easily. When 9 months old she had her first "turn" or "fit," she became unconscious and the face assumed a bluish tint. Three or four months after she had a similar seizure, and after this they recurred at irregular intervals about once a month. Her mental development proceeded normally and her mother thought she was quite as intelligent as other children of her age. When about $3\frac{1}{2}$ years old she measured 27″. She was brought to the hospital on February 26th, 1897, and then looked like a child of 18 months old. Her height was $28\frac{1}{2}''$ and weight 20 lbs. 7 ozs. The circumference of the cranium was $18\frac{1}{8}''$, that of the thorax 18″, and that of the abdomen 20″. The body was well nourished and fairly normal in proportions, the skin soft and natural, the hair light brown and fine, but rather dry and scanty, the forehead prominent and the bridge of the nose depressed. The abdomen, heart and lungs were normal and the hands and feet small and neatly formed. The limbs were firm and fairly muscular, and she was active and quick in her movements. Her intelligence seemed quite good, the memory particularly so. She was given thyrocol treatment and on January 12th, 1898, measured $30\frac{1}{2}''$ and weighed 21 lbs. 4 ozs. In March she got one of her old seizures. On April 6th she seemed quite well, but on April 17th became peevish and irritable and died

April 19th. The post mortem showed no abnormality but great congestion of the brain and the persistence of a greatly enlarged but otherwise healthy thymus gland. Thomson thinks the above was a case of ateleiosis, but Gilford thinks it a doubtful case. (Bibl. No. 366, p. 209.)

Fig. 780. *Carus' Case.* I. 1, an extremely small woman whose growth had been retarded by rachitis, gave birth to a stillborn child, who had all the signs of this disease. It had deformed ribs, the extremities of the bones of the limbs were greatly enlarged, and there was deviation of the spine. (Bibl. No. 66, p. 741.)

Plate LVII. Fig. 781. *Ekman's Case.* In the region of the iron mines in Dannemora a certain family was noticed, which for three generations produced small offspring whose distorted bones had a peculiar softness or fragility. As each male of these big-headed persons arrived at puberty, he married a healthy woman and had children. The bones of the limbs of these children were so frequently fractured, often without any noticeable shock, that when they reached a certain age they were more or less deprived of all strength and power of movement. The ancestor of this race, who died at the end of the century, was Nikolaus Ekroth, I. 1, born of parents who worked in the mines. From ecclesiastical annals and other trustworthy traditions it is certain that he was a little distorted man, quite unable to walk. The old inhabitants of Dannemora, who remembered his miserable condition well, stated they could see no cause for his deformities in his method of life. He married a domestic servant, I. 2, and had four children, II. 1—4. II. 1, born 1702, had in childhood no deformity, but when he became adult he lost power in both arms and legs, was unable to do any work and compelled to live by begging. He died 1775. II. 2, was born 1703. Nothing certain is known of her childhood, but in adult life she was of low stature with distorted body. Her gait was not unlike that of a goose and her feet were turned in. Of II. 3 nothing is known, she may have died in childhood or else migrated. II. 4, was well known among the people as a little man whose stature was very much less than that of other men and who was altogether deprived of the power of walking and compelled to sit. The form of his body was extraordinary with its curved and contorted arms and legs. He married a healthy wife, II. 5, and had a son, III. 1, born 1726. From III. 1's earliest years his legs and arms would break if the slightest force was applied, so that he became distorted like his father and grandfather and was compelled to remain seated. He could only move himself by supporting himself with his right arm and dragging the legs behind. He supported himself by begging and sometimes earned money by making nets. He died aged 56. He married III. 2, a tall strong woman and had two children, IV. 1—2. IV. 1, was born 1760. When aged 1 month the bones of his arms and legs were so soft that at the least contact they became curved and contorted. While less than a year old his bones were fractured three or four times, so that in early years he could only go on all fours unless supported by crutches. As he advanced in age his weak legs could not support the weight of his body, though light. He supported himself by making nets and begging. In 1782 it was said he fractured his tibia by a slight fall, but that it healed in a week. He sustained so many fractures in legs and arms that they became curved and contorted, but neither the joints nor epiphyses were injured. The vertebral column was erect, the head of natural size, the voice sonorous and the body so thin that the bones of the legs and arms could be easily distinguished. His mother said he had been healthy in childhood and no thinner than other children. He did not marry, but might have done so had he not fallen into a lake when drunk and been drowned. IV. 2, aged 23, was born 1765. Her mother said that eight days after birth her arms were fractured without any shock and that, like her brother, IV. 1, her arms and legs were frequently fractured by the slightest accident; even the femora, were often broken and healed again. Her bones became so curved and contorted that when sitting not only her legs but the femora were crossed underneath her. At the age of 14 she was attacked by some disease of which no description could be obtained, which lasted several weeks, and resulted in spinal curvature so that she could no longer keep her body erect. Her joints were flexible and free. The L. humerus was fractured three times in different places and was consequently shorter than the R. and twisted into a shape resembling an S. On account of the lower fracture she could not extend it properly, nor could she extend the fore-arm well as it was broken near the olecranon. The L. femur had been fractured three times in the same place. She was very thin, her head and pelvis were normal, her voice harsh but not unpleasant. III. 2, the mother, said that neither her husband nor his ancestors had ever suffered from syphilis. Their dwelling was not in a marshy place and their food was similar to that of hundreds of men who had robust and strong offspring. The case appears to be one of *congenital fragilitas ossium*; but perhaps rickets should be taken into consideration, although there is nothing definitely pointing to rickets. Other cases of familial *fragilitas ossium* are known; the dwarfism would be only a secondary feature. (Bibl. No. 44, p. 5.)

Fig. 782. *Bayon's Case II.* Nothing is said of Gen. I. II. 5, was twice married, and three times had a miscarriage in the third month. By her first husband, II. 4, she had five children, three sons and two daughters, III. 1—5, all quite normal, but one of the sons, III. 1, died of tuberculosis aged 16. By her second husband, II. 6, she had four children, III. 6—9. Of these, III. 6 died aged 6 months of diphtheria, and III. 7 died aged 2 years of scarlet fever. III. 9, Elizabeth Baunach, born 1883, walked at age of 1½ years and as a small child was alert and cheerful. At age of 5 she had inflammation

of the chest three times, and the third time she had also measles and ulcerative stomatitis. Since that time her mother noticed that she ceased to grow, her memory failed, the joints of her hands, knees and feet became thicker and she walked worse. She was sent to school at 6 years of age, but was sent back as being mentally and physically too weak. She could not learn her letters. At age of 10 she was sent to an Idiot Asylum. Here she apparently made fair progress, but was dismissed in a year as being good-for-nothing. She was in hospital from November, 1897, to May, 1899. Her skull was normal. Her weight 19 kilos. No sign of a thyroid gland could then be felt. The R. leg was in extreme genu valgum position. All the epiphyses were much enlarged (aufgetrieben). The back was rather hairy. Her hair was noticeable because it was like coarse bristles. There was nothing abnormal in her teeth except that an upper incisor was double. In both eyes there were spots on the cornea, and the L. eye showed traces of old iritis. Her speech was harsh, nasal and very quick. There was no deformity of the genitals, but no sign of puberty till, at the age of 19, her breasts began to develop. About $1\frac{1}{4}$ years later, in 1903, there appeared the first signs of pubic hair. The hair of her head had grown and the breasts were larger. The colour of the skin was normal, and the skin was not dry. There was no trace of myxoedema. The thyroid gland could be felt with difficulty in 1903. Bayon says he is unable to

	Height	Weight
Measurements:		
1897	92 cm.	19 kilos
1898	96 „	20 „
1899	98 „	22 „
1900 beginning	106 „	23 „
1901 middle	107 „	24 „
1902 beginning	108 „	25 „
1903 beginning	108 „	26 „

classify this case. II. 2, a cousin of II. 5, the mother, drowned himself and was declared insane. III. 10, child of II. 7, sister of II. 6, did not learn well at school. Possibly this is a case of cretinism. (Bibl. No. 436, p. 50.)

Fig. 783. *Dubois' Case.* I. 1, was a dwarf, $3\frac{1}{2}'$ in height. I. 2, was of ordinary stature. They had six children, II. 1—6. Three were normal, III. 1—3, and three were dwarfs, III. 4—6. Only a description of III. 6 is given. She was very small at birth and was exhibited under the name of "The Lilliputian." At the age of 23 she was $3'\ 2\frac{1}{2}''$ in height (French inches). She became pregnant in 1838; the confinement was most difficult. She had violent attacks of eclampsia and Dubois was called in. The forceps were used in vain, and he had to perform craniotomy before he could deliver the child, and even then rupture of the perinaeum occurred. This child, III. 1, weighed 5 lbs. (5 livres). Having become pregnant a second time she came to Dubois, who concluded that the child was very small and therefore waited till the 8th month. He then induced a premature labour, which passed off well. The child weighed 3 lbs. (3 livres). It is not stated whether the child lived, and no measurements or other data are given. Probably true dwarfism? (Bibl. No. 82, p. 513.)

Fig. 784. *Railton's Case.* I. 1, I. 2, and I. 4, were temperate. I. 3, was alcoholic. II. 2, and II. 3, were sober and healthy, not related, and from different parts of England. There was no general causes of ill health and no goitre in the family. II. 2, and II. 3, had seven children, III. 1—7. Of these, III. 2—3, and III. 5—7, showed no trace of cretinism or goitre. III. 1, aged 11, was $32\frac{1}{4}''$ high and weighed 34 lbs. He was the first child, born naturally and breast-fed. He cut his first teeth at 8 months old, "noticed" tolerably early and learned one or two of the usual childish words. When however he reached the age of 12 months, to use his mother's words "he seemed to stop short"; his development (both mental and physical) ceased almost completely. He did not walk till he was $3\frac{1}{2}$ years old. When seen, his stature, and one might almost say his intelligence, were those of a child of 2. His head though large in proportion to his height was not so for his age. It was 51 cm. in circumference and well shaped with the exception of some flattening at the vertex; the anterior fontanelle was closed. The hair and eyelashes were normal. The features were broad and coarse, with eyes set widely apart, the root of the nose being flattened while the alae and septum were thickened and the nostrils somewhat dilated. His thick everted lips were habitually open with a large tongue protruding between them. The face looked oedematous, the normal depression between the cheek and the lower eyelid being almost obliterated, but the skin was quite firm to the touch. There was a deep naso-labial line on each side of the mouth. He had only the blackened stumps of his milk teeth worn down to the level of the gum in the front of the upper jaw and in the lower his first teeth set widely apart were decaying and some permanent incisors were making their appearance behind. The thyroid gland was present and could be felt in his short neck, as a small firm immature body. The body was very bulky and the abdomen prominent. He had a small umbilical hernia. His chest was unsymmetrical and he had well marked natural curvature of the spine in

the lower dorsal and lumbar regions. There was also considerable lordosis. His limbs with their bony framework were short and thick, but there was no absolute distortion except a bowing of the tibia with the convexity anterior. The hands and feet were short and broad with stumpy fingers and toes. "The feet are flat and the second toe on each foot is smaller than normal and overrides the rest." The skin was harsh, dry and resistant. III. 4, aged 6 years and 3 months, was 33″ in height and weighed 32½ lbs. He cut his first tooth early and was said to be bright as an infant. He did not walk until he was 2 years and 3 months old. He waddled as he walked but he could even run. His head was fairly well shaped with a depression in the region of the anterior fontanelle, which was closed. His hair was normal. The root of the nose was broad and flat and the tip rather turned up, the alae and septum thickened and nostrils broad. He had rather a big mouth with a tendency to remain open. He had his first set of teeth which were set widely apart. His neck was short and thick and the thyroid gland was of much the same character as that of his brother. His abdomen was prominent and he had a small umbilical hernia. His chest was remarkable in the fact that it retained the circular form of infancy. There was slight beading of the ribs. The limbs showed the same peculiarities as his brother's, including that of the second toe of each foot. His perceptive faculties were probably more acute than those of his brother, but his intelligence was extremely limited. Probably sporadic cretinism. (Bibl. No. 404[1], p. 694.)

Fig. 785. *Manouvrier's Case III.* I. 1, was an enameller, very steady and not alcoholic. According to his wife, I. 2, there were no degenerates in either his family or hers. Both I. 1, and I. 2, were well made and of Alsatian origin. They had four children, II. 1—4. II. 1, was a pretty intelligent girl of 9. II. 3, died aged 2, of convulsions. II. 4, was vigorous and healthy. II. 2, aged 7, the dwarf, was microcephalic and a complete idiot though he seemed to recognise his mother and sister; owing to his movements his height could not be measured. The head measurements were as follows:

	1903 Aged 15	1895 Aged 7
Maximum antero-posterior diameter	133 mm.	130 mm.
Metopic antero-posterior diameter	125 „	125 „
Maximum transverse diameter ...	110 „	106 „
Vertical diameter	92 „	85 „
Bi-zygomatic diameter	103 „	94 „
Cephalic index	82·7	81·5

The mother said the forehead had been more flattened at birth, and that the head had greatly increased in size. She had noticed the bregmatic fontanelle did not exist at birth. The four canine teeth were remarkably pointed and sensibly longer than the other teeth; the large tongue was constantly protruded from the half open lips, the ears were normal. Only one transverse palmar fold existed in the L. hand situated nearly in the middle of the palm. The R. hand had not this characteristic. The child had had convulsions when from 2 to 3 months old. The mother stated that during her pregnancy with this child, she had worked in a match factory, by piece-work, carrying heavy loads of wood which she supported on her abdomen. During her other pregnancies her work had been different, either washing or working in a tobacco factory.

An additional notice of this dwarf was published by Manouvrier in 1903. He was then aged 15, and his height was 98·5 cm., but would probably have been 110 cm. if there had not been posterior deviation of the pelvis, with a bending forward of the trunk. The head measurements had not altered much. He had walked from the age of 9 and could pronounce a few words. He had had attacks of epilepsy since 1893. (Bibl. Nos. 302, p. 227 and 441[b], p. 591.)

Fig. 786. *Schmidt's Case III.* "Welsing." I. 1, I, 2, I. 3, and I. 4, were of average height, as were also II. 2, and II. 3. II. 2, was a sailor. III. 1—3, were strong and of normal size. III. 4, aged 15½, was big and strong at birth—and developed remarkably well till about his third year. He learnt to walk and speak like other children. At about 2½ years of age he was vaccinated, but the vaccine took but slightly. Two or three weeks after vaccination he became seriously ill, the chief symptom being continuous diarrhoea, a condition which lasted 12 years (?) and showed the first signs of improvement in the summer of the year he was seen by Schmidt. As a result of this illness he had become weaker and weaker and was so emaciated that he was almost a skeleton. In his fourth year he had measles badly, and from about his tenth year his legs were much swollen from time to time. He had been at school from his sixth to his fifteenth year and in spite of drawbacks kept pace with his companions of the same age. His gait had become tottering during his long illness, he could scarcely walk upstairs and showed a great desire to sleep. Since the diarrhoea had improved, his strength had increased and he had noticeably increased in height, although previously he had grown but little since he was 2½ years old. The parents said he had not suffered from rickets, but Schmidt thought that possibly he had, on account

[1] By an oversight this paper is dated 1902 instead of 1891 in the Bibliography.

of a considerable flattening of the occiput and a pronounced X-position of the legs. The genitals were in a child-like state of development. The mental faculties appeared quite normal. *Measurements.* Total length of body 119·9 cm. Length of head measured from the glabella to the most prominent point of the occiput with head horizontal 168 mm. Breadth of head 144 mm. Perpendicular length of spinal column 53·0 cm. Length of whole arm from acromion to the end of the 3rd phalanx of the middle finger 51·5 cm. Length of leg from trochanter to external malleolus 53·5 cm. A few other measurements are given. (Bibl. No. 270, p. 67 and pp. 69—74.)

Fig. 787. *Schmidt's Case IV.* "Theres Fend." I. 1, I. 2, I. 3, and I. 4, were healthy and of average size, as were also II. 2, and II. 3. III. 1, aged 21, and III. 2, aged 19, were normal and healthy. III. 3, aged 16, was 116·0 cm. in height. Her birth was normal and for the first eight years she was the size of other children, and was never ill. Then came a severe illness, which kept her in bed 14 days and she was delicate for a long time after, and from that time she at most grew 1″ to 2″ (Zoll). What the illness was could not be ascertained. She had never been ill again. Her body was well proportioned but the head was too small for a girl of 16, but showed no trace of the form of a typical microcephalic skull. The limbs and especially the hands were pretty, with fat and muscles well developed. The whole body would have been handsome, except for the projecting abdomen. The breasts were scarcely noticeable, the nipples quite undeveloped. There was no hair on genitals or axillae, but plenty of hair on her head. The shoulders were broad and the thorax particularly well formed. She said her legs were not the same length, her parents knew nothing of it, but measurements from the trochanter to the external malleolus proved she was correct, for the R. leg was about 0·5 cm. longer than the L. The R. upper and lower eye teeth had only changed at 16 years of age and were merely little points rising from the gums when seen. Her bodily strength was not weak relatively to her small size, but she was easily tired and could only do light physical work. Mentally she was perfectly normal, could write well, read fluently and knew some arithmetic. *Measurements.* Total length of body 116·0 cm. Length of head from glabella to external occipital protuberance 166 mm.; to most prominent point of the occiput when head horizontal 168 mm.; breadth of head 144 mm. Perpendicular length of spinal column 49·8 cm. Length of sternum 11·9 cm. Circumference of chest measured above nipples (average in quiet breathing) 60·0 cm.; of abdomen at umbilicus 59·6 cm.; of hips at crests 60·5 cm.; at trochanters 66·5 cm. Length of clavicle 10·0 cm.; humerus 19·0 cm.; ulna 17·6 cm.; radius 16·0 cm.; hand from end of radius to end of 3rd phalanx of middle finger 13·0 cm.; to beginning of 1st phalanx of middle finger 5·0 cm.; whole arm from acromion to end of 3rd phalanx of middle finger 48·0 cm.; circumference of middle of upper arm 17·9 cm.; maximum circumference of forearm 18·7 cm. Length of femur 28·0 cm.; tibia 25·7 cm.; foot 22·2 cm.; leg from trochanter to external malleolus, R. 53·5 cm., L. 53·0 cm.; circumference of middle of thigh 22·7 cm.; calf 23·9 cm. Other measurements are given. (Bibl. No. 270, p. 59 and pp. 69—74.)

Fig. 788. *Schmidt's Case V.* "Margaretha Reisberger." The account of this family was given to Schmidt by II. 3. I. 1, I. 2, I. 3, and I. 4, were of average height and no case of dwarfism was known in the family except III. 5. II. 2, a peasant, was of average size, and delicate but never ill. He died suddenly aged 62, according to his relatives of apoplexy. His wife admitted he was a drinker[1]. II. 3, aged 62, was of average height and had always been healthy. III. 1—4, were of normal height. III. 5, aged 26, was strong when born and had grown like any other child till five years of age and during this time was never ill. Then she had a severe illness which lasted seven weeks, her mother had no idea what it was, and the doctor who had attended her was dead. From the account of the mother it appeared to be a severe general illness. Since then she had not increased in height, but her head had grown and her body and limbs had become thicker. Since this illness, she had never ailed except for pains in the loins from time to time. She went to school for three summers but learnt almost nothing. She followed what was said to her but even her relations found it difficult to understand what she said. Her gait was waddling. She measured 108·3 cm. in height. The muscles were well developed, the breasts undeveloped, the nipples were developed, the genitals without hair and like those of a child. The abdomen was distended and the neck very fat behind. The breadth of the pelvis gave the whole figure a woman's character. The expression of countenance with its projecting lower lip and flat broad nose reminded one of a monkey. She had learnt to sew and knit a little but was chiefly employed in looking after children, which she did in a satisfactory manner. *Measurements.* Total length of body 108·3 cm. Length of head from glabella to external occipital protuberance 169 mm.; to most prominent point of occiput when head horizontal 168 mm.; breadth of head 138 mm. Perpendicular length of spinal column 51·5 cm. Length of sternum 13·9 cm. Circumference of chest above nipples 61·5 cm., average with quiet breathing 55·5 cm.; of abdomen at umbilicus 68·5 cm.; at trochanters 64·2 cm. Length of clavicle 10·9 cm.; humerus 19·4 cm.; ulna 16·5 cm.; radius 14·2 cm.; hand from end of radius to end of 3rd phalanx of middle finger 13·2 cm.; from end of radius to beginning of 1st phalanx of middle finger 6·1 cm.; whole arm from acromion to end of 3rd phalanx of middle finger 47·8 cm.; circumference of middle of upper arm 18·8 cm.; maximum circumference of forearm 18·9 cm. Length of femur 23·7 cm.; tibia 23·0 cm.; foot 15·9 cm.; leg from trochanter to external malleolus 45·5 cm.; circumference of middle of thigh 30·8 cm.; calf 23·7 cm. Other measurements are given. (Bibl. No. 270, p. 60 and pp. 69—74.)

[1] Owing to an error of engraver I. 2 instead of II. 2 has been marked on plate as alcoholic.

Fig. 789. *Schmidt's Case VI.* "Wilhelm Willkowsky." I. 1, I. 2, I. 3, and I. 4, were of average size. II. 2, was a mason and of average size, as was also his wife, II. 3. They had twelve children, III. 1—5, of whom eight, III. 1, died in childhood of acute infectious diseases. III. 2, aged 24, the eldest of the survivors, was a sailor, with rather a big head but otherwise mentally and physically normal. III. 4, aged 17, an errand boy, was normal. III. 5, aged 7, had a skull which was slightly of the hydrocephalic type, but she was otherwise normal. III. 3, aged 20, was a hydrocephalic dwarf. His large head was noticed immediately after birth. He was breast-fed and at first developed well, but at 3 months old he had violent attacks of vomiting which lasted four weeks. He was blind for a year with some disease of the eyes. He never learnt to walk, but grew at first like other children, then more slowly, but growth did not cease altogether. He learnt to speak in normal fashion at first, but stopped speaking in his third year and forgot what he had learnt, he began to learn speaking again in his sixth or seventh years, and according to his mother had learnt to answer a few questions but had forgotten them again. With the exception of the hydrocephalus the body was in proportion and rather fat, but the muscular system was badly developed. The expression of the face was very thoughtful and by no means idiotic. The development of the genitals was not retarded, but the hair was very scanty. *Measurements.* Total length of body 132·0 cm. Length of head measured from the glabella to the external occipital protuberance 200 mm.; breadth of head 175 mm. Cephalic index 87·5. Other measurements on the head and face are given. (Bibl. No. 270, p. 65 and pp. 69—74.)

Fig. 790. *Schmidt's Case VII.* "Struss Family." According to II. 3, I. 1, I. 2, I. 3, and I. 4, had been physically and mentally normal. II. 2, was a master smith and normal. II. 3, was seen by Schmidt and apparently was normal. III. 1, aged 22, the eldest child, was a healthy journeyman smith. III. 3, who was a decided microcephalic, died in his 13th year, he had been 5 or 6 years in the Idiot Asylum at Alsterdorf. No measurements of him are given. III. 2, aged 10, died in the Idiot Asylum, but her parents refused to allow an autopsy. She was a microcephalic dwarf. II. 3, said nothing particular occurred while pregnant with III. 2—3. III. 2, had suffered from convulsions and often cried. Neither III. 2, nor III. 3, had ever learnt to speak nor could they recognise anyone. III. 2, walked at 2 years old but uncertainly. III. 3, could only crawl and apparently had pronounced pes varus. They never understood what was said to them. In her 11th year III. 2 was only the size of a five year old child, and III. 3, in his 13th year, only the size of an eleven year old boy. Their growth was slow but constant. *Measurements* of III. 2. Length of head from glabella to external occipital protuberance 127 mm.; to the most prominent point of the occiput, with the head horizontal 131 mm.; breadth of head 117 mm. These measurements (with others) are taken from a cast of the head. (Bibl. No. 270, p. 66 and pp. 69—74.)

Fig. 791. *Baginsky's Case I.* I. 1, had had epileptic attacks till his 8th year. I. 2, was healthy and had never miscarried. II. 1, aged 10, had had epileptic attacks till his 5th year, he had congenital defect of the iris, nystagmus of both eyes and was highly nervous and timorous. II. 2 (no sex given), aged 6, had epileptic attacks till the 2nd year of its age. II. 3 (no sex given), was normal and well developed. II. 4, was born 9. 10. 1888 and brought to Baginsky on 22. 6. 1889. The thorax was normal, the extremities abnormally short, firm and thick, and the tibia and fibula were slightly curved. Her expression was imbecile, the root of the nose was depressed and the tongue enormously large. They operated on the tongue and the child died in consequence. There was a post mortem. *Measurements.* Circumference of head 41·5 cm.; of thorax at axillae 36·5 cm.; at height of the xiphoid process 36·5 cm. Length of body 59 cm.; the upper limb from the acromion to the wrist 13 cm.; the lower limb from the iliac spine to the external malleolus 20 cm. Circumference of forearm 12·25 cm.; of calf 15 cm. (Bibl. No. 261, p. 515.)

Fig. 792. *Baginsky's Case II.* Of I. 1, no statement is made. I. 2, was phthisical, but there were no traces of syphilis. Of her seven children, II. 1—7, four, II. 1—4, had died of acute disease. II. 7, born 24. 8. 1881, was brought to the hospital 25. 11. 1881. She was a badly nourished child, with thick protuberant tubera parietalia and frontalia and large open fontanelles. The arms and legs were remarkably thick and short with massive bones and large epiphyses. The skin lay in folds on them. Both tibiae were so incurved on the anterior edge that the lower third of the leg appeared to be convex posteriorly and concave anteriorly. She had genu valgum on both sides. *Measurements.* Circumference of head 41 cm.; of upper part of chest 33 cm.; of lower 33 cm. Length of body 50 cm.; upper arm 8 cm.; forearm to wrist 6 cm.; hand from wrist to the tip of the middle finger 5 cm.; the thigh from the iliac spine 10 cm.; the leg to the external malleolus 7·5 cm. (Bibl. No. 261, p. 528.)

Fig. 793. *Baginsky's Case III.* Of I. 1, and I. 2, nothing is stated. II. 8, born 10.6.1886, was brought to the hospital 22.6.1886. No trace of syphilis could be ascertained. She was the eighth child of her parents, three of the others, II. 1—3, had died of acute diseases. The tubera parietalia and frontalia were greatly developed, the face normal, the limbs remarkably short and thick. The right upper limb lay immovable, and exhibited a slight thickening of the humerus in the middle. At any attempt at movement flexibility and crepitation were noticed (fracture of the humerus). The elbow joint was normal. The epiphyses of the humerus and wrist were thickened at both sides. The diaphysis of the L. humerus appeared to be normal. The femora on both sides were thick and curved, the tibiae thick and bent. There was nothing abnormal in the spine. *Measurements.* Circumference of head 34·0 cm

Upper circumference of chest 32 cm.; lower 32 cm. Circumference of abdomen 35·5 cm. Total length of left upper limb to the wrist 12 cm.; of both legs from the iliac spine to the external malleolus 19 cm. (Bibl. No. 261, p. 529.)

Fig. 794. *Kühn's Case.* I. 1, aged 50, was a robust muscular man almost 6 "Fuss" in height and a mason by trade. He met with an accident and Kühn was called in and saw his family. His wife, I. 2, was strong, healthy, intelligent and talkative. She was 5 "Fuss" in height. She married in her 25th year and never had had syphilis or any serious illness. Her confinements had been easy and she breast-fed her children to their second year. After her first two boys, II. 1—2, she had a miscarriage, II. 3, a boy. The miscarriage was the result of a fall. Her second daughter, II. 5, died of smallpox in infancy. I. 1, according to all accounts, had led a blameless life both before and after marriage. He was very intelligent. His genitals were normal and healthy. There was no alcoholism and none of their ancestors as far as they knew had been defective either in stature or intellect. The seven children, II. 1—7, had been born at intervals of 3 years, and five were alive. II. 1, aged 24, had a fairly good head-piece, his father had sent him to learn writing and arithmetic in town. His height was "3 Fuss 2 Zoll." He had neither a thick body nor thick head, his limbs were straight and well proportioned, his teeth were white and perfect and there was no eruption on his skin. His hair was long and dark brown in colour, but there was no trace of hair on his face or pubes and he had no sexual feeling. Besides childish diseases, in his 14th year he had a very prominent abdomen, but had no other signs of rickets or worms. He sometimes got sudden violent cramp in his right leg and fell, and he sometimes had catalepsy but his father revived him by rubbing and warmth. He was very strong in the loins and back and could carry heavy loads of wood. II. 2, aged 21, was big and strong like his father, with a wrinkled face, a spiteful, obstinate and ill-tempered disposition, and deficient intellect. He was a big eater and drinker, with a voice like a man, but his genital organs were like those of a two year old boy, there was no hair on chin or pubes and his testicles were the size of those of a cockerel. II. 4, aged 16, was very imbecile, with a face almost like an animal's, and disfigured by smallpox. She could not speak clearly and showed no signs of puberty; she was no taller than I. 1 and should have been marked as a dwarf; her limbs were in proportion. II. 6, aged 10, and II. 7, aged 7, were both "zwei Schuh" in height, their bodies were not deformed and their features were more passable than those of their elder brothers and sister. If spoken to they only laughed foolishly, they could speak no word beginning with a consonant and might almost be considered dumb. Their tongues were thick and large and the other organs of speech could not be examined. (Bibl. No. 39, p. 367.)

Fig. 795. *Depaul's Case.* No statement is made with regard to I. 1. I. 2, aged 38, came for her confinement to the hospital. She was a tall woman of excellent constitution, a washerwoman by trade. She had never been ill and had no trace of scrofula, rickets or syphilis. She had had two boys and a girl, II. 1—3, all normal and well developed, but the boys had died. She then had a miscarriage at six months, *male* twins, II. 4—5. Her last confinement followed this by eleven years. When II. 6 was born and she saw what he was like, she said she had seen a man with very short arms. II. 6, a boy, was born naturally but died shortly after birth. He had a large head which looked larger than it really was and short limbs. The spinal column was normal but the cervical portion seemed short. The face was normal, the thorax small with ribs regularly curved, but the clavicles were disproportionately long relatively to the size of the thorax, 3·5 cm. without considering the curves, the shoulders were consequently forced backwards. The humerus at each side was much bent anteriorly, the chord of its curve being only 1 cm. in length, the bony surface of the concavity appeared flattened and as if excavated. The radius and ulna, which were parallel, were both 2 cm. in length, but while the radius exceeded the ulna at the lower end, the ulna exceeded the radius at the upper end. The four extremities of these bones were of abnormal volume, especially the lower extremity of the radius and the upper extremity of the ulna. The bones of the hand were regular in form and direction. Both femora were curved in the same direction but the right was straighter than the left. They had first a very marked curvature with the concavity directed backwards and inwards, and secondly a less pronounced curvature with internal concavity. Their lower extremities were considerably enlarged. The tibiae were thick and short, the anterior surface which measured 10 mm. being much shorter than the posterior which measured 18 mm., they were slightly curved with concavity directed backwards and outwards. The fibulae were slightly curved, the concavity being directed forwards and inwards. Their anterior length was 11 mm., posterior length 18 mm., and their extremities were enlarged. The feet were slightly turned out, the vertebral column was of normal height, viz. 15 cm. The child weighed 2700 grammes. *Measurements.* Length from vertex to heel 35 cm.; to coccyx 29 cm.; to umbilicus 25 cm. Head diameters: occipito-mental 13 cm. (skull 10 cm.); occipito-frontal 11 cm.; sub-occipito-bregmatic 10 cm. (skull 9 cm.); bi-parietal 9 cm. (skull 9 cm.); bi-temporal (skull 7·5 cm.); vertical (skull 8·5 cm.). (Bibl. No. 165, p. 643.)

Fig. 796. *James' Case.* I. 1, and I. 2, were normal Hindus; there were no dwarfs among their relations or immediate ancestors. Three of their sons, II. 1—3, were of normal size. II. 4, Piyara Lal, aged 20, was a Hindu goldsmith. He studied at school up to the 5th Primary class and stated that he worked with boys of his own age. He was quite intelligent and seemed quick at grasping new ideas. He had a falsetto voice. The penis and scrotum were small and undeveloped and he had no hair on his

face or pubes. For a native his complexion was very fair. He ran fast and seemed in every way to be an active, sharp-witted youth. The thyroid gland could be felt in his neck. He was said to have ceased growing at the age of 10. His height was 3′ 3″. Weight 2 stone 11 lbs. The limbs were absolutely symmetrical and their proportions to the head and trunk were those of a man not a boy. Major James considers this a case of infantilism. It may be classed, we think, as ateleiosis. (Bibl. No. 629, p. 445.)

Fig. 797. *James' Case II.* II. 8, Sewa Singh, aged 28, was a Sikh born in the Bikanir State. He was a member of a large family, having had four elder brothers, II. 1—4, and three elder sisters, II. 5—7, and seven younger sisters, II. 9—15, but out of these only two younger sisters were alive. All the others died before they grew up from various diseases. All were said to have been normal in stature and the two living sisters were tall women. Hitherto there had been no dwarfs in the family. II. 8, was 3′ $4\frac{1}{2}$″ in height and weighed 2 stone 11 lbs. He had good health, led an active life, could play many games besides being a good rider, a roller skater and as sharp as a needle in repartee where banter and pleasantry were concerned. He had slight moustaches which began to appear at age of 25, and a little hair on the pubes but his sexual organs were not fully developed. His voice was small and childish. The thyroid gland was present, there was no deformity or bending of bones, the limbs were quite symmetrical and their proportions to the head and trunk those of a man. Major James considers this also a case of infantilism, probably it may be classed as ateleiosis. (Bibl. No. 629, p. 445.)

Fig. 798. *Sutherland's Case.* As to I. 1, no statement is made. I. 2, was not small. She was said to have had a previous child, II. 1, who only weighed two pounds at birth, it lived 5 months though a seven months child. II. 2, aged $10\frac{1}{2}$, was $38\frac{7}{8}$″ in height. I. 2, said she had been frightened by a monkey when six months pregnant with him. The doctor who attended the confinement said he was a midget when born. His mother said he weighed nine pounds at the age of 3. He seemed well formed and no marked physical defects were obvious. The hands were somewhat blue and the finger tips a little clubbed suggesting some congenital defect of the heart but nothing abnormal could be detected. His mother said his actions were like those of a monkey. He was mentally backward, had not learnt his alphabet, and had not begun to talk till he was 4 years of age. His habits were mischievous and destructive. (Bibl. No. 435, p. 192.)

Fig. 799. *Case from Gentleman's Magazine.* This paper gives an account of the sudden death of I. 1, John Marshal, aged 62, who was long known in Leeds as Crutchy Jack. He was not more than 36″ in height and was the father of eight fine robust children, II. 1—8, four of whom survived him, the youngest, II. 8, being aged about 5. (Bibl. No. 56, p. 92.)

Fig. 800. *Meige and Allard's Case.* I. 2, married twice and had seven children by each husband, II. 1—14, but it does not state whether II. 14 was the child of the first husband or the second husband. I. 5, had four children, II. 15—18, all healthy. II. 14, and II. 15, were cousins-german but no statement is made as to whether they were paternal or maternal cousins, so which two individuals in Gen. I. were related remains uncertain. II. 14, was still alive, he had always been healthy and never had syphilis, but sometimes drank too much. He was of average height. II. 15, who had died 18 years before, was small in stature. She had had seven children, III. 1—7, of whom III. 7 was the only survivor. All the rest died young, two died at birth, three of meningitis, the last accidentally. The eldest, III. 1, who reached the age of 9, was strong and well-made. III. 7, aged 19, had been sent out to nurse as an infant and was said to have been neglected, he returned to his parents at age of 4, frail and difficult to rear. He did not grow. At age of 6 he had bronchitis and since then had a cough. His limbs were the limbs of a child, and he looked like a boy of 6. He had tuberculosis and Meige states he was certainly myxoedematous. His neck was very short, his shoulders high and his fingers deformed "en baguette de tambour." His intelligence however had not remained stationary, he had always been on a level at school with children of his own age. In mind he was 20 years old. (Bibl. No. 339, p. 106.)

Fig. 801. *Landau's Case.* I. 1, died from haemorrhage [Blutsturz]. I. 2, was alive and healthy as were also II. 1—6. II. 7, aged 29 from Breitenbach near Schettstadt, came to hospital for her first confinement. She could not say when she had learnt to walk, she had scarlet fever at the age of 4 and later had suffered from contraction of the chest and from cough. She was a small dwarfish weakly built brunette, height 129 cm., with poor muscular development. Her expression was childish, the chin retreating, the under-jaw small and the frontal protuberances prominent. The cranial portion of the skull was greatly developed so that the whole head appeared large in proportion to the body. The collar-bones were greatly curved and a distinct rosary existed. The upper extremities were very long relatively to the trunk, and when she stood upright they almost reached the knee. The hands were short and broad with short fingers. There was a moderate degree of genu valgum. The pubes and mons veneris were only slightly developed. During pregnancy she had severe bronchitis. At the beginning of the 10th month the child was born naturally, it was a male infant, small but mature and lived. He weighed 2000 grammes and his length was 45 cm. Landau apparently thinks this case is allied rather closely to cretinism and myxoedema and says this hypothesis is supported by the fact that the woman came from a neighbourhood where cretinism was known to be endemic. No enlargement of the thyroid body could be felt. The head measurements of child are given, but are without value being only to centimetres and not properly defined. *Measurements of Mother.* Stature 129 cm. Circumference of head (glabella-occiput) 51 cm.

Distance from nasion to vertex 10 cm.; from chin to nasion 11·5 cm. Length of upper limb from the acromion to tip of middle finger 60 cm.; upper arm from the acromion to the external condyle of the humerus 26·5 cm.; forearm from the external condyle to end of radius 19·5 cm.; hand from end of radius to tip of middle finger 14 cm.; middle finger 7·5 cm.; thumb 5 cm.; little finger 6 cm.; lower limb from the trochanter to the sole 70 cm.; from the trochanter to external condyle of femur 34 cm.; leg from the external condyle of femur to the external malleolus 31 cm.; foot from heel to tip of great toe 20·5 cm. Circumference of chest over the breasts 63—68 cm. (Bibl. No. 264, p. 6.)

Fig. 802. *Courtois-Suffit's Case.* This was probably a case of infantilism. I. 1, was tuberculous, I. 2, died of heart disease. II. 3, was tuberculous. II. 2, was also tuberculous, the disease began in the spinal column and ended in the lungs and she also suffered from Pott's disease. She died young—she had no miscarriage. Her husband, II. 1, appeared to be well, but looked frail and prematurely old, he was nervous and excessively excitable. He said he never had syphilis or any disease, and was medically examined with a negative result. The couple had three children, III. 1—3. III. 1, died young of croup. III. 2, died young of convulsions. III. 3, aged 23, was a seven months child, weak and frail and was reared with difficulty. He had measles slightly in early infancy but no other disease till he was 13. Till then his growth and intelligence had been normal. At the age of 13, he suffered from intense pain in his head and had to take to bed, and three days later became completely blind and remained so. Afterwards followed a series of symptoms which the doctors called "tuberculous meningitis," they lasted six months. He never had vomiting or constipation but had attacks of delirium followed by stupor. After six months they ceased but his lower limbs became completely paralysed and remained so for another six months. The paralysis passed off and he appeared cured but for eight years after had slight attacks, which finally became regular epileptic attacks. He suffered from intense thirst and from polyuria to a considerable extent. His physical development stopped at 13 and he looked like a boy of 13, but his intelligence was unaffected. The head was well developed and there was no bony deformity in trunk or limbs. The genital organs were those of a child of 13. His height was 130 cm. and his gait rather peculiar. (Bibl. No. 257, p. 588.)

Fig. 803. *Bourneville and Lemaire's Case I.* There was nothing to note in I. 1 and I. 2. II. 5, was neurotic, his brother, II. 4, was alcoholic. II. 5, married II. 6, who was also neurotic, and III. 2 was their son, no other child is mentioned. It is stated that a paternal cousin-german of III. 2, III. 1, committed suicide. III. 2, walked at age of 2 and spoke at 6. He had convulsions at 6, and suffered from "gatisme," at his entrance to the hospital. His mother had sent him to the country, said he had returned an idiot, and an onanist. He was born October 1873 and came under observation March 1885. The effect of the thyroid treatment was as follows: 1893,—weight 39·5 kilogs, height 140 cm. 1894,—weight 36·4 kilogs, height 141 cm. 1895,—weight 36·5 kilogs, height 142 cm. 1896,—weight 37·0 kilogs, height 144·5 cm. At age of 27 he had grown to the height of 149 cm. Hair and genitals were normally developed. (Bibl. No. 463, p. 387.)

Fig. 804. *Bourneville and Lemaire's Case II.* Nothing is said of I. 1. I. 2, was very neurotic, II. 2, was violent tempered, II. 3, alcoholic, II. 4, neurotic, II. 4 married II. 5, who was neurotic and hysterical, and suffered from migraines and visual hallucinations; her father, I. 3, was passionate and a brother, II. 6, died young of convulsions. There were five children of the marriage, III. 1—5, one of whom a boy, III. 4, died aged 6 of meningitis. III. 5, was born March 1878 after a troublesome pregnancy. She had her first tooth at age of 8 months, walked at 12 months and began to speak at 18 months. She had chorea at age of 1½ years and was passionate during childhood. Her first attack of hysteria was in 1892. She was at La Salpêtrière for nine months and came under Bourneville's care in February 1893. Her height was 143 cm. in February 1896 when thyroid treatment was tried, and in May 1897 her height was 143·5 cm. She left in 1898, married and had a child, IV. 1, aged 4 in 1904, and afterwards a miscarriage, IV. 2. The case is described as dwarfism without infantilism. (Bibl. No. 463, p. 387.)

Fig. 805. *Mary A. Smith's Case.* I. 1, and I. 2, were healthy. They had four healthy children, II. 1. I. 2, was aged 33 when II. 2 was born. The birth was only possible after perforation of the skull. The head of II. 2 was large and long relative to the body and limbs. The arms were not as long as the face was wide, the legs were short and thick, the abdomen enlarged. There were several anomalies in the skull. The femur was stumpy ("plump"), with extremely thick extremities and short diaphysis. The tibia and fibula were thick and short. A long description and very full table of measurements are given, only some of which are reproduced below. *Measurements.* Minimum (owing to injury) horizontal circumference of head 350 mm. Minimum (owing to injury) height of skull 135 mm. Length of L. arm from shoulder joint to end of middle finger 117 mm. Length of R. arm 122 mm.; hand 37·5 mm.; humerus 40 mm.; ulna 37 mm.; radius 33 mm.[1] Distance from antero-superior iliac spine to middle of knee 69 mm.; antero-superior iliac spine to ankle 130 mm.; ankle to tip of great toe 46 mm. Length of the sole 57 mm. Distance from the highest point of the end of head of femur to the lowest point of the internal condyle 55 mm. Length of L. tibia from the internal condyle to inner edge of lower end 48 mm.; to outer

[1] These measurements fail to give total length of arm.

edge of lower end 35 mm. Length of fibula 40 mm.; foot from posterior end of calcaneus to the point of the great toe 56 mm.; clavicle, markedly S-shaped, 34 mm. (Bibl. No. 179, p. 92.)

Fig. 806. *Morley's Case.* I. 1, a small Scotchman, aged nearly 60 and 2′ 6″ in height, was exhibited at Brookfield Market about 1698. He had been married several years and had two sons, II. 1—2, one of whom used to accompany him. He had kept a writing school at one time. (Bibl. No. 118[b], p. 321.)

Fig. 807. *Colegate's Case.* This case was sent by Colegate to Barlow who described it. No statement is made with regard to I. 1. I. 2, was healthy and had given birth to five healthy children, II. 1—5. II. 6, the sixth child, was extremely blue at birth and only breathed a few seconds. The limbs were stunted in length; the humerus showed relatively large epiphyses and a short stout shaft. The radius showed a remarkable sigmoid curve, an exaggeration of the normal one. The hand was short and stunted. No measurements are given but there is a long description of the brain. (Bibl. No. 210, p. 459.)

Fig. 808. *Uhthoff's Case.* I. 1, and I. 2, were alive and healthy; they had six children, II. 1—6, five were normal, II. 5, aged 15, being 1·66 metres in height. II. 6, aged 14, looked like a child of 9 or 10. She had been perfectly healthy till the age of nine and then got inflammation of the lungs and her growth stopped. She weighed 52 "Pfund" and was 131 cm. in height. The thyroid gland had almost disappeared, there was no trace of myxoedema and her intelligence was intact. She had temporal hemianopsia with descending (absteigende) atrophy of the optic nerves, and hemianoptic reaction of the pupils. The L. eye was quite blind. (Bibl. No. 335, p. 462.)

Fig. 809. *Fischer's Case.* No statement is made with regard to I. 1. I. 2, aged 34, had always been healthy and had never suffered from rachitis or syphilis. She had had three children, II. 1 was born 5 months too soon. II. 2, aged 1¼ years, showed evident symptoms of the beginning of rachitis, its joints were swollen and its lower extremities curved. The third child, II. 3, was born at term, but died in consequence of long parturition. She weighed 3200 grammes, length 40 cm. The extremities were very short, thick and deformed with curved bones, they looked like fishes' fins. The head was too large, the neck short and thick, the chest in the neighbourhood of the fourth rib very narrow, and the genital organs normal. The skin felt rough, and in places, especially on the extremities, was in thick folds. The lower limbs were very short and greatly curved, both feet being in varus position. The upper limbs were short and thick, the nails extending beyond the tips of the fingers. There was a distinct rosary. The xiphoid process was split in the middle. The dorsal spine was slightly scoliotic to the left, the lumbar spine very scoliotic to the right. The humerus was curved and its diaphysis and epiphysis were thicker than in normal cases. The radius and ulna were considerably curved, but the hands were normal. The thighs were greatly twisted, so that the patella faced laterally instead of anteriorly without being dislocated; the head and lower epiphysis were much enlarged. The tibia and fibula were so twisted that the concave edges were turned towards each other and the greatest distance between them was 1·1 cm. There was no abnormality in the bones of the foot. Measurements are given. (Bibl. No. 153, p. 46.)

Fig. 810. *Hertoghe's Case.* Of I. 1, and I. 2, no statement is made. II. 1, aged 19, and II. 2, aged 18, had had such serious affections of the throat when II. 1 was aged 21 months and II. 2 aged 7 months that they were not expected to live. They did not walk till they were 7, and were only sent to school at age of 12. II. 2, learnt to read and write but II. 1 objected to learning. They were very slow in their movements and to the superficial glance looked more of the rickety than of the myxoedematous type, to which in reality they belonged. Their abdomens were large and they had umbilical hernia. II. 1, had subclavicular tumours, flat nose, thick lips, and large cheeks, the eyelids were very heavy, the eyebrows had almost disappeared and the hair was very thin. II. 2, had a similar but slightly worse appearance but his eye was livelier and he was more intelligent. They received thyroid treatment. At the beginning of the treatment, 18 August, 1894, II. 1 measured 113 cm. and weighed 27·10 kilogs; at end of treatment 23 July 1895, he measured 125 cm. and weighed 31·00 kilogs. II. 2, in same period increased in height from 109·5 cm. to 118 cm., and in weight from 25·30 kilogs to 28·63 kilogs. (Bibl. No. 304, p. 912.)

PLATE LVIII. Fig. 811. *Mason's Case.* I. 1, I. 2, I. 3 and I. 4, were of the well-to-do farmers' class, all alive and well, their parents had all lived to be old people. II. 5, knew of no case of consumption or scrofula in his family. The family of II. 4 were all healthy except an elder sister, II. 2, who came from North of Ireland to the States, married, and died of some wasting disease after her first confinement. II. 4, was healthy, married when aged about 22, and III. 2 was her first child. The labour was normal. The head presented a square appearance at birth and properly speaking there were no parietal bones. The circumference of the head was 12½″. The deficiencies and deformities in the trunk and extremities were more marked on the left side. The clavicles were deficient in thickness, the left broken in the middle. The cartilages of the ribs at their junction with the sternum were moderately enlarged. Both humeri were curved, the convexity being inward, the ends were enlarged, the middle portions deficient and the left was broken. The abdomen was prominent, and was 11″ in circumference. The femora were curved with convexity outward. In all the long bones the ends were enlarged and the middle portions reduced. The tibiae were sharply curved, with convexity to the front. The child was tongue-tied. It lived for 3½ days. In the plate accompanying the memoir the limbs look short; no other measurements are given. (Bibl. No. 292, p. 670.)

Fig. 812. *Patel's Case.* I. 1, was very alcoholic but died old. I. 2, died very aged, she was always well and had no miscarriage. Seven of their children II. 1—7, were alive and normal and several of them

had normal children, III. 1. II. 8, Pierre Guilhaumont the youngest, aged 48, was born in the Canton Riotord in Haute Loire, he was deformed and his height was 1·08 metres. He had never heard of any dwarf in the family. He had heard he was born so frail and deformed that it was thought he would never live. He had always been small, but grew a little between 15 and 20, not since. He had had no illness till age of 48. He had gone to school for two years and could write, he answered questions clearly but told lies. He said he never touched alcohol yet he was intoxicated every day. He had a large head and short thick neck. The arms seemed long and slender relatively to the lower limbs and the rest of the body. He had marked scoliosis and lordosis. The thorax was globular, projecting in front, the abdomen large and protuberant. The thighs were very oblique. The femora are said to have met at the level of the internal condyles (Les fémurs se rejoignent au niveau des condyles internes), but the significance of this is not quite obvious. The legs were straight and the feet large. Further on the account states that the upper and lower limbs were normal and the hands well formed. *Measurements.* Head: maximum circumference 55 cm.; antero-posterior arc (from the glabella to external occipital protuberance) 30 cm.; transverse bi-parietal arc 27 cm.; maximum longitudinal diameter (the glabella being taken as fixed point) 18 cm.; maximum transverse diameter 15·5 cm.; cephalic index 85. Circumference of neck 31 cm. Length of upper arm 24 cm. Circumference of upper arm 19 cm. Length of forearm 21 cm. Circumference of forearm 17 cm. Length of hand 14 cm. Breadth of hand 8 cm. Total length from the acromion to tip of middle finger 57 cm. Length of spinal column from the 7th cervical to base of sacrum 25 cm. Circumference of thorax under the axillae 73 cm.; at nipples 75 cm. Sternal circumference 75 cm. Lower limb: length of thigh from great trochanter to external condyle 25 cm. Circumference of thigh at the middle 33 cm. Distance of inguino-crural fold from external condyle 15 cm. Length of leg from the external condyle to external malleolus 23·5 cm. Total length from the trochanter to the ground 53 cm. Length of foot 18 cm. Breadth of foot 11 cm. It may be noted that the total length of upper limb given does not tally exactly with sum of lengths of the different parts. The right testicle was normal, the left small and defective. Patel says this dwarf had a strong resemblance to the dwarf Anatole described by P. Marie whose height was 1·22 metres (the Anatole of Bibl. No. 371, not the Claudius of our Fig. 674), but Guilhaumont was not achondroplasic, his spinal column was not straight but curved laterally, it described an Italian S exactly; had it been straight the height would probably have been increased by 15 cm. This deformity caused projection of the shoulder blades and the odd configuration of the thorax and abdomen. The upper segments of both limbs were shorter than the lower in Marie's case. (Bibl. No. 391, p. 301.)

Fig. 813. *Boquel's Case.* I. 1, aged 55, was alive and healthy. I. 2, aged 50, was also alive and healthy. She had had eleven children and two miscarriages, II. 1—13. II. 13, aged 25½, began to walk at age of 7½. She had had bronchitis at age of 10. Her height was 132 cm., and she was not vigorous. It was her first confinement. Caesarian section was performed and a male child, III. 1, extracted who weighed 3210 grammes. Mother and child lived. No measurements. (Bibl. No. 377, p. 416.)

Fig. 814. *Arendes' Case.* In this family (Achtermeier) there was no case of dwarfism of either paternal or maternal side except that I. 1, a great grandfather of the mother IV. 2, had been 'very small.' IV. 2, and IV. 3, were normally built, of medium height, of good health, and had never been seriously ill. They were labouring class people at Neuendorf near Duderstadt and had six children, V. 1—6. Three of these children, V. 3 and V. 5—6, had, according to IV. 2, developed normally; V. 2 and V. 4 were dwarfs. V. 1, died aged 3 of convulsions; he was rickety. V. 3, T. A., aged 6, born January 16, 1879, was 112 cm. in height. He weighed 36½ Pfund (18·25 kg.). He had learnt to walk at 1¼ years old and had got his teeth on the right side then; evident symptoms of former rickets existed. He had all his milk teeth, some were carious. He had caput quadratum, circumference of head 51·3 cm., a well marked rickety rosary; also thickening of the epiphyses. The tibiae were somewhat curved. Otherwise the boy was healthy and mentally well developed. The sutures and fontanelles were closed and the four first molar teeth had appeared. V. 5, W. A., aged 2, born 14 April, 1883, was 86 cm. in height, weight 12·25 kg. He showed signs of rickets, enlarged epiphyses, but no curvature of his legs. He had all his milk teeth well developed but two. These showed slight rachitic changes. He had a large square head, with the sutures all closed. Had learned to walk early. V. 6, C. A., about 1 year old, born 18 November, 1884, had begun to walk at 1¼ years old, could speak a few words, had all her incisors and the first molar teeth above and below on both sides. The epiphyses were slightly enlarged and the skull showed traces of rickets, the fontanelles and sutures were closed. Otherwise she was healthy, walked well, weight 7·5 kg. and was 79 cm. in height. V. 2, Franz Achtermeier, born 12 May, 1877, was aged 8 when he came to the hospital, his brother, V. 4, Karl Achtermeier, born 18 January, 1881, was aged 4. Both children were normally born and of normal size at birth. They learnt to walk at the end of their first year, and never had curved legs or fractured bones. They had teeth at the end of their first year and learnt to speak at the right time, they ate very little but were cheerful and fond of play. V. 2, at age of 2½ had stomatitis (thrush) and quinsy (Rachenbräune) at the same time. In his fourth year he was ordered daily seawater baths. Otherwise he had been healthy. While suffering from prolonged stomatitis he lost his upper incisors and shortly afterwards his other teeth became very bad. V. 4, had apparently never been ill, his teeth were sound for about a year after eruption and then became bad.

The bodies of these boys appeared well nourished, their extremities were remarkably small but apparently in the right proportion to the size of the body—with the exception of the head. Relatively their heads were the largest part of their bodies and especially out of proportion to the chest. They were of caput quadratum form with the vault of the skull flattened and the sutures and fontanelles depressed. The tubera frontalia and parietalia were very prominent. The forehead was much arched, and the back of the head projected noticeably. The facial bones were all too small in comparison with the upper part of the head. In the case of V. 2, the length from the nasion to the point of the chin was 8 cm., and from the nasion to the middle of the large fontanelle 13 cm. The sagittal suture was not closed and was $6\frac{1}{2}$ cm. in length. The frontal suture was also open. V. 4, had the great fontanelle open, it was 2 cm. in length and 1 cm. in breadth. The lesser fontanelle was completely closed, as also the sagittal suture. The hair of both was thin, fair in colour and felt soft. The neck was very short, the circumference of the chest was less than the periphery of the skull. There was no spinal curvature. At the union of the rib cartilages and bones the characteristic rosary beads could be felt, and even seen, though indistinctly. The bones of the extremities were on the whole straight, only the tibiae were somewhat curved. A slight thickening of the epiphyses was noticeable in the forearm. The intelligence of the boys was on the whole good. The elder, V. 2, had been at school for four weeks and according to the father had made good progress. The younger, V. 4, seemed to have more intelligence than his brother. They often slept with half opened eyes. In order to show the growth of the dwarfs, three sets of measurements were taken during their residence at the hospital of which we reproduce the first and last.

Measurements:

	Dwarf V. 2		Dwarf V. 4	
	June 1885	March 1886	June 1885	March 1886
Weight	9·375 kgs.	11·250 kgs.	7·000 kgs.	8·125 kgs.
1. Length of head from vertex to mental point ...	18·0 cm.	18·0 cm.	17·5 cm.	18·2 cm.
2. Length of neck from mental point to upper edge of sternum	6·0 „	6·0 „	5·8 „	5·8 „
3. Length of sternum to extremity of the xiphoid process	10·2 „	10·3 „	7·7 „	8·0 „
4. Length of abdomen from the tip of the xiphoid process to the upper edge of the symphysis ...	18·0 „	19·0 „	17·5 „	18·5 „
5. Total length from the top of upper edge of the pubic symphysis to middle point of internal malleolus	29·5 „	29·7 „	26·5 „	27·1 „
6. Distance of the middle point of the internal malleolus from the sole	3·4 „	3·4 „	2·8 „	2·8 „
7. Transverse diameter of the head above both auricles	14·7 „	14·9 „	13·8 „	13·8 „
8. Direct diameter from the greatest arch of the forehead to the occipital protuberance ...	17·3 „	17·4 „	16·2 „	16·5 „
9. Circumference of head	51·3 „	51·5 „	47·5 „	48·0 „
10. Circumference of chest at nipples	45·2 „	49·0 „	41·2 „	45·0 „
11. Antero-posterior diameter of chest from the middle of the sternum to the spinous process of the 5th dorsal vertebra	12·8 „	13·0 „	11·7 „	12·2 „
12. Transverse diameter of chest	13·1 „	14·5 „	12·2 „	12·8 „
13. Breadth of hips (cristae)	13·2 „	14·1 „	11·7 „	13·1 „
14. Breadth of shoulders	15·0 „	17·0 „	14·2 „	16·0 „
15. Vertex to border of orbit	12·5 „	12·5 „	12·2 „	12·3 „
16. Larynx to axilla	10·5 „	10·6 „	8·5 „	8·5 „
17. Axilla to crest of hip-bone	13·6 „	13·6 „	16·0 „	16·2 „
18. Upper limb	31·6 „	31·6 „	29·5 „	29·6 „
19. Upper arm	12·0 „	12·0 „	10·8 „	10·8 „
20. Forearm	11·1 „	11·1 „	10·3 „	10·3 „
21. Length of hand	8·5 „	8·5 „	8·2 „	8·2 „
22. From crest of hip-bone to the knee	22·0 „	22·3 „	21·5 „	21·7 „
23. From the knee to the sole of foot	19·2 „	19·2 „	18·0 „	18·3 „
24. Length of the foot	12·4 „	12·4 „	10·4 „	10·4 „

Stature	June 1885	March 1886	Normal child of same age
V. 2	75·0 cm.	76·0 cm.	116·0 cm.
V. 4	69·0 „	70·0 „	93·0 „

These measurements are taken from pp. 14 and 24 of thesis, the latter presumably being those of March 1886. Neither is in agreement with the sum of 1 to 6 above, which presumably were not vertical but arcual lengths. (Bibl. No. 227.)

Fig. 815. *Ender's Case.* No statement is made with regard to I. 1 and I. 2. II. 2, aged 27, was a seamstress in poor circumstances with a pale old-looking face and marked rachitis. Her height was '3 Fuss 3 Zoll.' The under jaw extended beyond the upper jaw. She walked with difficulty and took short steps. Both thighs were much curved anteriorly and the lower part of the legs was similarly curved inwards. The knee-joints could not be straightened. The arms were also curved. The spinal column and the rest of the trunk were normal. Caesarian section was successfully performed, the child, III. 1, was strong and healthy and the mother recovered. *Measurements of pelvis.* Diagonal conjugate 3″ 4‴; external conjugate 6″. Antero-posterior diameter of pelvic space 3″ 6‴; of pelvic inlet 3″. Interspinous 8½″. Intertrochanteric 10″. Pelvic circumference 30″. Distance between tubera ischii 2″ 9‴. From tip of coccyx to lower edge of symphysis pubis (pelvic outlet) 3″ 3‴. Distance of L. iliac crest from the symphysis pubis 3″ 6‴; R. 3″ 9‴. Height of posterior pelvic wall 4″; of pubic arch 1″ 4″. (Bibl. No. 133, p. 43.)

Fig. 816. *Ornstein's Case.* I. 1, was dead. I. 2, was alive. II. 2, was alive and married; these three were all of middle height and had no deformity. II. 3, Hadsi Konstantinu, born at Lemessos on west coast of Cyprus, aged 39, was a beggar in the streets of Athens. He had a large and quadrate head, prominent forehead, long and strong trunk, good teeth, fairly thick hair, moustache and whiskers, and a short neck. The penis was from 5—6 cm. long and of normal circumference, the testicles were about the size of sparrows' eggs. He had no sexual feeling. His crooked legs were short and muscular, both thighs and legs having the curvature inwards. He had a slight degree of pes varus, and short and fleshy hands and fingers. The plate shows that with hanging arms the tips of the fingers just reached the tops of the thighs. *Measurements.* Height 118 cm. Circumference of chest 90 cm. Total length of arm 42 cm.; leg 35 cm. Span 102 cm. Horizontal circumference of skull 61 cm. II. 3, looks in plate of the characteristic achondroplasic type. (Bibl. No. 272, p. 541.)

Fig. 817. *Nijhoff's Case II. Familiar Rickets.* I. 1, and I. 2, were normal. Of their five children, three were normal, II. 1—3. II. 4, height 96 cm., was a rachitic dwarf, a merchant. It does not state whether he was married or not. II. 5, aged 40, was also a rachitic dwarf and single. Her height was 86 cm. The pelvic measurements were: Interspinous 18 cm. Intercristal 17 cm. Intertrochanteric 20·5 cm. External conjugate 14 cm. Diagonal conjugate 7 cm. She became *enceinte* and caesarian section was performed 9. 11. 1900. The mother recovered, the child, II. 1, was dead. He weighed 3890 grammes and his length was 54 cm. (Sent by the kindness of Professor Nijhoff of Groningen: see Plate NN (82)—(84).)

Fig. 818. *W. Adams' Case.* No statement is made with regard to I. 1. I. 2, aged 41, was a healthy woman who had ten healthy children, II. 1—10. The eleventh, II. 11, was born with the arms shorter than natural and the legs shortened and twisted. It must have been stillborn or died at birth, as the specimen was presented by Dr W. H. Williamson to the (Royal) College of Surgeons (of London). The mother said she had seen a cripple similarly deformed when pregnant. (Bibl. No. 146, p. 263.)

Fig. 819. *Paal's Case.* Of I. 1, no statement is made. I. 2, was told by her father that as a small child she had curved legs, and only began to walk at 3 years of age. Later she was always healthy. She had five children, II. 1—5. II. 1, was stillborn and its arms and legs were not properly formed "auch nicht recht ausgebildet gewesen sein." II. 2, lived for ¼ year. According to the doctor who visited it when vaccinated it had club feet. The mother said it had always been anaemic. II. 3, was normally developed, and looked strong and healthy. II. 4, was stillborn at term and had the same curved and short extremities as II. 1 and II. 5. II. 5, was stillborn, according to the mother it was a seven months child. *Measurements.* Length of body 34 cm. Circumference of head 30·5 cm.; horizontal diameter 9·25 cm.; bi-parietal diameter 7·75 cm.; bi-temporal diameter 7 cm.; greater oblique diameter 10 cm.; lesser oblique diameter 8 cm. There was no craniotabes and the fontanelles were small. The skin of the whole body was firm and much swollen, especially on the extremities. The long bones of the extremities were much curved. The humerus was curved, 4 cm. in length, and its whole diaphysis was completely ossified. The radius and ulna were also ossified, the latter formed a half circle whose diameter from the olecranon to the styloid process measured 2 cm. The lower extremities were Ω-shaped with decided pes varus. The distance of the greater trochanter from the external malleolus was 7 cm. (Bibl. No. 286, p. 29.)

Fig. 820. *Sonntag's Case.* I. 1, was tall and robust and had always been healthy, he is called the "amicus" of I. 2, so probably they were not married. I. 2, aged 30 in 1844, was of medium height, slight build and healthy appearance and had always enjoyed good health. No certain symptom of rachitis could be found in her, and she said that she and her eight brothers and sisters, I. 3, had all walked when 1 year old. Of her children, II. 1, born 1837, came 14 days too soon but was well formed. II. 2, born 1841, was stillborn and had to be extracted. Its birth took place 52 days too soon. It showed no deformity. II. 3, born 1843, was a well-formed seven months child. II. 4, a male infant, was born alive in 1844, 43 days before its time, and died shortly after birth. The child weighed "unciarum XXX, drachmarum IV et granorum XV." The skin was soft, reddish, and covered with lanugo. The head was very large, almost equalled the rest of the body in length and was covered with hair. The neck was very short, the thorax very short, arched and compressed at the sides (pigeon-breast), the abdomen was distended. The umbilicus was situated "duo circa digitos" above the pubic symphysis. The arms hung near the trunk and were very like the front fins of seals; all the limbs were very short but the hands and feet were beautifully formed. The distance from the middle of the wrist to the tip of the third finger was half the length of the upper extremity. The external genital organs were normal. *Measurements.* Total length 9″ 4‴. Length from vertex to perineum 6″ 10‴; perineum to soles of feet 2″ 6‴. Vertical diameter of head from vertex to foramen magnum 3″ 2‴. Transverse diameter from one frontal eminence to the other 2″ 8‴. Oblique diameter from the chin to the middle of the lesser fontanelle 3″ 3‴. Diameter from the nasion to the external occipital protuberance 2″ 11‴. Length of upper limb 2″ 2‴. Breadth of metacarpus 8‴. Length of lower limb 2″ 6‴; foot 1″ 3‴. Maximum breadth of foot 10‴. Length of clavicle 1″; humerus from the head to the external condyle $10\frac{2}{3}$‴; to the internal condyle 9‴; radius 7‴. Breadth of ulna at elbow 5‴; near wrist $3\frac{1}{2}$‴. Length of femur 9‴; tibia $6\frac{1}{2}$‴; fibula 5‴. The bones of the extremities were curved and the soft palate was cleft but there was no hare-lip. A plate of the child is given. (Bibl. No. 87, p. 1.)

Fig. 821. *Symington and Thomson's Case.* I. 1, aged 26, was healthy. I. 2, who was a year or two younger, was said by the doctor to be epileptic. She had two healthy children, II. 1—2, aged 4 and 2. There was no history of any deformity in the family. About three months before the birth of II. 3, the mother had been violently assaulted by another woman. II. 3, a female foetus, weighed 8 lbs. 2 oz. at birth and had very short, thick extremities marked by deep transverse sulci. The head and trunk were nearly of normal size. The upper part of the head was somewhat enlarged and the fontanelles abnormally open. *Measurements.* Total length from vertex to heel 40 cm. Length from the vertex to the perineum 36·5 cm.; to the umbilicus 28·5 cm.; from finger tip to finger tip with arms abducted at right angles to trunk 28 cm. Total length of arm measured from the base of the axilla to the finger tips 7·6 cm.; lower limb measured from the centre of Poupart's ligament to heel 8·7 cm. Bi-parietal diameter of head 10·8 cm.; occipito-frontal diameter of head 12·3 cm. Circumference of head 20·5 cm. Length of humerus 3·6 cm.; radius 2·7 cm.; ulna 2·8 cm.; metacarpals 1·2 cm.; femur 4 cm.; tibia 3·8 cm.; fibula 3·3 cm.; metatarsals ·8 cm. (Bibl. No. 274, p. 237.)

Fig. 822. *Brodowski's Case.* This case is quoted in Virchow and Hirsch's *Jahresbericht* for 1874, and was not seen in the original. Brodowski exhibited two dwarfs before the Warsaw Society of Medicine. They were the youngest children of I. 1, a man of medium height and their older brothers, II. 1, were of normal size. II. 2, aged 20, height 93 cm., weighed 37 "Pfund." II. 3, her brother, aged 17, height 90 cm., weighed 39 "Pfund." Their growth was normal till the age of 8, when it ceased. They looked like children aged 8, but their heads were larger. They had no sexual feeling, and their strength was that of children. They were not idiots. (Bibl. No. 152, p. 300.)

Fig. 823. *Hecker's Case.* No statement is made with regard to I. 1. I. 2, aged 23, was a dwarf with exceedingly short extremities. She had been born with short extremities, learnt to walk at age of $1\frac{1}{2}$ years, but at the eruption of each group of teeth, she always forgot how to walk for two or three months. She was so intelligent she decided to become a teacher. She came to hospital for her first confinement. Caesarian section was performed and twins were extracted, a boy, II. 1, weighing $4\frac{3}{16}$ "Pfund" and a girl, II. 2, weighing $4\frac{1}{2}$ "Pfund," they were both unusually well developed. The children were alive, the mother died 40 hours after the operation and the skeleton came into possession of the Obstetric Clinic. (Photograph given.) *Measurements of skeleton.* Total length 131 cm. Length of spinal column 59·75 cm. Total length of arm 48 cm. Length of humerus 18 cm.; ulna 17·5 cm.; radius 15 cm.; hand 15 cm.; scapula 14 cm. Breadth of scapula 9 cm. Total length of leg 54 cm. Length of femur 26 cm.; tibia 21·5 cm.; fibula 24 cm. Height of foot 6·5 cm. Length of foot 18·5 cm. Circumference of skull 51 cm. (Bibl. No. 131, p. 73.)

Fig. 824. *Guéniot's Case.* I. 2, was very small and deformed, but her height is not given, nor does Guéniot state positively that she was a dwarf. Caesarian section was twice performed on her, the first time in 1891 when a well-developed girl, II. 1, who was alive $3\frac{1}{2}$ years later, was extracted. The second operation took place about three years later, resulting in a boy, II. 2, weighing 3000 grammes, who also lived. (Bibl. No. 289, p. 16.)

Fig. 825. *Christopher's Case.* Only a few lines of description of this case were given during the discussion on Morse's paper. No statement was made with regard to I. 1 and I. 2. With regard to II. 1—3, Christopher said, "I have charge of a family of three such children that I have been watching for three or four years. They are not idiotic, but do not do well at school. Dr Walker has charge of them for me and thinks them rachitic but I cannot think so." The fingers were very short, very broad and nearly of equal length, the feet short and legs very short. Their appearance was suggestive of cretinism. (Bibl. No. 416, p. 577.)

Fig. 826. *Chaussier's Case.* No statement is made with regard to I. 1. I. 2, aged 33, was very strong and had always been healthy. She came to hospital for her fifth confinement. It states she had four children previously but does not say whether they were healthy or not. The birth was quick but the child, II. 5, only lived 24 hours. It weighed 2508 grammes and its total length was 30·9 cm. The head was long and large, about one-third the total length of the body, all four limbs were short, thick and stunted, and the surface was deeply furrowed. The bones of the limbs were shorter but larger and thicker than normal, more or less curved, and all showed several fractures, some united and some not. The spine, pelvis and jaws showed nothing abnormal, but the ribs had many fractures. There were 70 fractures in the ribs altogether. (Bibl. No. 57, p. 306.)

Fig. 827. *Rohrer's Case.* I. 1, was a tall, strong man, he had had syphilis. I. 2, died aged 45 of phthisis, she was a tall woman. II. 1, aged 8 weeks, died of convulsions. II. 2, aged 22, slim and tall, was 171 cm. in height. II. 3, aged 20, was 120 cm. in height. He was normal at birth and developed normally. At the age of 2 he fell and hit his head against a chamber utensil, at 11 he fell down the stairs of a cellar and in the same year got a blow on the head from an axe, which resulted in long continued suppuration. Since then he had suffered from incontinence of urine, and his growth and development had stopped. His voice was a boy's soprano. The senses were normal but he was somewhat myopic. His sexual organs were altogether undeveloped, like those of a boy of 6 or 7 years of age. There was no trace of pubic hair. (Bibl. No. 222, p. 197.)

Fig. 828. *Menzies' Case.* Of I. 1, nothing is known. I. 2, died aged 95 years of "apoplexy." II. 2, is stated to be short and stout, and in good health at the age of 70 years. II. 3, died aged 53 years of "paralysis," after a severe illness of five weeks' duration, but had been an invalid for seven years before this. Siblings of these, II. 1 and II. 4, exist but unknown. II. 5, is stated to have died aged 34 years, cause unknown. Stated to have been "tall." II. 9, the youngest of her family, is now aged 69 years 9 months. She is quite healthy and strong. She is taller than her daughter, III. 20, whose height is 5′ 6″. II. 6, 7 and 8, were all about the same height as II. 9. II. 6, died aged 58 years, cause unknown. III. 1—9, are all of ordinary growth and as far as is known none of them suffer from illness of any kind. III. 1, is unmarried. The others have offspring as shown, IV. 1—5. These are all well-grown, healthy children. III. 10, cabinet maker, aged 37 years, of height 5′ 5″ without shoes. He is said to be "tired," but shows no other peculiarities. No history of syphilis and no signs of it. Does not remember having had any illness. Physical examination negative. III. 20, aged 35 years, of height 5′ 6″, a fairly strong and healthy woman, not of neurotic type. She does not remember ever having had any illness. Physical examination negative. She is the youngest of her family. III. 12, died aged 29 years, at a confinement. She is described as "short," but height unknown. To judge from her photograph, which was shown, she was a small and somewhat "wizened" woman. But she was not deformed or in any way diseased. III. 14, and 15, died young, cause unknown. They "were of ordinary size." III. 16, was a soldier who died aged 37 years of "bronchitis." He was a "short" man of about the same height as his sister, III. 20. III. 18 and 19, died young, cause uncertain, but it is thought that this was "bronchitis." They were of ordinary growth as far as is known. All the other members of this generation were of ordinary size and growth and, as far as is known, quite healthy. In the IVth generation IV. 17, 18, 19, 20 and 21 are of ordinary growth and, as far as is known, quite healthy. IV. 22—27, all died young, it is thought of "bronchitis." They were of normal growth. IV. 28, aged 21 years, is a corporal in the army. IV. 6, aged 13 years 7 months, is short and light for age, but is quite healthy and shows no abnormalities of growth. He has a marked left internal strabismus. He has never had any illness of any kind, but was run over at the age of 10 years. He is bright and intelligent. He is in the sixth standard and "does very well at school." His hair and eyes are light. IV. 7, aged 12 years 3 months. Short and light for age, and of distinctly "stumpy" type. She is a healthy, bright, intelligent and amiable child. She shows no abnormalities of growth. She has had chicken-pox but no other illnesses. She is in the seventh standard at school where she "does very well." She has light hair and eyes as her brother has, while both parents are "black." IV. 8, aged 10 years and 5 months, shows a R. internal strabismus but no other peculiarities. She is half a head taller than her sister. She also is a quite healthy, bright, intelligent and amiable child. She is in the third standard at school. She "does well at school but is not so bright as the other two." She has never had an illness. She has dark hair and eyes like her parents. IV. 9, aged 9 years 5 months is rather short and light for age but shows no abnormality beyond a R. internal strabismus. He is in the first standard at school. He is a perfectly healthy child, fairly bright and intelligent. He has never had any

illness. He has dark hair and eyes like his parents. IV. 13, died aged 9 months, of "convulsions" after diarrhoea and vomiting. IV. 15, died aged 1 year 3 months, of "convulsions." IV. 16, died aged 1 month, of "bronchitis." IV. 14, is aged 2 years 3 months, shows no abnormalities and has had no illnesses; a healthy child. Hair and eyes light. IV. 10, E. P., is aged 8 years 2 months. Height 31⅛". Weight 11 kilogs. He shows a marked abnormality of growth. He was born at full term after an uneventful pregnancy and a normal confinement. There was no hydramnios. He was very small at birth, very much smaller than the other children. He seemed to grow slowly until about 4 years of age, when growth seemed to cease; for the last four years he has hardly grown at all, but seems to have begun to grow again during the last few months ("I know this because until then he could not see on to the table; now he can"). Apart from his small size he showed no peculiarity at birth. He had no "snuffles." Breast fed for about five months. Bottle fed until about 14 months. First tooth appeared at seven months. Began to talk at 18 months and to walk at 3 years. He has never had any illness except diarrhoea when teething. "He is a very hearty eater and never ill." He has been at school for three years but is only in the first standard, the same as his sister, nearly two years younger. He does his drawing well but otherwise does not learn with much aptitude[1]. He romps about with other children of his age, but on account of his small size, etc., it is proposed, after another trial of four months at the general school, to send him to a special school, if he does not improve. He is quite reliable in such matters as buying such articles as bread, etc. for his mother. Height 2′ 7⅛". A relatively large, high quadrate skull of 19⅝" circumference, with bulging forehead, prominent frontal and parietal eminences and relatively small face, the bridge of the nose is very markedly depressed but not broadened; the nose itself is small and straight. The mouth is kept shut, the tongue is not protruded. The proportions are normal, the limbs and spine show no curvatures and there is no rosary or other evidence of rickets. The hands and feet are perfectly formed in every way. The hair and skin are smooth and not dry or scaly, the subcutaneous tissues not increased. The thyroid gland is palpable and is not enlarged. There is no evidence of syphilis or other constitutional disease and physical examination shows that all viscera are normal. He is markedly more sallow than his brothers and sisters and has dark hair and eyes. The extremities of the fingers extend as low as the junction of middle and upper third of the thighs. *Measurements.* Total height 31⅛". Lower extremity from antero-superior iliac spine to internal malleolus 15⅝". Length of femur from antero-superior iliac spine to articular margin of internal condyle 9"; tibia from internal condyle to internal malleolus 6⅝". The proportions are thus normal for age. Upper extremity from acromion angle of scapula to extremity of styloid process of radius 10½". Length of humerus from acromion angle to external epicondyle 5½"; radius from external epicondyle to extremity of styloid process 5". The proportionate length of segments to one another is thus normal. (Measurements were made between aniline pencil marks over these points.) Maximum circumference of chest 20". The mid-point between the vertex and the soles of the feet lies above the upper border of the symphysis one third of the distance between this point and the umbilicus as in the normal for age. All the limbs are fairly stout and well formed. There is no muscular wasting. He is quite intelligent and good tempered. He and his brother, IV. 12, and sister, IV. 11, spend a great deal of their time in laughter. But he has obviously less "vitality" or spontaneity and is more "backward" than they are. Dentition about normal for his age (thus both permanent molars are present in the upper jaws) but many teeth carious. Palate very broad and flat (not high-arched) and as in the normal children seen. IV. 12, W. P., aged 5 years and 4 months. Shows much the same features as his brother, IV. 10, but to a less degree. Thus his height is 34⅛", or three inches more than his brother who is nearly three years older than he. The maximum circumference of his head is 20" or ⅜" greater than that of his elder brother, and his maximum chest circumference is ½" greater. Born at full term after an uneventful pregnancy and a normal labour. No hydramnios. He was of ordinary size and very fat at birth. No "snuffles." Breast fed nine months. First tooth at 5½ months. Began to talk at 18 months and to walk at 3 years 7 months. "He did not seem to grow after he was 3 years of age." He is quite intelligent[2], but "he does not run about like the other children, but likes to sit down all the time." Attends school and does fairly well there. He has only once been ill, when, at the age of 1 year 9 months, he "had the yellow jaundice, rickets and diarrhoea," and was an in-patient at the London Hospital for 7 weeks 3 days. Dentition normal for age, teeth sound. Palate higher and narrower (as in IV. 11) than in the normal children. He presents all the features shown by IV. 10, but to a less degree. A relatively large, high, quadrate skull of maximum circumference of 20". Bridge of nose depressed, but not flattened; nose short and straight. Face comparatively small; but this feature much less marked than in his brother. He has a distinctly "cretinoid" appearance. The mouth is kept shut and there is

[1] His teacher reports of him: "Very backward for a boy of 8 years. He is only up to the standard of a child of 5 years. His speech is *very indistinct*. He is fairly good at numbers, adding such numbers as 6 and 5 without effort. He takes a keen interest in stories and can reproduce them. During the last year he has much improved and now seems to take a real interest in his work."

[2] His teacher reports of him: "He has only attended school for 4 months. He makes very feeble attempts at writing and drawing. He can count a very little. His idea of number is poor and below the average. His speech is *very indistinct*, and it is often quite impossible to understand him. He takes a keen interest in home affairs and answers intelligently questions relating thereto. He is constantly sucking his thumbs and first fingers."

no protrusion of the tongue. He is somewhat "adipose," the subcutaneous tissues being very thick, the hair and skin are, however, normal and not dry, rough, or scaly. The hands and feet are normal in every way. The thyroid gland is palpable and not enlarged. There is no rickety rosary or other evidence of rickets, of syphilis, or of other constitutional disease, and physical examination is negative. He shows no deformity of any kind. *Measurements.* Height 34⅛″. Lower extremities: antero-superior iliac spine to internal malleolus 15″; length of femur, antero-superior iliac spine to articular margin of internal condyle 8″; tibia, internal condyle to internal malleolus 7″. Mid-point between vertex and soles falls about 1½″ above upper border of symphysis pubis, as in the normal for age. Upper extremities: acromial angle of scapula to extremity of styloid process of radius 10⅛″; length of humerus, acromial angle to external epicondyle of humerus 5⅛″; radius, external epicondyle of humerus to extremity of styloid process of radius 5″. The proportions are thus normal for age. Circumference of chest 20½″. This child has dark hair and eyes like his brother, IV. 10, and his father and mother. His complexion, like that of IV. 10, is more sallow than that of the other children. IV. 11, L. P., aged 6 years 9 months. Height 37⅛″. As regards the cranium she presents some of the features shown by IV. 10 and IV. 12 (though to a less marked degree), and she is very small for her age. The cranium is, relatively to the size of the face, a little larger than normal at her age, somewhat high and distinctly quadrate. Its maximum circumference is 19⅝″. But the parietal and frontal eminences are not so markedly prominent as in the other two cases, the forehead is only slightly bulging; the bridge of the nose is depressed but not exceedingly so. She is not very much smaller, for her age, than her sister, IV. 7, whom she resembles further in having light hair and eyes. Apart then from her small height and the cranial peculiarity she shows no abnormality, and compared to her two brothers might be described as a partial case. She looks an intelligent, healthy child. She "does very well at school," where she is in the first standard[1]. She appears to be of very amiable disposition and "is always laughing." She has never had any illness. Dentition is normal and all the teeth are quite sound. The palate is more high-arched and narrower, as in IV. 12, than in IV. 10 and the other children. The skin, hair, hands and feet show no peculiarity. The subcutaneous tissues are not increased. The thyroid gland is palpable and not enlarged. There is no evidence of rickets, syphilis, or other constitutional disease. Physical examination is negative. She shows no deformity of any kind, and the proportions are normal for age. The limbs are well formed and fairly strong, and show no muscular wasting. *Measurements.* Height 37⅛″. Maximum circumference of cranium 19⅝″. Lower extremity: antero-superior iliac spine to internal malleolus 17″. Length of femur, antero-superior iliac spine to articular margin of internal condyle 8⅝″; tibia, internal condyle to internal malleolus 7⅜″. Upper limb: acromial angle to extremity of styloid process 11¼″, made up of humerus 6″, radius 5¼″. Maximum circumference of chest 19⅝″; of head 19⅝″. She was born at full term after an uneventful pregnancy and a normal labour. There was no hydramnios. She was of ordinary size at birth and was "a fine fat child." No "snuffles." Breast fed 10 months. First tooth 9 months. Began to talk and to walk at 1 year 4 months. She has grown slowly until about nine months ago, but does not seem to have done so since then. In this family and its antecedents and collaterals there is no history, as far as is known, of mental or nervous disorders, tuberculosis, syphilis, or any other disease. (Unpublished case. Reference to the family was provided by Dr Menzies and the above report is due to Dr Rischbieth, who expressed doubts as to classification.)

Fig. 829. *Levi's Case III.* The father and mother, I. 1 and I. 2, were Jews and first cousins. I. 1, aged 46, height 162 cm., was well built, vigorous and very intelligent. As a young man he had four blenorrhagic infections and soft chancres but he denied syphilis. His wife, however, had had four miscarriages and his children showed suspicious signs. I. 2, aged 47, height 158 cm., had irregular and bad teeth and was very irritable. II. 2, aged 22, height 161 cm., was delicate in appearance and, like her mother, suffered from dyspepsia and headache. She showed no trace of rachitis or hereditary syphilis. She married II. 1 when aged 20, and had a child, III. 1, which died a few days later. She was very intelligent. II. 3, miscarriage at three months. II. 4, miscarriage at four months. II. 6, aged 19, height 160 cm., was a tall, thin boy. He had very defective and irregular teeth and a vaulted palate. The sexual functions and intelligence were normal; right testicle palpable in inguinal canal, but not descended. II. 7, aged 16½, height 145 cm., was small, strong and vigorous with good teeth. She was very intelligent. II. 9, aged 10, height 120 cm., was a fine strong girl. II. 10, aged 5, height 106 cm., was weakly; his teeth were defective and he showed some signs of rachitis. II. 11, miscarriage at two months. II. 12, miscarriage at three months. II. 5, aged 20½, was born at term, was breast-fed, but small and weak from birth. She grew normally till her tenth year, and then growth was checked. At 17 she suffered from anaemia. At age of 18 in 1906 her sight became affected and two months later she could not walk in the streets alone. She had frequent headaches. She came to hospital in 1907, but there was no improvement. She returned in 1908. All parts of her body were in proportion, the hands and feet well made. There was no rachitic symptom, the glandular system was normal, the genital organs infantile.

[1] Her teacher reports of her: "Though really a bright little girl, she sometimes appears backward owing to shyness and *indistinct speech*. At numbers and at writing she is equal to the best in the class but she is rather backward in reading. She shows a keen and intelligent interest in stories and in games."

A very detailed description of her condition is given. The mental condition appeared normal at first but examination showed some slight defects. Measurements are given below. II. 8, aged 15½, was born normally and breast-fed. At first she had excellent health. Her parents said her growth had stopped at age of 8. There was no trace of sexual development, the genital organs were infantile. All parts of her body were in proportion. Her mental development resembled that of her sister but she was more lively. A detailed description of her is given.

	II. 5	II. 8
Measurements:		
Total height	133 cm.	130 cm.
Height of trunk when sitting from ischium to vertex	66 ,,	64 ,,
Maximum circumference of skull	49·5 ,,	51 ,,
Fronto-occipital diameter	160 mm.	175 mm.
Maximum parietal diameter	135 ,,	140 ,,
Bi-temporal diameter	115 ,,	120 ,,
Bi-frontal diameter	98 ,,	90 ,,
Circumference of thorax at nipples	64 cm.	61 cm.
Circumference of abdomen at umbilicus	65 ,,	58 ,,
Length of whole arm	57 ,,	60? ,,
Length of upper arm	25 ,,	25 ,,
Length of forearm	17 ,,	63 { 22 ,,
Length of hand	15 ,,	16 ,,
Length of lower limb from antero-superior iliac spine to heel	77 ,,	80 ,,
Length from great trochanter to heel	71 ,,	73 ,,
Length of thigh from great trochanter to articular line of knee	33 ,,	35 ,,
Length of leg from this point to lower extremity of internal malleolus	32 ,,	35 ,,
Maximum length of foot	21·5 ,,	23 ,,

Said to be infantilism of "type Lorain." (Bibl. No. 588, p. 298.)

Fig. 830. *Schmidt's Case VIII.* "Peter Rose." I. 2, said no case similar to that of II. 2 had occurred in the family. I. 1, had died of phthisis. I. 2, had fallen in the seventh month of her pregnancy with II. 2, and she had three normal children, II. 3—5, after II. 2. The account does not say whether or not she had children older than II. 2. The family had always been poor and needy. II. 2, aged 19½, was of height 128 cm. He was born an idiot. He learnt to walk at 4 years old. His head was symmetrical and of normal size. His speech was difficult to understand. He had never suffered from epilepsy. His mother said he had had a good appetite, had always been clean in his habits, and had never been ill till he went to the Idiot Asylum. Schmidt saw him after he had been 11 years in the Asylum, he was then phthisical, coughed, had no appetite and his limbs were flabby and extremely emaciated. His genitals were still in a childlike condition, the hair had just begun to grow, but was absent in the axillae. Both feet showed a tendency to pes varus. His expression was idiotic, he could only speak a few words with difficulty and could not use his hands, which were always kept flexed. He had a dragging gait. The various parts of the body were in proportion. *Measurements.* Total length of body 128 cm. Length of head from glabella to external occipital protuberance 169 mm.; breadth of head 140 mm. Length of whole arm from acromion to end of middle finger 560 mm. (Bibl. No. 270, p. 64 and pp. 69—74.)

Fig. 831. *Schmidt's Case IX.* "Heinrich Nisse." I. 1, was a healthy labourer not alcoholic. I. 2, was healthy. I. 1, and I. 2, had seven children, II. 1—7. II. 1, was hydrocephalic. II. 2—4, and II. 6—7, were healthy. II. 5, aged 17, was a congenital idiot and a dwarf, there was no similar case in the family. He entered the Idiot Asylum at age of 9, and the doctor of the Asylum said that then the growth and development of his body corresponded tolerably well with his age. The muscular system especially that of the lower limbs was weak and the shape of his head was somewhat distorted. He was always dribbling. He knew his name and could make his wishes and needs known to others but could not dress himself. He only learnt at 8 years of age to walk alone. Schmidt saw him eight years later. His body had the proportions of a child. The genitals were not in proportion, the testes being atrophied while the penis was fairly well developed. His skull was unsymmetrical and too small for a boy of 17—the horizontal circumference being 475 mm. The spinal column in the thoracic part was slightly curved to the left and in the sacral part to the right, he was flat-footed and his gait was unsteady.

During his residence in the Asylum his mental faculties had improved. He had become clean, had ceased to dribble, and could dress and undress himself. He attended the kindergarten. *Measurements.* Total length of body 11·49 cm. Length of head from glabella to external occipital protuberance 156 mm.; to the most prominent point of the occiput, when head is parallel to the horizontal plane 162 mm.; breadth of head 132 mm. Perpendicular length of spinal column 572 mm. Length of sternum 132 mm. Circumference of chest measured at the nipples, average with quiet breathing 652 mm.; abdomen at umbilicus 570 mm.; hips at crests 559 mm.; at trochanters 560 mm. Length of clavicle 118 mm.; humerus 236 mm.; ulna 179 mm.; radius 175 mm.; hand from end of radius to end of the middle finger 140 mm.; from end of radius to beginning of first phalanx of middle finger 70 mm. Total length of arm from acromion to end of middle finger 515 mm.; circumference of upper arm at middle 155 mm.; maximum circumference of forearm 172 mm. Length of femur 310 mm.; tibia 286 mm.; foot 210 mm.; leg from trochanter to external malleolus 590 mm. Circumference of middle of thigh 305 mm.; calf 205 mm. (Bibl. No. 270, p. 64 and pp. 69—74.)

Fig. 832. *Flemming's Case.* No statement is made with regard to I. 1. I. 2, was anaemic but otherwise healthy. She had one healthy child, II. 1, and in the eighth month of her pregnancy was confined of a stillborn male child, II. 2. This child had a remarkable appearance. Except for the enlarged belly there was little to note in the trunk, the head was rather large but soft and shapeless, the face expressionless and heavy, the nose wide and flat, the skin about the neck very thick. The limbs were very short in proportion to the trunk, they were markedly curved and bent; with relatively small hands and feet. The width from shoulder to shoulder was normal, but the arms were very short and deformed, the upper arm and forearm both bent to nearly a right angle, the concavity being forwards and inwards; the wrists were markedly pronated. The hands were very small but not deformed. The legs, like the arms, were short and bent; the curve of the thigh was a double one but not so acute as in the arm; the most marked curve was at about the junction of the lower and middle thirds and had its concavity forwards; above this was a slight curve backwards and a little inwards. The legs had the most marked deformity of all, being bent backwards to less than a right angle. The feet were in a position of equino-varus due to deformity rather of the leg than of the tarsus. The writer considers the case as one of achondroplasia. (Bibl. No. 355, p. 21.)

Fig. 833. *Sandel's Case.* Sandel says the growth of these two dwarf children was stunted by bad nursing and that the boy was an example of the "perniciousness of the practice so common among the lower class, both in Stockholm, here (Hedemora) and other places, to give their children bread dipped in brandy for laying them quietly to sleep." I. 1, was a sailor; he and his wife, I. 2, were of medium bulk and stature. II. 1, aged 9, would have been reckoned short for age of 4. No further particulars are given about her. II. 2, aged 7, was so small that he would not have been taken to be above 2. When stripped and weighed, he weighed 15½ lbs. and his total height was $\frac{5}{4}$ of an ell and 1 inch (the Swedish ell is but 2 feet). The calf of his leg measured 9″ round, his arm 5″ and his belly $\frac{3}{4}$ of an ell and 4 inches. His head was no more than proportioned to his body, his limbs in no part exceeded the natural size and no mark of rickets was to be seen on him. His back was also free from any defect. His skin was soft, flaccid and of an uncommon dryness. His face of mulatto complexion, though this might be owing to sun and air. Possibly chronic alcoholism of childhood. (Bibl. No. 26, p. 68.)

Fig. 834. *Schmidt's Case X.* "Jakob Maier." I. 1, I. 2, I. 3, and I. 4, were of average height. II. 2, tall, strong and well proportioned, died aged 57, from 8 to 14 days after the excision of a "wart" on his lip (probably a malignant tumour). He tore off the bandage and bled to death. II. 3, well built and strong, of average height, died aged 65, of "dropsy of the heart" (Herzwassersucht). III. 2, aged about 46, was well built, strong and cheerful. She appeared very intelligent and gave the account of her brother, III. 5, with intelligence and certitude. She had married a healthy countryman, III. 1, but had no children by the marriage. Before marriage she had two illegitimate daughters, IV. 2—3, both quite normal. IV. 2, aged 25, was married and had two normal children, V. 1—2. IV. 3, aged 23, had an illegitimate child, V. 3, who was also well formed and normal. III. 4, died aged 6 months. It was "like other children." III. 5, the microcephalic dwarf was born 1859 and died 1878, aged 18. When born he appeared normal, but at the age of 6 months it was noticed he was not all right. For the first year he grew quickly then more slowly till his 7th year and from then very slowly, but he always continued to grow a little except the head which remained the same size. His mental faculties were of the lowest grade, he never learnt to eat alone, or to distinguish any one except his mother. In general he appeared to notice nothing, played with nothing and lay almost motionless. He was very thin, with little strength, and very badly developed muscles. He often had a violent cough. His hair was fine and fair. His fingers had to be kept bound up, as he was constantly biting them. His skeleton measured about 93·0 cm., and he looked like a 4 year old child except for his powerfully developed teeth. The skeleton appeared well proportioned and even the microcephalic skull was symmetrically formed. A very long table of measurements is given, only the most important are

reproduced below. (Schmidt gives photographs of skeleton and skull.) *Measurements.* Total length of body 93·0 cm. Circumference of skull 590 mm. Length of skull from glabella to external occipital protuberance 133 mm.; to most prominent point of the occiput, parallel with the horizontal plane 132 mm. Length of skull measured as 'Intertuberallänge,' without paying attention to the horizontal plane, from a point in the middle of the line joining the tubera frontalia to the external occipital protuberance 132 mm. Breadth of skull 111 mm. Height of skull between basion and vertex 95 mm. Vertical length of spinal column 380 mm. Cervical portion of spinal column 70 mm. Dorsal portion of spinal column 150 mm. Lumbar portion of spinal column 86 mm. Length of clavicle 83 mm.; humerus 165 mm.; ulna 141 mm.; radius 132 mm.; femur 247 mm.; patella 27 mm.; tibia 182 mm.; fibula 179 mm. (Bibl. No. 270, p. 55 and pp. 69—74.)

Fig. 835. *Langdon Down's Case.* I. 1, and I. 2 were healthy, there was no history of mental or physical deviation on either side. I. 2, stated that her first child, II. 1, was perfectly healthy until it died from measles. I. 1, about that time gave himself up to intemperance and I. 2 subsequently gave birth to a child, II. 2, who had the same physical peculiarities as II. 3, it died aged 3. II. 3, aged 5, like II. 2, was procreated while I. 1 was suffering from alcoholic intoxication. Then followed a miscarriage, II. 4. After this I. 1 became sober, thrifty and prudent, and II. 5, aged 16 months, was born. It was normally developed and in good bodily and mental health. II. 3, was only 22″ in height, she could not speak or walk but could stand with the help of a chair. There was no deformity of body or limbs. The face was of earthy complexion and the integument generally had a wrinkled appearance, as if it were too large for the diminutive body. The hair was sparse and coarse, the eyebrows obliquely placed, the tongue large and rugous. On each side of the neck above the clavicle there was a venous tumour. She understood what was said to her, but her mental condition was that of a child of 15 months old. (Bibl. No. 140, p. 419.)

Fig. 836. *Taruffi's Case IV.* A longer description of this case is given by Taruffi in another paper (Della Microsomia, *Rivista Clinica di Bologna*, 1878, Nota 5). He states that the following is a condensed description. I. 1, and I. 2, were normal, and had six normal children, II. 1—6. II. 7, Tommaso Businaro, was born 1855. The pregnancy of his mother with him was normal. When aged 5 months he was vaccinated and became eczematous on the arms and afterwards on the thorax and this affection lasted for five years, producing a remarkable retardation of growth. When examined in 1875, his height was 70 cm. Taruffi saw him in 1877 and his height was then 110 cm., so he had grown 40 cm. in two years. He had a well formed skull, the nose was short and depressed at the bridge. His arms were rather short, the forearms were however relatively to the humerus rather long. A similar disproportion existed between the two segments of the lower limbs. There was no hair on the chin or pubes, the testicles were very small and he had no sexual instincts. His intelligence was defective, he could neither read nor write and was idle, passionate and obstinate. The case may be one of cretinism or syphilis through vaccination. *Measurements.* Horizontal circumference of head 49·5 cm. Length of arms 17·2 cm.; legs 52·0 cm. Cephalic index 82·31. (Bibl. No. 248, p. 447.)

Fig. 837. *A. Marie's Case.* We are indebted to Dr A. Marie for the particulars given below of this case, which he most kindly sent us. Dr Marie saw these four dwarfs at a fair or "Kermesse," and could only observe them cursorily. I. 2, the mother, had died of chronic tuberculosis, she was rachitic and said to be alcoholic (buveur). No note of I. 1, the father, or any other relatives is given. II. 1, the eldest dwarf, was a case of simple infantilism. The two next, II. 2 (erroneously marked tubercular in pedigree) and II. 3, had round faces (faces lunaires), "les mains en palette," the segmental shortening of the limbs characteristic of a myxoedematous condition ("d'un état strumprive") and diminished vitality combined with arrest of the skeletal development. The youngest, IV. 4, had less oedematous infiltration, but on the other hand his skin seemed old and wrinkled, and his appearance was more markedly cretinoid. (Plate JJ (72).)

Fig. 838. *Horand's Case II.* I. 1, was healthy, neither syphilitic nor tuberculous, but "il buvait du vin comme un bon vigneron." He was 1·55 m. in height and was aged 36 at the birth of II. 3. I. 2, was healthy, did not suffer from myxoedema, tuberculosis, rheumatism, alcoholism or any nervous disease. She had three children, II. 1—3, and was aged 32 at birth of II. 3. II. 1, aged 14½ years, was 1·50 m. in height and very intelligent. II. 2, aged 10½ years, was 1·20 m. in height and intelligent. II. 3, was aged 6. I. 2, had threatened miscarriage with escape of waters two months before the birth of II. 3; otherwise the pregnancy was normal. The confinement was normal, the child was well formed but very small. It was breast-fed for one year. For two years she weighed 7·50 kilos. She had no illness, but whooping-cough followed by bronchitis with oedema of feet and hands. In 1903 she fell from a chair on her head and was for two months in "coma vigil." In 1904 she had "thrush." Her circulation was bad. She walked late, could not even walk alone at age of 6. Her intelligence was defective, she seemed to understand everything, but had never spoken except to say "papa," "mamma." She was very merry but irritable. Her dentition was good. The skull was oval in shape; the anterior fontanelle still

persisted. She had a large, round, rather flat nose, a normal tongue and palate and a neck so short as to be almost absent. The thyroid body was absent, and the genitals but little developed. The upper limbs were normally formed. The fingers were fairly long. With the arms extended by the sides the middle finger reached to the upper half of the thigh. At 6 years of age measurements were as follows. *Measurements.* Weight 9 kilos. Height 62 cm. Occipito-frontal circumference of head 47 cm. Circumference of trunk 49 cm. Both humeri 10·25 cm.; both ulnae 9 cm.; both radii 9 cm.; both femora 15·5 cm.; both tibiae 11 cm. The lower limbs looked like sticks; the nails and toes were short. Sensibility was normal. Probably cretinism. (Bibl. No. 486, p. 930.)

Fig. 839. *Clauder's Case.* There is really no description given of this case, all that is stated is "Sic maritus quidam cum uxore generavit octo liberos utriusque sexus, quorum pars dimidia fuere nani, ubi praecipuè notabile alternatem semper esse productos modò solitae magnitudinis, modò nanos." So apparently I. 1, and I. 2, were normal, and of their eight children, II. 1—8, four were dwarfs whose births alternated with those of the normal children; this alternation of births is similar to that in the Boruwlaski family. (Bibl. No. 14, p. 543.)

Fig. 840. *Zweifel's Case*, given by Caruso. I. 2, aged 21, was rickety and scrofulous, she began to walk at the age of 4 and at 15 had periostitis of the right upper arm. She was a healthy looking, strongly built, well nourished person with rickety rosary, genu valgum and thickening of the epiphyses. Her height was 120 cm. She came for her first confinement, Caesarian section was performed and a female child extracted. Weight 2950 grammes, length 49 cm. (Bibl. No. 241, p. 219.)

Fig. 841. *H. D. Smith's Case.* I. 1, had black blood in her, was dark, either half-caste or quadroon. I. 2, was pure white. No note is made of any of their children except II. 3, who became a missionary. Of his 22 children by his first two wives, II. 1, and II. 4, eight died apparently young, they were all normal, but it is not certain whether three or four of the first family died, or four or five of the second. The numbers in pedigree are those which are thought correct, namely that three, III. 6, died in first family and five, III. 16, died in the second family. There appears little to note in the normal descendants of II. 3, except a tendency to dark colour. III. 2, died unmarried. III. 5, is quite dark, practically a coloured man, and his children, IV. 2, are dark with woolly hair. The dwarf, III. 12, is very ugly, but it cannot be definitely stated how he should be classified, probably he is an achondroplasic dwarf, as he is said to have a large head and short legs which are not bowed. III. 13, a girl came from England, and was married to him. They have three children, IV. 9—11, two of whom are dwarfs. III. 12, has been off and on in the public service of his state and is said to be clever at his work. Some of the brothers of III. 12 are professional men. (Unpublished.)

INDEX TO DWARF NAMES.

B. = *Bibliography* (Bibliography numbers in italics signify that the dwarf is referred to in original although not mentioned in the résumé given), *Ic.* = *Iconography*, *Pl.* = *Plate*, *F.* = *Figure of Pedigrees*, *p.* = *Page of Text.*

[1] A most excellent reproduction of Knoumhotpu has just been published by M. A. Ruffer in his paper on "Dwarfs and other Deformed Persons in Ancient Egypt," *Bulletin de la Société Archéologique d'Alexandrie*, No. 13, 1911.

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Rischbieth

Rischbieth

Rischbieth

Apert & Severtre

Guéniot & Potocki

Lepage

Launois & Apert

Comby

Lauro

Launois

Porter

Baldwin

Decroly

Peloquin.

Boeckh (original)

Litchfield

Herrgott

No Consanguinity recorded.

True dwarf

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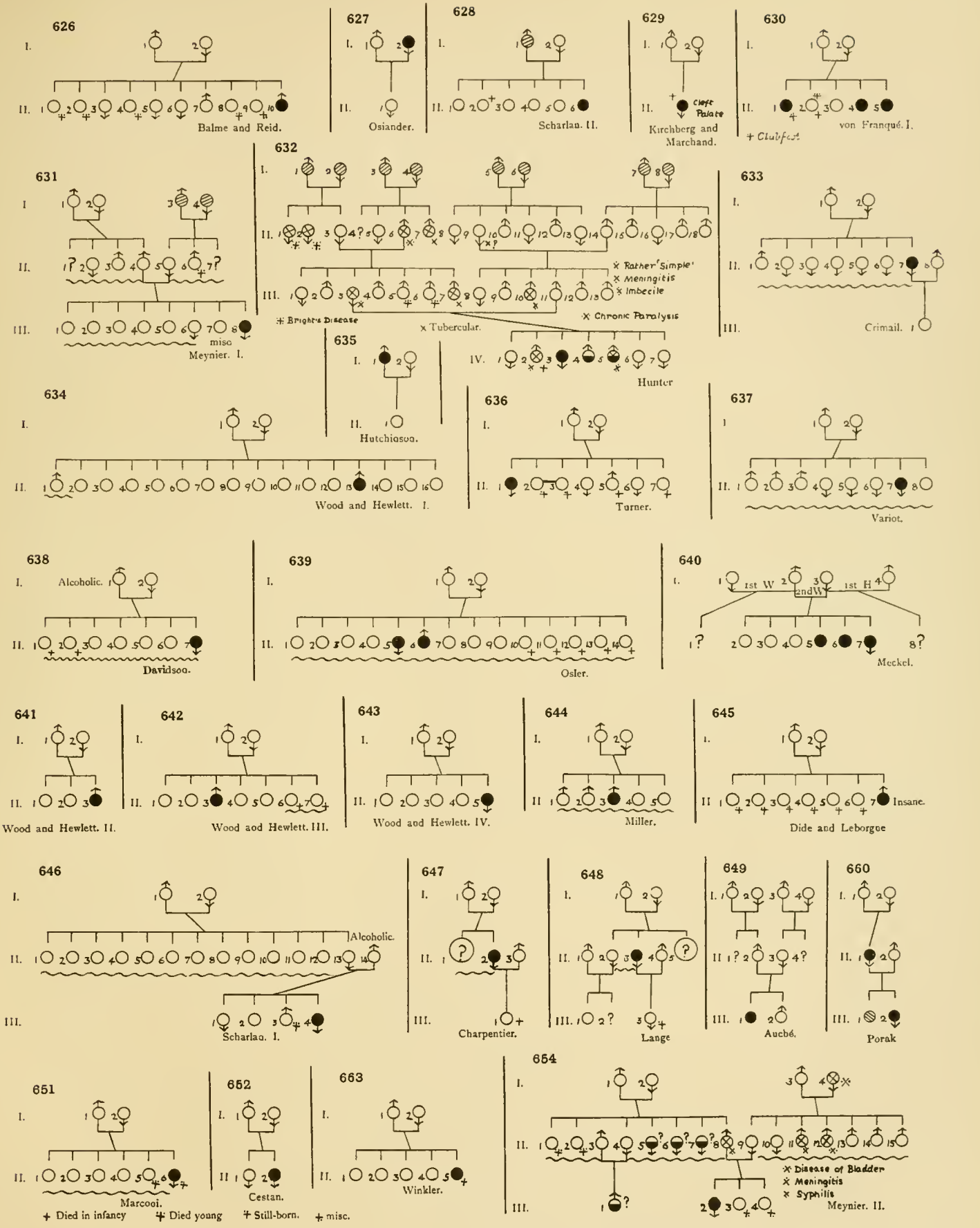

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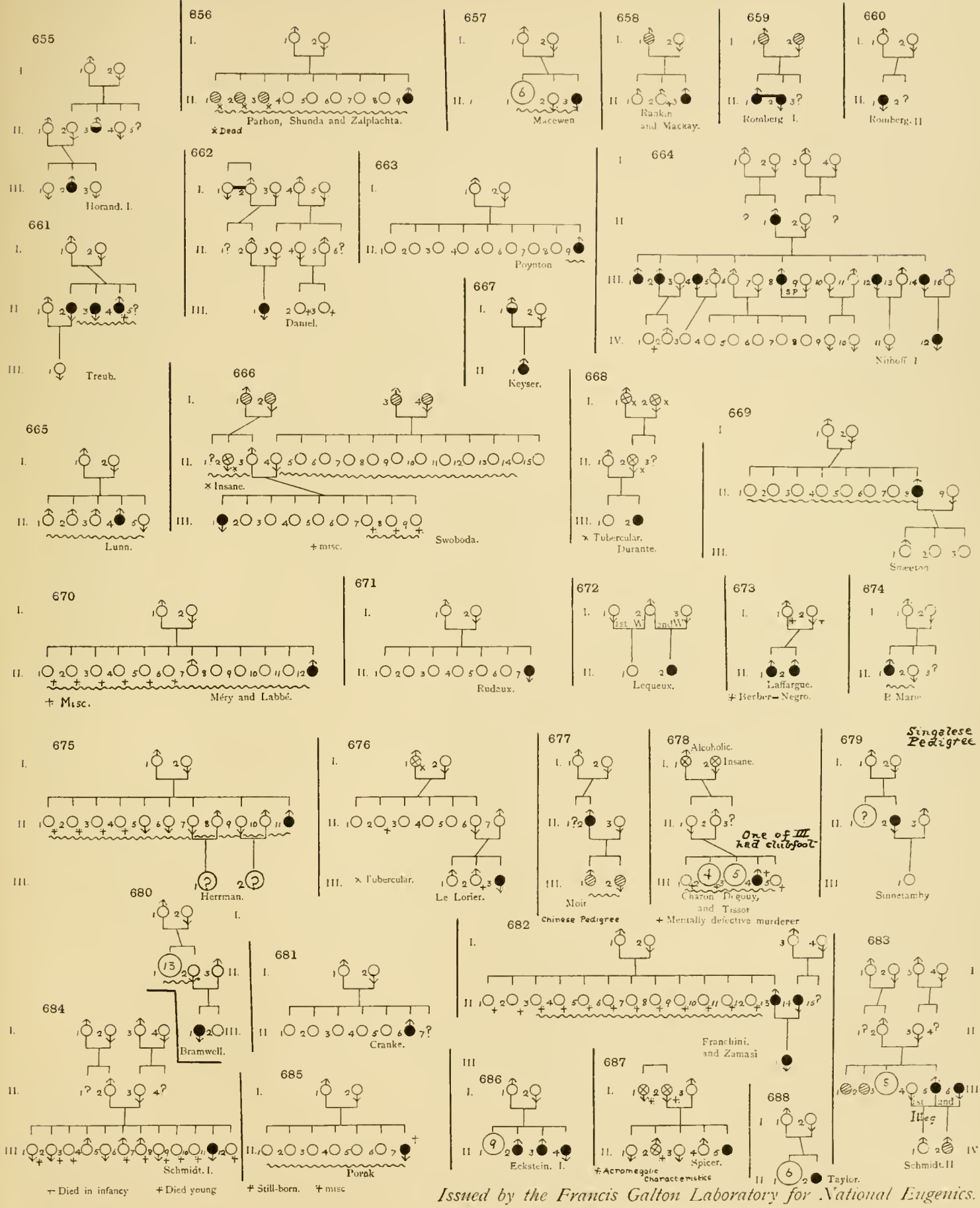

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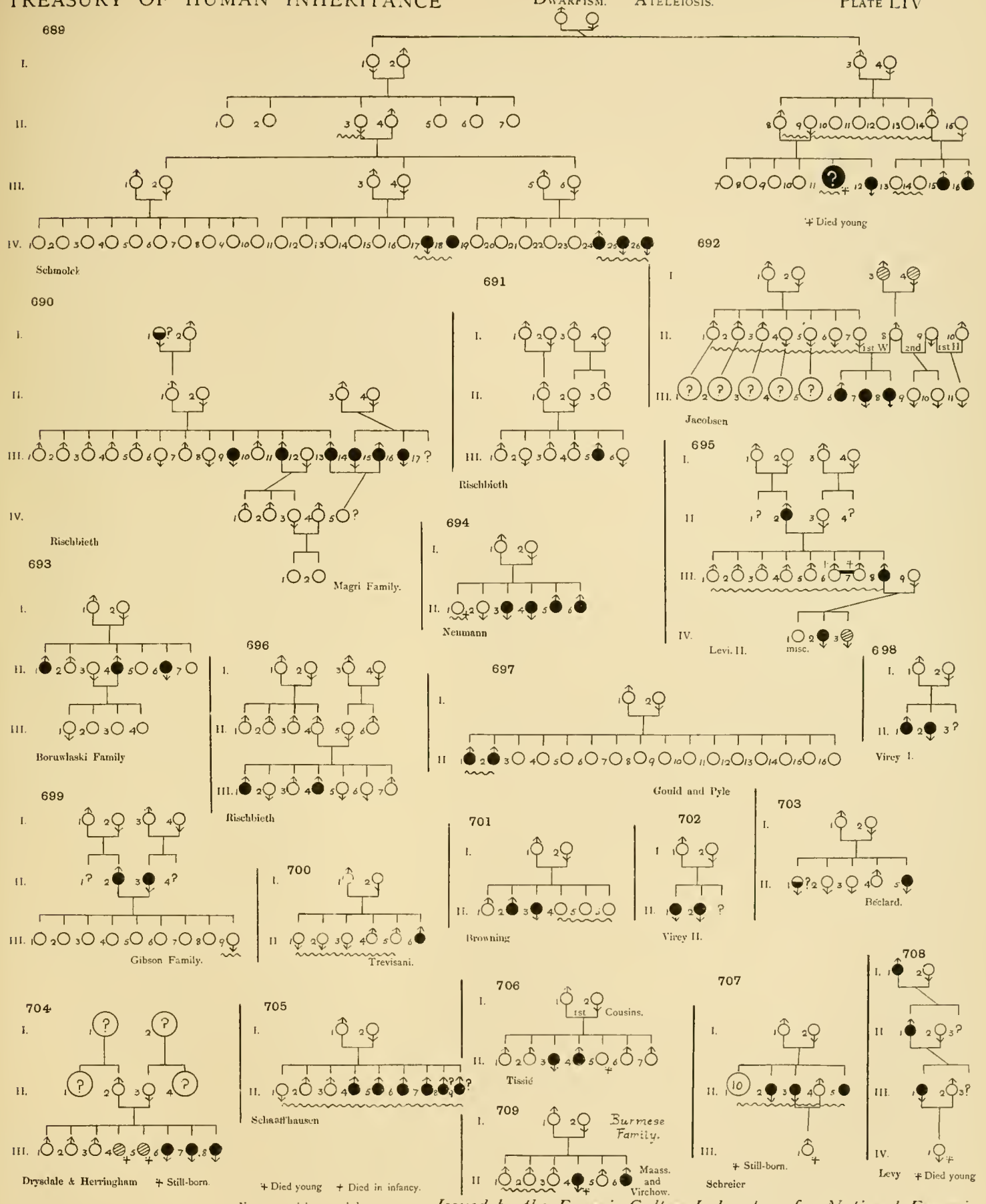

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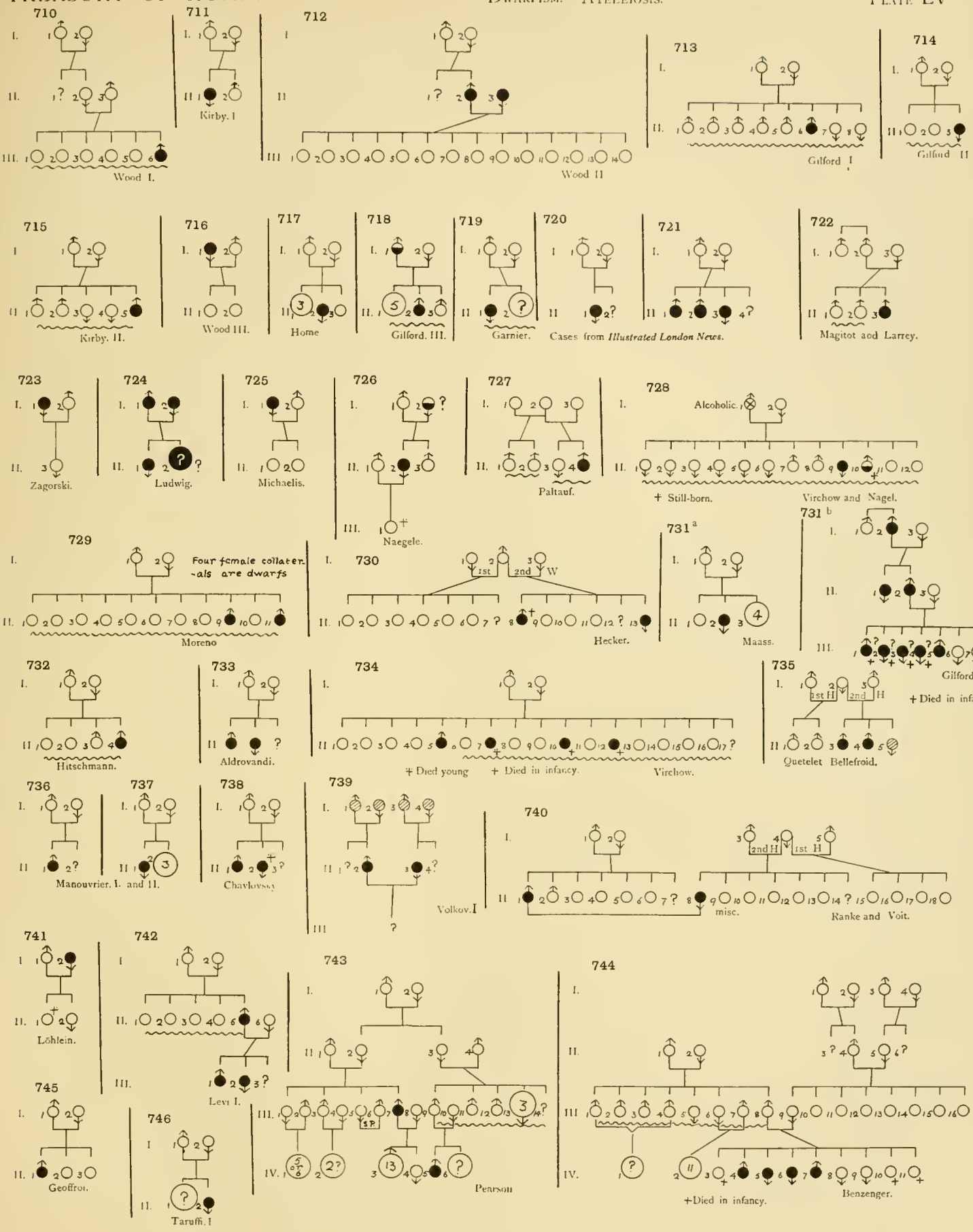

†Stillborn + Died in infancy .
†Died young.

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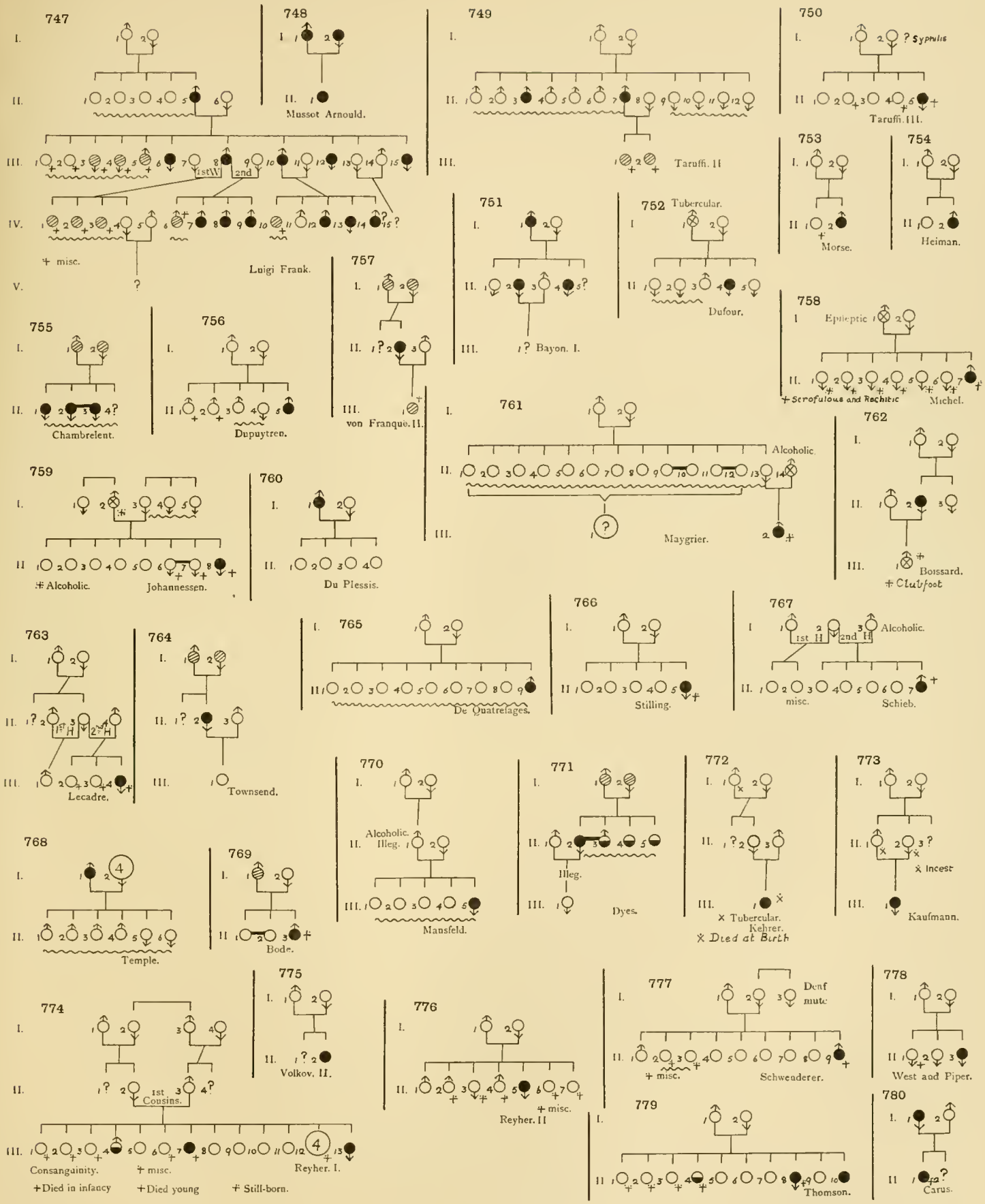

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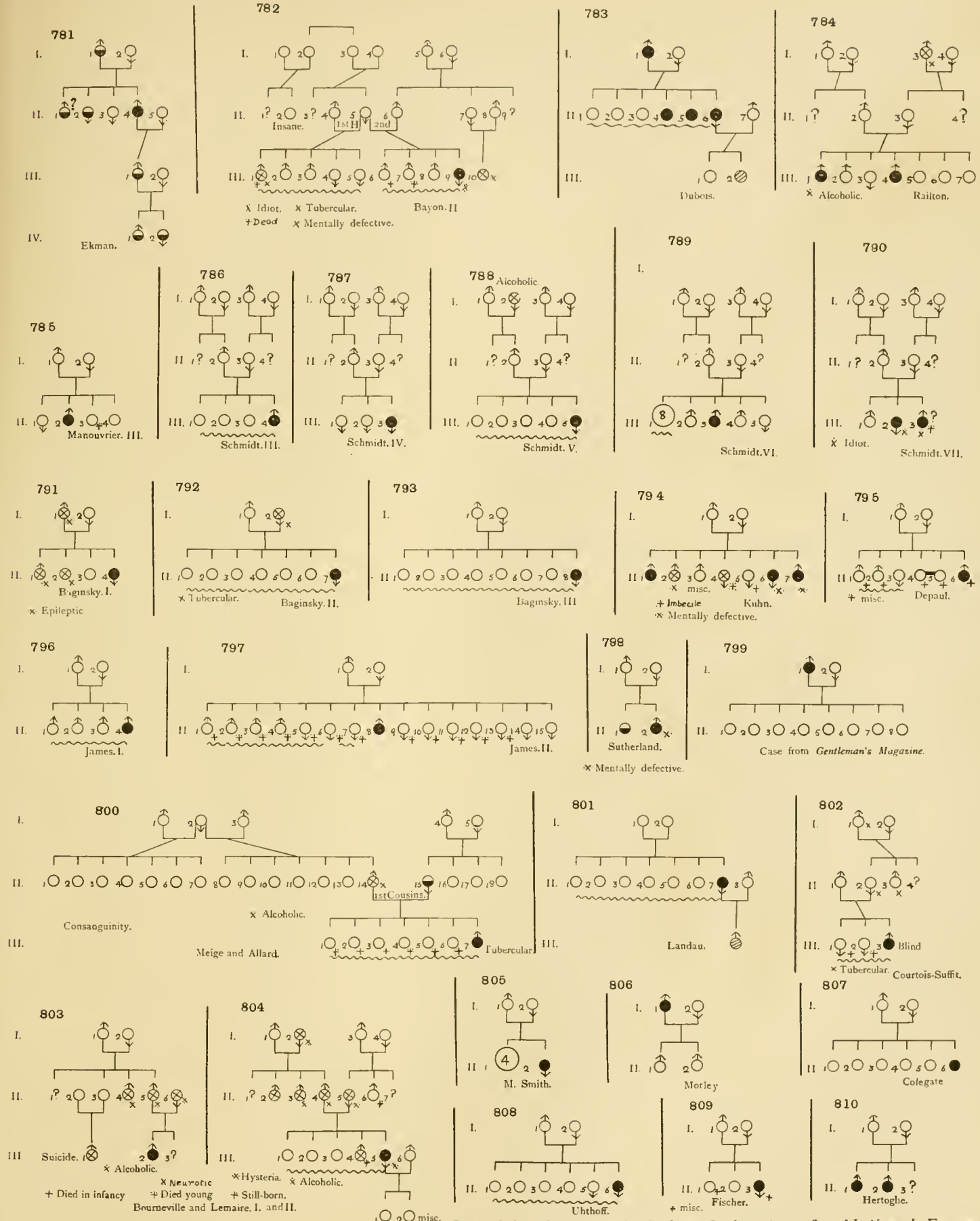

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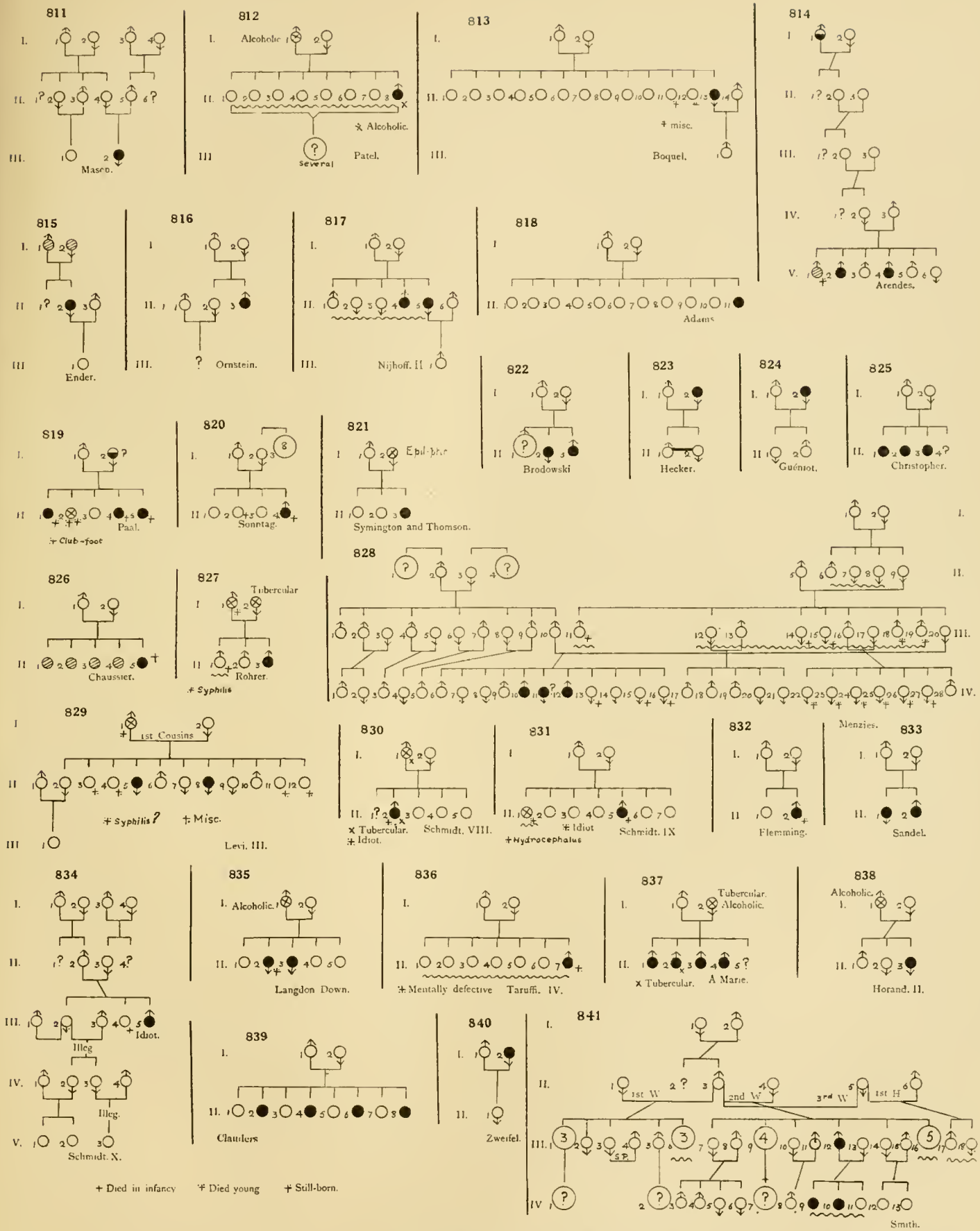

+ Died in infancy ⁜ Died young ✝ Still-born.

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This index lays no claim to completeness. Almost monthly we receive notices of new cases of ateleiotic dwarfism. Many of these cases are known by "show" names, and, apparently for commercial purposes, great reticence is maintained as to family history and locus of origin. Thus it is not always possible to ascertain whether a "new" dwarf is really an old friend under a novel name. Still this index may be of some service to the future student of heredity endeavouring to link up the dwarfs of his day with those of the past.

DESCRIPTION OF PLATES[1].

A. Ethnic Dwarfism[2].

PLATE O (1). Akkas as representatives of ethnic dwarfism. The only peculiarities these show are those of race. They are normally proportioned. Average stature 3′ 6″ to 4′. These dwarfs are shown here in contrast to those whose dwarf growth is due to disease. By kind permission of Sir Benjamin Stone.

The reader should also compare Plate VV (107).

B. Dwarf Growth of Pathological Origin: Achondroplasia.

PLATE P (2). Twins, aged 15 months. By kind permission of Dr Robert Hutchison. Male normal, a well grown child; but for left internal strabismus (a condition which the father also shows) he shows no peculiarity. Female achondroplasic. Note the large head (the maximum circumference of the head in these two children is equal). Trunk of about the same length as in the male child; measurement from episternal notch to upper border of symphysis pubis is as nearly as possible equal in the two, though a little the greater in the male. All extremities in the female are markedly shortened (micromelia). This affects the proximal segment more than the intermediate, *i.e.* arm and thigh are shorter than forearm and leg respectively, whereas in the normal they are longer; thus the micromelia is of rhizomelic as opposed to mesomelic type. The hands are short, broad and thick; the fingers are of nearly equal length and show the "trident hand" deformity ("main en trident"). The feet are also typical. But neither hands nor feet are properly shown here. All the long bones are slightly curved, convexity externally, and the epiphyses are somewhat enlarged. The abdomen is prominent. She has a rickety lumbar kyphosis (not shown here), but no "beading" of ribs, "rosary" or other evidence of rickets. The head shows the following features: prominence of frontal and parietal eminences, depression of the bridge of the nose, which is tip-tilted, with wide nostrils. The face relatively to the size of the cranium is small and somewhat narrow, the malar bones being small. The general contour of the face is thus somewhat like an inverted pear, while that of the normal child is oval. This is a common feature in achondroplasia, though shown more markedly in some other conditions, *e.g.* hydrocephalus. All normal skin folds exaggerated. Not a fat child, little increase of subcutaneous tissues (unusual features in achondroplasia). Intelligence normal. Beginning to walk and talk. Mesial and lateral incisor teeth in upper jaws and mesial incisors in lower ones present. See Fig. 609.

(3). L. D., whose pedigree is shown in Fig. 610. An achondroplasic girl aged 7 years and her sister aged 5 years, of normal proportions and average growth for age to show contrast of growth with the normal. The length of the trunk measured from episternal notch to upper border of symphysis pubis is equal in these two cases. The circumference of the cranium is half an inch greater in the achondroplasic child than in the normal one. The distance between the vertex of the skull and the upper border of symphysis pubis is equal in the two cases. The mid-point between the vertex and the soles of the feet, which in the normal adult lies just at the upper border of the symphysis pubis, is situated in the normal child above this point, about one-third of the distance upwards between the upper border of symphysis and the umbilicus. This represents the normal proportions of limb length to stature in infancy and early childhood and is shown in "true dwarfism," see Plate EE (57). In the achondroplasic child the mid-point lies about midway between the umbilicus and the distal extremity of the xiphi-sternum. The lower extremities are thus markedly shortened (micromelia). The upper extremities are also markedly shortened (micromelia); whereas in the normal child the finger tips extend to the middle third of the thigh, in the achondroplasic they only extend to a point midway between the iliac crest and the great trochanter of the femur. This shortening affects the proximal segments of the limbs more than the intermediate segments, *i.e.* it is of rhizomelic type. In the upper extremity of the normal child the arm, measured from the acromial angle of the scapula to external epicondyle of humerus, is longer than the forearm measured from external epicondyle to tip of styloid process of radius by $\frac{1}{16}$ of the length of the former. In the lower extremity the thigh, measured from the antero-superior spine of the ilium to the lower border of internal condyle of the femur, is longer than the leg, measured from the upper border of internal tuberosity of the tibia to the tip of the internal malleolus of the latter by $\frac{2}{11}$ of the length of the former. In the achondroplasic child, these segments, measured from the above points, show that the femora are a shade

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[2] A study of eleven pigmy crania found in ancient Egyptian cemeteries has recently been made by H. Dorothy Smith: see *Biometrika*, Vol. VIII. p. 262, with full scale photographs.

shorter than the tibiae. The limbs have a thick-set, massive appearance. The arms are held with elbows abducted from the sides and not, as in the normal, at the sides. This, as is shown by radiograms, is partly due to the large size of the heads of the humeri, partly to curvature of bones. It is probably due also, in part, to intervention of muscular masses. All the long bones of the limbs are somewhat curved; this, however, owing to thickness of muscles, can only be seen in the case of the tibiae. It takes the form of slight angular curvatures in the region of junction of epiphyses and diaphyses and does not affect the shafts, as in rickety curvature. All the epiphyses of the extremities as revealed by palpation of the long bones are somewhat enlarged, but the bones of the thorax are apparently normal, with the exception of the sternum, which is bowed with convexity forwards. There is no "rosary" or "beading of ribs." All the normal skin folds are exaggerated and the subcutaneous tissues increased. The normal child has a commencing mid-dorsal scoliosis of adolescence, convex to L., which makes the R. shoulder lower than the L. as she stands.

(4). The achondroplasic child aged 7 years shown in (3). Note, in addition to features already described, the large size of the head, with prominent frontal and parietal eminences. The bridge of the nose is depressed and it is tip-tilted. The buttocks are prominent; there is apparent lordosis. The abdomen is very prominent. Note here the massive appearance of the limbs, the large, broad and thick feet and hands and the peculiar shape of the latter. The sternum is somewhat bowed, with convexity forwards. There is an appearance of obesity owing to the increase of subcutaneous tissues. Intelligence normal. Compare with Plate V (19) and (22).

(5). The hands of achondroplasia contrasted with normal hands; they are those of the cases shown in (3). Note that the achondroplasic hands are very short, and are broad and thick. The fingers are relatively short and thick. They are more nearly equal in length than the normal. In complete extension the distal extremities of the digits (or finger-tips) cannot be approximated but diverge or radiate like the spokes of a wheel. This constitutes the "trident hand" or "main en trident" of Pierre Marie. Compare with Plate Q (6), Plate R (11), Plate U (19), and Plate V (27). The fingers show another peculiarity when compared with the normal in that the three segments of each of the digits form, as it were, three separate superimposed cylinders of progressive diminution in diameter. This appearance has also been described as that of a truncated cone. Contrast this with the gradual tapering of the normal fingers. The subcutaneous tissues are increased and all normal skin folds are exaggerated. Photographs (3) to (5) are due to the kindness of Dr Robert Hutchison.

PLATE Q (6)—(8). D. W., aged 28 years. Height 4′ 1″. Acrobat. An achondroplasic male adult. Note shortening of all four extremities in comparison with size of the head and trunk. The mid-point between the vertex and the soles of the feet falls half an inch above the umbilicus, instead of, as in the normal, at the upper border of symphysis pubis. The lower extremities are thus much shortened, and their length, if proportions were normal, would correspond to a total height of just over 3′, instead of, as here, to 4′ 1″. The head and trunk, showing normal proportions to one another, would correspond to a height of just over 5′, if the proportions in length of the lower extremities to trunk and head were normal. The upper extremities are markedly shortened; the finger tips extend only as far as the great trochanters of the femora, instead of to the middle third of the thighs. This micromelia of all four extremities is of rhizomelic type, affecting the humeri and femora more than the bones of the forearm and leg. Thus the measurements are: R. humerus 5″ (acromial angle to external epicondyle), R. radius 6″ (external epicondyle to styloid process), R. femur 8″ (antero-superior iliac spine to internal condyle), R. tibia 9″ (internal tuberosity of tibia, *i.e.* the line of the knee joint, to internal malleolus). The radio-humeral index, *i.e.* (length of radius)/(length of humerus) × 100 is 120; the tibio-femoral index, obtained in the same way, is 112·5. In normal European adults[1] the first varies between 82 and 88, and the second between 84 and 90 (Porak). In achondroplasia the radio-humeral index may be as low as 66, 58 or even 53; the tibio-femoral as low as 78 or even 64 (Regnault). In Porak's *Maternité* skeleton they were 86 and 85. Compare (5), Plate R (13). Length of foot 8″, *i.e.* equal to that of the femur. The long bones of the extremities show no curvatures that can be made out on palpation. The arms are held with elbows abducted from the sides. Note the massive musculature of the limbs, prominent buttocks, straight or flat back, but nevertheless an appearance of lordosis, and, though this individual is not corpulent, the prominence of the abdomen. The hands, which are typical as well as the feet, are relatively short, broad and thick. The head is large and brachycephalic; the frontal and parietal eminences are prominent. The bridge of the nose is depressed, its extremity *retroussé*, the nostrils are large and broad. The measurement from nasion to inion is very short. Compare with other cases of achondroplasia and contrast with the "true" dwarfs. There is a slight degree of prognathism. The face relatively to the cranium, which overshadows it, is small and narrow, but does not show the "inverted pear" contour so clearly as in some other cases. Intelligence good. Reads and writes as well as can be expected of any individual of his profession. Genital organs normal, secondary sex characteristics present. Compare with Plate R (11—13), which shows achondroplasic modification of head in the brachycephalic Mongolian skull; and with Plates S (14, P), U (18) and (19), V (29).

[1] [This statement appears to be incorrect: see my footnote p. 378. EDITOR.]

(9). Elizabeth Dörffler, *née* Kipke, aged 42 years, whose pedigree is given in Figs. 608 and 620. With her is her daughter, aged 17 years. On Plate FF (62) is shown her elder sister, aged 43, with her ateleiotic husband and son, aged 18. All three women are typically achondroplasic. Cf. Boeckh's Case, Plate S (14, F., F.').

(10). Lili W——, aged 28 years, whose pedigree is given in Fig. 11, a typical achondroplasic adult female.

Plate R (11)—(13). A Chinaman, aged 58 years. Height 3′ 6¾″. Marked micromelia of all four extremities. This, however, is not rhizomelic, as is usual in achondroplasia, but the normal proportional length of segments is preserved, at least in the lower extremities. The finger tips extend just beyond the crests of the ilia, but do not reach the great trochanters. The mid-point between vertex and soles of the feet is 2⅜″ above the umbilicus (which is 19″ from the ground) instead of, as in the normal adult, at the upper border of symphysis pubis. The head is large, relatively and absolutely, circumference 22¼″. Mastoid to mastoid 7″. The frontal and parietal eminences, though not very prominent, are much more so than is seen in the normal brachycephalic or mesaticephalic Mongolian skull. For a Mongol, in whom in the normal the malar bones are very prominent and the face therefore very broad and flat, the face is very narrow and small in comparison with the cranium. That is the features of achondroplasia as shown in the European are here somewhat modified in the Mongolian skull. The bridge of the nose is depressed; it is "tip-tilted" and with wide nostrils. The trunk is relatively long and shows no peculiarities. Chest expanded measures 27″. The radius is 2¾″ in length. The ulna, from extremity of olecranon process to ulnar styloid process is 5″ in length (difference of radius and ulnar seems excessive). In the lower extremities the thigh, measured from antero-superior iliac spine to internal condyle of femur, is of 9½″ length; the leg, measured from internal condyle of femur to internal malleolus of tibia, is of 8″ length. Radio-humeral index cannot be stated, since the measurement of the humerus is not given. Tibio-femoral index, *i.e.* (length of tibia)/(length of femur) × 100 = 84·2. Contrast with Plate Q (6)—(8) and compare with Plate U (18). The shortening of the lower extremities is thus not rhizomelic but the normal proportion in relative length of segments is preserved. (Compare with skeleton Plate U (18) in which this is also shown. Contrast with (3), (8), (14), (17) and (19), in which the usual rhizomelic shortening or micromelia of achondroplasia is shown.) The arms are held with elbows abducted from the side. This is, however, not so marked a feature in this case as in some others. It occurs in all individuals of great muscular development. The hands show the typical deformity of achondroplasia very well indeed. They are short, broad and thick. The fingers are of nearly equal length and show the "trident hand" deformity. The digits have the peculiar shape characteristic of the condition. This has been considered by some to be like a truncated cone, by others like a series of three cylinders of progressive diminution in diameter superimposed. Compare (5), (19) and (27). Note the marked muscularity of this individual. He is intelligent, bright and alert; speaks English slightly, and earns his living by dancing and buffoonery. A native of Hankow, 500 miles up the river Yang-tse-kiang. Discovered by Dr Gordon Moir, R.N., whose case he is, at Shanghai[1].

Plate S (14). Cases of achondroplasia, referred to in the Bibliography and Pedigrees. F., F'.: Boeckh's Case. See Fig. 620 and Bibl. No. 280. G.: Joachimsthal's Case. See Bibl. No. 363, S. 288. H., H'.: Charpentier's Case. See Bibl. 247, p. 25. I., J.: Porak's Case. See Bibl. 247, p. 21, and Fig. 650. K.: Joachimsthal's Case. See Bibl. No. 363. L., M.: Thomson's Cases. See Bibl. No. 281. N.: Baldwin's Case. See Bibl. No. 254 and Fig. 621. O., O'.: P. Marie's Case (Claudius). See Bibl. No. 371 and Fig. 674. P.: P. Marie's Case (Anatole). See Bibl. No. 371. Note the appearance of obesity in all three women and the muscularity of the men. These are characteristic features as regards the two sexes. The two children G. and K. are rather thin, an unusual type of the disease.

(15). Shows: (*a*) An achondroplasic foetus, Q., R. Note the large head with depressed bridge of nose; the short massive limbs, somewhat curved; exaggeration of all normal skin folds; prominent abdomen. (*b*) The shortening of the long bones of the extremities, S. (*c*) An achondroplasic upper extremity, T. Statuettes: (*d*) The Egyptian gods Ptah-Sokar, U., and Bes, V., showing achondroplasic proportions. (*e*) The Roman Emperor Caracalla, X, in caricature, with achondroplasic proportions, thus proving that the condition was well known to the Egyptians and the Romans. See Plate UU.

Plate T (16) and (17). Bones of the extremities of an achondroplasic child at birth (17), compared with those of a normal child at birth (16). (Note: The former is a wet specimen, the latter a dry one.

[1] Another instance of typical achondroplasia in a Chinaman named Li is described by Dr Molodenkoff in the *Nouvelle Iconographie de la Salpêtrière* for June 1910. Aged 33 years. Height 115 cm. A photograph shows that he presents the same features, as regards the shape of the head, as the above case. Thus the frontal and parietal eminences, though not so prominent as in the skulls of most cases of achondroplasia occurring in Europeans, are more so than is usual in the normal brachycephalic or mesaticephalic Mongolian skull. The face also is relatively narrow, the malar bones being very little prominent. The bridge of the nose is relatively depressed. It is tip-tilted and with wide nostrils. In the same number a Roumanian case is described by Dr Zosin. His head presents the features shown in any of the other European cases figured here, *i.e.* the skull is brachycephalic; the frontal and parietal eminences are very prominent; the bridge of the nose is greatly depressed, its tip upturned and the occiput is vertical.

The relative thickness of the former is therefore somewhat exaggerated, while, owing to dryness and shrivelling, the latter appears unduly thin. This especially applies to the hands and feet. Therefore only the comparative *length* of segments of each can be considered.) In the achondroplasic limb all the long bones are short and very thick. They all show abnormal curvatures, such as are described in the text; these are juxta-epiphysial for the most part, and in some bones there are more than one. In the radius and humerus there are two curvatures occurring in opposite directions, an S or Z shape, somewhat like that of a clavicle, being produced. This is a common feature of achondroplasic foetal bones and differs markedly from the C curves, all in the same direction, of rickety bones; a condition of double curvature similar to that shown in humerus and radius can just be made out in the femur. All the epiphyses are greatly enlarged. Section through the epiphysial line, such as that of the lower end of the femur here depicted, shows a band of fibrous tissue, or, it may be, membrane, continuous with the perichondrium and periosteum, interposed between the epiphysis and diaphysis for a considerable distance. This condition is characteristic of achondroplasia and occurs in no other known condition. The nature of this band is discussed in the text, pp. 379—81. The tibio-fibular interosseous space is increased owing to bowing of the fibula outwards. The radio-ulnar joints show partial dislocations. The sacrum articulates with the ilium in a plane which is more nearly horizontal than normal, the upper end of the sacrum being displaced downwards and forwards and its lower end upwards and backwards. The clavicle is not shortened. The hands and feet are relatively very large (the foot is longer than the femur). The fingers are nearly equal in length; the third and fourth show the deformity "en trident." The clavicle and foot are longer in the achondroplasic than in the normal specimen, so that, apart from shortness of limb, the former was doubtless the bigger child of the two.

Actual Measurements:—

Lengths of:	Normal bones	Achondroplasic bones
Clavicle	$1\frac{5}{12}$ inches	$1\frac{7}{12}$ inches
Humerus	$2\frac{7}{12}$ "	$1\frac{2}{3}$ "
Radius	2 "	$1\frac{1}{6}$ "
Femur	3 "	$1\frac{11}{12}$ "
Tibia	$2\frac{1}{3}$ "	$1\frac{1}{4}$ "
Foot	$1\frac{5}{6}$ "	$2\frac{7}{12}$ "

(*a*) Radio-humeral index. Normal = 77·4; achondroplasic = 70·0.

(*b*) Tibio-femoral index. Normal = 77·7; achondroplasic = 65·2. See the remarks under Plate Q (6—8) [and the footnote, p. 555]. Thus, though it is usual for the femur and humerus to be more shortened than the tibia and radius, at least in the adult achondroplasic, this is not invariable; and it is not the case in the foetal limbs here shown, nor in (11)—(13) nor (18)[1].

Plate U (18). Skeleton of an achondroplasic adult. Note the marked shortening of all four extremities, relatively to the trunk and head (micromelia). In the normally proportioned adult the mid-point between the vertex of the skull and the soles of the feet lies at the upper border of the symphysis pubis; in this case, however, this mid-point lies at the middle of the body of the first lumbar vertebra. There is thus marked shortening of the lower extremities. In the normal adult the finger tips extend to the middle third of the thigh; in this case, however, they reach no further than the great trochanters of the femora. The upper extremities are thus markedly shortened also. The shortening involves the intermediate segment of the limb as much as the proximal, *i.e.* the shortening is not rhizomelic. This is not usual in achondroplasia but has been observed in several cases. See account of (16)—(17). The hands and feet (distal segment of the limb) are, comparatively, much less affected than the other segments and are relatively very large. This is the rule in achondroplasia. The bones of the pelvis and the scapulae are very small. The clavicles are nearly as long as the humeri. The upper end of the sacrum is tilted downwards and forwards and the promontory thus projects markedly forwards. The plane of the sacrum is thus more nearly horizontal than normal. The skull is large, relatively to total height it is very large; it is brachycephalic; the parietal eminences are very marked but the frontal eminences are not specially so. Bridge of nose not much depressed. Dentition normal. The vertebral bodies are very broad and

[1] [I doubt whether it is the general rule in achondroplasia, especially when the measurements are made on the skeleton, that the femur and humerus are shorter than the tibia and radius respectively. At least the measurements in this work show very many exceptions. The femur and humerus may have a greater percentage reduction on the normal than the tibia and radius without necessarily becoming absolutely shorter than the latter, and it might be well to interpret the term rhizomelic in this more restricted and guarded sense. Editor.]

thick. The ribs are very thick and strong and show excessive groovings and markings for muscular origins and insertions. The sternum is very broad, thick and strong. All the long bones of the extremities are exceedingly thick and strong, and show excessive markings for muscular attachments. Their ends, corresponding to old epiphyses, are relatively enormous. This is especially well shown, about the knees, in the condyles of the femora and the tibial tuberosities, at the upper extremities of the humeri, the heads and tuberosities of which are relatively enormous. The site of insertion of the deltoid muscle is exceedingly prominent. The neck of the femur is very short and forms an angle with the shaft which constitutes the condition of coxa vara, though of slight degree. The trochanters are enormous. The upper extremities of the fibulae enter into the articulations of the knee joints, as is the rule in achondroplasia. The lower extremities of the fibulae forming the external malleoli of the ankle joints extend abnormally far beyond the internal malleoli. The interosseous spaces of the legs and forearms are much increased in size. The bones of the forearms and arms show some degree of curvature, but this is less than is frequently seen in achondroplasia. The bones of the legs show very little curvature and such as there is occurs at the union of epiphyses and diaphyses and not in the shaft itself. Compare with bones of new-born infant shown in (17), with adults (6)—(8) and (11)—(13), and with radiogram (19). Radio-humeral index = 83·3. Tibio-femoral index = 70·0. (Measurements taken from the following points: Head of humerus to external epicondyle. Head of radius to extremity of styloid process of radius. Head of femur and great trochanter to margin of articular surface of internal condyle. Articular surface of internal tuberosity of tibia to extremity of internal malleolus of ankle joint.)

(19). Radiograms of skeletons of a normal female child, aged 9 years, and of an achondroplasic girl of the same age. Note the trunk is as nearly as possible of the same length in each. The head is nearly the same size in each. Measurement from vertex to upper border of symphysis pubis is approximately equal in each. All four extremities are shortened in the achondroplasic child. In the normal child the femora and humeri are much longer than the bones of the leg and forearm respectively. In the achondroplasic child the bones of the arm and forearm, thigh and leg are respectively as nearly as possible of equal length. The bones of the extremities in the latter are considerably thicker than in the former and in some instances the sites of muscular insertion can be seen to be hypertrophied (*e.g.* that for the deltoid); all their epiphyses are much enlarged. The hands show the "trident" deformity, the fingers diverging in extension and radiating like the spokes of a wheel. The bones of the feet are short and thick. The cranium shows prominence of the frontal eminence, it is high vaulted and platybasic, with a vertical occiput; the bridge of the nose is depressed and the upper part of the face is in retreat beneath the forehead; there is a slight degree of prognathism; the distance between the cervical spine and the pterygoid processes is, however, reduced in the case of the achondroplasic skull. Compare with this: (3), (4), (5), (6—8), (11—13), and (22).

(20). *Pseudo-achondroplasic-rickety foetus* (Véron's Case, see Bibl. No. 510). Curvature of the bones of the legs. Fractures of R. humerus, L. radius, and both femora (a part of the latter has been removed for histological examination). Thinness of bones of forearm. In the lower extremities, as in the achondroplasic, supplementary transverse folds of skin can be seen at the sites of curvatures and of fractures.

(21). *Normal foetus.*

(22). *Achondroplasic foetus.* Porak and Durante's Case. Extreme shortening of the long bones of the extremities which, nevertheless, are almost as thick as the corresponding bones of the normal infant. The epiphyses, which are very large and which form the greatest part of the bones, are not visible, as they are as yet unossified, but occupy the spaces seen between the bony extremities. Sigmoid deformity of radius very marked on right side.

(23). *Normal chondral ossification.* (*a*) Zone of proliferation of cartilage cells; (*b*) zone of columns of cartilage cells; (*c*) line of ossification; (*d*) marrow and bony trabeculae.

(24). *Achondroplasia. Longitudinal section of the upper epiphysis of the femur.* (*a*) Zone of indifferent cartilage; (*f, f'*) band of fibrous tissue separating the indifferent cartilage and the zone of the columns of cartilage cells; (*g*) vascular loop making communication between the vessels of the fibrous band and those of the bone marrow; (*s*) zone of columns of cartilage cells. The columnar arrangement is completely lacking and is replaced by a fibro-cartilage sown with cartilage cells, sparse and scattered without order. The inferior border of this zone is calcified in places (*c*) in the neighbourhood of the line of ossification, which is irregular; (*o*) bony trabeculae, large and well calcified without cartilaginous débris; (*m*) medullary spaces.

(25). *Congenital rickets* (*pseudo-achondroplasic*). (*a*) Zone of proliferation of cartilage cells; (*b*) zone of columns of cartilage cells, very clearly shown. The cellular columns, perfectly regularly disposed, are separated by a hyaline interstitial substance (matrix) which is more abundant than normal; (*c*) line of ossification beneath which are prolonged the persistent cartilaginous trabeculae (*d*); these, mixed with the bony trabeculae, constitute the bony layer.

(26). *Periosteal dysplasia.* (*b*) Zone of columns of cartilage cells very marked, longer and more regular than normal; (*c*) line of ossification produced by the individual opening of each of the cartilaginous columns into the medullary spaces; (*d*) bone marrow with large medullary spaces and narrow bony trabeculae well calcified.

(*Nouvelle Iconographie de la Salpêtrière*, 1905, pp. 502—503, Plate LIV, Porak et Durante.)

Plate V (27). Radiograms of achondroplasic hand. Note the hand is short and relatively broad. There is marked shortening of the metacarpal bones and phalanges of the middle digit so that this is hardly longer than the index. The ring digit is even more shortened and is no longer than the little digit—the metacarpal bone is the one chiefly affected in these two digits. The fingers are thus of nearly equal length, but the shortening has affected the middle and ring fingers more than the index and little fingers and the metacarpal bones of these more than the phalanges of the digits. The fourth metacarpal is the bone most affected. All these long bones approach more nearly to the shape of a square, rather than an oblong, than is the case in the normal; that is to say, they are shorter and relatively thicker than normal. The divergence of the fingers in extension ("trident" hand) is not very well shown in this case, only the index and middle fingers are divergent. The epiphysial ends of all these bones are enlarged. That of the radius is also enlarged. The styloid process and lower extremity of the ulna are present but are much shortened. The ulna does not extend far enough to articulate normally with the radius in the inferior radio-ulnar joint. Compare with this plate, (5) and (11—13).

(28). Radiograms of achondroplasic feet. The features are broadly the same as in the hand. The digits are of nearly equal length. Some digits are more shortened than others, *e.g.* the fourth, and the metatarsal bone is the one chiefly affected. The bones are relatively short and thick and have enlarged epiphysial ends. The feet do not show any divergence of digits corresponding to a "trident" hand.

(29). Radiogram of the achondroplasic skull. The cranium is very large relatively to the size of the face. The prominence of the frontal eminences, so usual in this condition, is not well shown in this particular case, and the parietal eminences cannot here be seen. The bridge of the nose is depressed and the upper part of the face is "in retreat" beneath the forehead by which it is overshadowed; there is, however (probably in consequence of this) the appearance of prognathism, *i.e.* the jaws appear very prominent. The measurement from nasion to inion is considerably shorter than in normal European skulls of the same cranial circumference. (Compare with (6—8) which show these features well.) The distance between the posterior border of the maxilla and the cervical spine is shorter than in the normal, owing to premature synostosis of the basi-spheroid and basi-occipital bones and cessation of growth there, with the result that the occipital condyles and the pterygoid processes are abnormally approximated. The skull is platybasic with vertical occiput. Compare this with (6—8), (14) and (19) which show many of these features.

Plate W (30). Skeleton of achondroplasic foetus at full term, from the Royal College of Surgeons, by kind permission of Professor A. Keith. Note (1) Large head with voluminous cranium and small face. The frontal and parietal bosses are very prominent. The bridge of the nose is much depressed. The skull is platybasic and with a vertical occiput. (2) Micromelia. All four extremities equally involved. The clavicles are longer than the humeri. The radii are longer than the humeri; the tibiae are longer than the femora. (3) All the diaphyses of the long bones of the extremities are very massive and curved. The radii show double curves, forming an S or Z-like shape. The interosseous spaces of forearms and legs are much increased in size. (4) The fibula enters into the articulation of the knee joints. (5) The "main en trident" or trident hand is well shown. (6) The back is very straight, the lumbar concavity being practically absent. This skeleton was mounted without disarticulation and none of these appearances are artifacts. Owing to the size of the cranium this case was at one time regarded as hydrocephalic.

(31). Achondroplasic foetus. Note the large head, with prominent frontal and parietal eminences, vertical occiput and depressed nasal bridge; the trunk is not shortened but the extremities are markedly shortened; the segments of these are sausage-like (see p. 374); the fingers radiate like the spokes of a wheel and are all of nearly equal length. Note the peculiar conformation of segments of the digits. The feet are rather short and are broad and square and the toes are of nearly equal length. The back is straight, the buttocks and abdomen prominent. All skin folds are exaggerated. The limbs are somewhat curved, but this feature cannot be seen well.

Plate X (32). Radiogram of achondroplasic foetus, from the Royal College of Surgeons, by kind permission of Prof. A. Keith. Note the conformation of the skull already described, the shortening of the bones of the extremities, particularly the femur and humerus, the sigmoid curvature of the radius, and the shortening of the fingers, which are of equal length. The back is straighter than the normal, the dorsal and lumbar curvatures being lacking there is on the contrary a slight lumbar kyphosis. The sacrum is tilted. Centres of ossification are lacking for the bodies of all the cervical vertebrae (except for odontoid process and for II) and for the coccyx. There are no centres of ossification for the epiphyses of any of the long bones except the head of the femur.

Plate Y (36)—(37). An achondroplasic female child (37), aged 9 years. Typical features of achondroplasia (compare with (4)). Note shortening of extremities, characters of this as already described, peculiar hands; prominence of buttocks and abdomen and appearance of lordosis (pseudo-lordosis), shape of head, bridge of nose, etc. In contrast to this is (36), a woman, aged 42 years, showing the dwarfing of growth due to infantile myxoedema. In the former the mid-point between the vertex and soles of the feet lies midway between the umbilicus and the xiphi-sternal articulation; in the normal adult it lies at the upper border of symphysis pubis; in (37) it lies about midway between the latter and the umbilicus; this measurement represents the relative length of limbs to trunk and head of the normal infant and young child, *i.e.* in this dwarf there is practically no micromelia; the finger tips reach to the middle third of the thighs as in the normal. The segments of the limbs present the normal proportions in length to one another and there is no disturbance of this like that shown in (37). The arms are not held abducted from the sides in extension as in (37). There is no great prominence of buttocks as in (37), except such as is due to the general obesity, which is very marked, and though the abdomen is prominent this is due to the same cause. There is no lordosis as appears in (37). The frontal eminences are not prominent, the bridge of the nose not depressed. The facial expression, though not very marked, is heavy and toad-like. The condition of the hair, dry, sparse and brittle, the spade-like hands and the skin typical of myxoedema and cretinism cannot be shown in a photograph such as this.

(33)—(35). For general comparison a case of family rickets has been reproduced showing the dwarfing of growth, deformities and abnormal proportions produced by this condition. This is to be contrasted with any of the achondroplasic dwarfs shown in our plates. There is no real shortening of the long bones of the extremities, but they are curved and bent and the lower extremities appear shortened in consequence (pseudo-micromelia). As a result of the curvature of the lower extremities the hands reach abnormally far down the thighs. The bendings, curvatures and deformities are bizarre; they are the result of gravity, of the influence of the body weight upon unduly soft growing bone, brought to bear in some instances, in faulty attitudes. The curves involve the *shafts* of the long bones and are "en grand arc"; they occur in various directions. In achondroplasia, curves when present are "angular," involve the region of junction of epiphyses and diaphyses, are slight, have convexity outwards, are usually more of the nature of partial displacements of epiphyses than true curves and the shafts themselves, in all cases except that of the achondroplasic infant, are practically straight. In these figures of rickety dwarfs the arms, not having been involved in the support of body weight, are comparatively straight and have no appearance of shortening like that shown by the legs. Indeed in some cases they appear abnormally long in contrast. The shape of these heads is in no way peculiar and the bridge of the nose is not depressed in any case. Two cases show real lordosis, but in them the buttocks are not very prominent. In two the abdomen is prominent, in the elder possibly as an effect of lordosis, in the youngest it is probably merely the tumid abdomen of still active rickets. They show the deformities known surgically as coxa vara, genu varum, genu valgum, curved tibia, statical scoliosis and cubitus varus.

C. Dwarf Growth of Pathological Origin: Ateleiosis or "true" Dwarf Growth.

Cases of ateleiosis ("nanisme vraie," "echter Zwergwuchs," or the true dwarf growth of foreign writers) fall into three groups according to the age at which the growth change commences and the particular features which, in consequence of this, they exhibit. This was shown by Hastings Gilford, to whom the whole credit for this classification of cases is due. They present three degrees. In Group I there is evidence that the change began before birth. The only certain case of this group is the one of which the skeleton is here shown. (Caroline Crachami. History and clinical features quoted in Fig. 717 as recorded by Sir Everard Home. Some of the other cases possibly belong to this group but this can only be conjectured; "grouping" is, of course, an artificial process for purposes of convenience. It will be clear that the "groups" shade off into one another as do "groups" of cases of other conditions.) In Group II the growth change begins in early infancy and the characters shown correspond, broadly, to this age. In Group III the growth change begins in later childhood but before puberty; the characters shown correspond, broadly, to that age. It is held by some observers that there may be a further fourth group. But this is problematical, and there is no such case shown in the following series of photographs. Most cases of true dwarf growth or ateleiosis show "infantilism," but this is not invariable—thus of two brothers in Group II, aged 60 and 62 years respectively, each of height 3′ 9″, one shows "infantilism," but the other (Plate CC (48)) does not[1]. (The term "infantilism" is defined on page 368.) (61) shows ateleiosis in the equine species as well as the condition (Groups II and III) in the human subject. In the following series of photographs the order of arrangement follows the above grouping; the first in each group shows the condition contrasted with the normal individual of as nearly the same size as could be obtained.

[1] [It is by no means easy to discriminate with respect to infantilism between Ernesto and Primo Magri; in facial expression they are now almost interchangeable, and no medical examination has been reported since that of 1865: see our p. 502. Editor.]

PLATE Z (38). Caroline Crachami and a normal child. The proportions shown by these two skeletons are nearly the same, but the head of the dwarf is, relatively to height, larger than that of the normal child. The following table shows their comparative proportions. (Since nearly all epiphyses are lacking in both, measurements of diaphyses alone are considered in relation to measurements of head and trunk and total stature.)

Normal Infant	Ateleiotic Child
$\text{Index} = 100 \times \frac{\text{Length of radius}}{\text{Length of humerus}} = 100 \times \frac{10}{12} = 83{\cdot}3$	$100 \times \frac{8}{10} = 80{\cdot}0$
$\text{Index} = 100 \times \frac{\text{Length of tibia}}{\text{Length of femur}} = 100 \times \frac{13}{15} = 86{\cdot}7$	$100 \times \frac{11}{13} = 84{\cdot}6$

The comparative measurements of these two skeletons from vertex of skull to margin of inferior surface of os calcis is as 40 is to 34. Made up as follows:—

Vertex to upper border of symphysis pubis 23	19
Upper border of symphysis to margin of lower surface of os calcis 17	15

In these proportions the following measurements occur:—

Episternal notch to upper border of symphysis 12	10
Vertex to episternal notch 11	9

The mid-point between vertex and inferior surface of os calcis is above the symphysis and is at the centre of the body of the 5th lumbar vertebra in each case. The proportions of these two skeletons are thus nearly the same, and differ only in the fact that the intermediate segment of the limbs is relatively shorter in the ateleiotic, a condition which approximates, more nearly than the infant's does, to the condition in the normal adult. Both show the ordinary proportions of infancy and childhood in the respect that the lower extremities are a little shorter in proportion to total height than in the normal adult[1]. The ossification of the cranial membrane bones is apparently as far advanced as the normal, and so is the part of the clavicle (*i.e.* the whole of the shaft and the acromial end) that developes in membrane. The lower jaw also appears to be normally advanced as regards ossification; but this again is mainly a membrane, bones being developed primarily in the tissue investing Meckel's cartilage. Bony union of the symphysis occurs in the second or third year and has occurred here. But the angle of the jaw is very open, as in the foetus (contrast with that of the child shown, young as this is). The epiphysis for the sternal end of the clavicle is not present, but this does not normally appear until the 18th to 20th year. The ossification of all other bones is greatly retarded and for the most part less advanced than in the case of the normal infant shown. Thus the only epiphysis present is that for the head of the femur which normally does not appear before birth. That for the lower end of the femur, which normally appears before birth, though present in the infant skeleton shown, is lacking in the case of the dwarf. The three centres for each of the innominate bones of the pelvis (namely one for the ilium, one for the ischium and one for the pubis), normally present at birth, are here present, but union between the rami of the pubis and the ischium has not yet occurred. This normally takes place at about the 8th to 9th year and should therefore, probably, have appeared if ossification were normal. At her age (9 years) the following centres should be present, but are all lacking: one for the head of the humerus (normally appearing in the first year of life), one for the great tuberosity (3rd year), one for the lesser tuberosity (5th year), the last two should be united (7th year), one for the capitellum (5th year), one for internal epicondyle (7th year), lower end of radius (2nd year), upper end of radius (5th year), lower end of ulna (5th year), centres for all the bones of the carpus (1st to 8th year) except the pisiform (12th). Ossification should be present in the epiphyses for all the metacarpal bones and phalanges of the digits (3rd to 5th year), for the great trochanter of

[1] Such proportions of infancy and childhood are maintained through life in ateleiosis, that is to say these individuals never attain the normal adult relative proportions of length of extremities to head and trunk, however old they may live to be. (See accounts of individual cases.) The difference is slight, however, and becomes less as age advances and growth slowly proceeds. The same is seen in cretinism and other varieties of dwarf growth that present bone changes like these. It is in marked contrast to the state of affairs in achondroplasia.

the femur (4th year), upper extremity of tibia (normally present at or soon after birth), lower extremity of tibia (2nd year), lower extremity of fibula (2nd year), upper extremity (4th year), the epiphyses of all the bones of the tarsus and metatarsus (normally occurring in all at the end of the 4th year) and the epiphyses of all the phalanges of the digits (8th year). There is no sign of ossification of any of these epiphyses nor of its appearance in the bones of the carpus or tarsus except in the os calcis and astragalus (in which centres normally appear in the 6th and 8th months of extra-uterine life respectively). There is no sign of a patella (here ossification normally begins during the 3rd year). The tibia shows no tubercle (for attachment of the ligamentum patellae) and no sign of a crest, being cylindrical in section instead of triangular. Thus the bones in which, at the time of death, ossification could be said to have occurred normally are the bones of the cranial vault, the parts of the clavicles present and possibly the pelvis. The vertebrae also cannot be stated to be defective in ossification to date. In the case of the ribs centres for the head are present in all. These usually only appear some years later (at puberty). So that the ribs are in advance of the normal in this respect. Dentition corresponds to the normal for the end of the 2nd to the 6th year. All the bones are very thin, light and smooth, and show no markings for muscular attachments. Although the latter could hardly be marked at her age yet the bones are actually smoother than those of the infant shown. The processes of ossification, both chrondral and periosteal, have thus been for the most part reduced to a minimum, or, with the above exceptions, brought to entire abeyance since the time of birth.

(40). Skeleton of ateleiosis in a male (Nicholas Ferry, "Bébé") who died aged 22 years. Height measured at death was 89 cm.[1] If this skeleton be compared with that of (38), who died aged about 9 years, it shows the difference that centres of ossification for all the epiphyses are present and that chrondral ossification seems to be as far advanced as the normal for the age. Both patellae are present. With a magnifying lens it can be seen that the epiphyses of the long bones of the extemities are not united to the diaphyses. But this cannot be definitely stated to be abnormal at this age. As in (38) all the bones are exceedingly thin and light. They are smooth and show practically no markings of ridges and grooves for muscular origins and insertions as occur in the normal. All the tuberosities are very ill developed. The crests and spines of the ilia, and the tubera ischii are very little developed, and the pubic bones are very thin and light; the pelvis, except for the fact that processes of ossification are, as regards time, more advanced, resembles that of a young child. The vertebrae and ribs, with the above exceptions as to lightness, thinness, etc., appear to be normally ossified for age. The skull shows the following peculiarities: both jaws are edentulous and whether as cause or effect of this, or not, their alveolar margins are exceedingly ill developed. The lower jaw, *as regards its angle*, resembles (38) in showing the peculiarity that it approximates to that of the foetus (though not to the same degree as (38)) more nearly than does the lower jaw of the young child shown. The mental foramen is situated almost at the alveolar margin of the jaw, thus resembling the condition in the edentulous jaw of old age and differing from that of the infant. The nasal bones are markedly prominent but this would not appear to have any pathological significance and is probably an individual variation or peculiarity (within the normal) or it may be a racial feature. The shape of the cranium resembles that of (38). It is quadrate, and brachycephalic with a breadth/length index of 83·3, and should be compared with those of other ateleiotic dwarfs here shown. The height/length index is 91·6 approximately. The general proportions approximate to those of infancy or early childhood, the mid-point between the vertex and the soles of the feet falling well above the upper extremity of the symphysis pubis. The femora are, however, relatively a little longer than in the infant, as is also the case in (38). The tibio-femoral index is 75·0, the radio-humeral index is 57·14[2]. In brief it may be said that the peculiar features shown are nearly the same as those of (38) but are all less marked, probably because this individual (39) was of adult age while (38) was aged about 9 years at time of death. These skeletons should be contrasted with those of achondroplasia shown, when the marked differences in the relative length, thickness and curvature of long bones (in the latter condition), shape of skull, proportions of length of limbs to trunk, etc. etc. will be clearly seen.

Group II. Plate AA (41). Male ateleiotic dwarf, aged 28 years. Height 3′ 7″. Standing between an adult man of medium height (seen only in part) and a normal boy of 6 years. "The physiognomy and proportions are childish and the sexual organs infantile while the attitude, expression and markings of face are suggestive of age." (Hastings Gilford.) The muscular development is very feeble, as of a child; muscular outlines are very feebly marked; they are those of childhood; except for this, however, the shape of the figure is that of a later age. Though the proportionate length of lower extremities to total height is that of childhood the lower extremities are considerably longer, proportionately, than in the child by his side. The tibio-femoral index, *i.e.* 100 (length of tibia)/(length of femur) is as follows in these two: In the normal child = 100 × 8/10. Index = 80·0. In the ateleiotic individual these bones are as 9 is to 12. Index 75·0. As in Cases (38) the femur is proportionately longer in the ateleiotic case

[1] [The skeletal height is 92·5 to 93·5 cm. but we think has been exaggerated in the setting up. Hastings Gilford (Bibl. No. 664, p. 638) states that the skeleton is 93·5 and "his height at death must therefore have been quite 95 cm." Knowing how skeletons often are set up, we do not agree wholly with the "therefore." Editor.]

[2] [I am unable to verify Dr Rischbieth's value; it would, roughly from the photograph of the skeleton, appear to be nearer 72·0. Editor.]

than in the normal child of about the same height and in this respect approximates more nearly to the condition in the normal adult.

(42). Female ateleiotic dwarf, aged 18 years. Height 2′ 9½″. "A normal adult hand is introduced for the sake of comparison. Note the infantile physiognomy and the crowded teeth. A radiogram showed that ossification was equal to the normal for 6 years." (Hastings Gilford.)

(43). Heads of 17 cases of ateleiosis of the second group. "Their proportions and facial characters are childish, though they show the superficial markings of age."

The following table gives the names of these dwarfs as far as we have been able to ascertain them.

<table>
<tr><td>Minnie Warren
pp. 361, 568
Bibl. No. 240
= (117)</td><td>Commodore Nutt
pp. 361, 568
Bibl. No. 240
= (117)</td><td>French Dwarf
= (42)</td><td rowspan="2">Prince
Mignon
= (118)</td></tr>
<tr><td></td><td></td><td>Annie Nelson
(Laible)
p. 406, ftn.</td></tr>
<tr><td>Boruwlaski
pp. 360, 404
Fig. 693
= (67)—(68)</td><td>?</td><td>?</td><td>Franz
Rossow
Fig. 697</td></tr>
<tr><td rowspan="2">Baron
Ernesto
Magri
p. 406
Fig. 690
Bibl. No. 248
= (48)</td><td rowspan="2">Count Primo
Magri
p. 406
Fig. 690
Bibl. No. 248</td><td rowspan="2">Lavinia
Warren
Countess Magri
pp. 361, 568
Bibl. No. 240
= (120)</td><td>Karl
Rossow
Fig. 697</td></tr>
<tr><td>?</td></tr>
<tr><td rowspan="2">Franz and Karl
Rossow
Bibl. No. 332
Fig. 697</td><td colspan="2">Mulatto Dwarf,
Chiquita</td><td>= (41)</td></tr>
<tr><td>Charles Stratton
(Tom Thumb)
pp. 361, 568
Bibl. No. 240
= (120)</td><td>Lavinia Warren
(Mrs Tom Thumb)
pp. 361, 568
Bibl. No. 240
= (120)</td><td></td></tr>
</table>

Photographs (41)—(43) are reproduced by kind permission of Mr Hastings Gilford and the Royal Society of Medicine.

Plate BB. Tyrolese Dwarf (44). Josefa Prinz (see Fig. 689), aged 26 years, height 109 cm. (in shoes), and her mother, aged 70 years, of normal size. Josefa is "well formed, has graceful limbs, animated, quick and precise of movement, of friendly disposition and pleasant facial expression. The shape of the head is not in the least peculiar, the thyroid gland not enlarged; her voice is childish; she sings well. Her intelligence is obviously quite normal." "She follows dress-making as a trade, and supports herself and her mother thereby; she is regarded as a very stylish tailoress. She has frequently exhibited herself in Munich, Innsbruck, Bozen, and St Moritz." She differs from the cases (41) and (42) in that her facial appearance and expression are not infantile or childish but are those of an adult and so, as far as can be judged, are her proportions.

(45). Rudolf Prinz (see Fig. 689), aged 24 years, height, in shoes, 104 cm., without shoes hardly 100 cm. "Shape of head quadrate, parietal eminences very prominent, the transverse interparietal diameter very great. Bridge of nose depressed, slight moustache. Voice childish, somewhat squeaky, no enlargement of thyroid gland; skin of face wrinkled. Movements animated and precise." Ulrich Prinz (brother of the last), aged 22 years, "of the same height and presenting the same features except that he shows no trace of moustache." "With these is their eldest brother, of normal proportions (height 5′ 8″

in shoes). Rudolf is a tailor by trade but does not work regularly at it because there are enough tailors in the valley (Innthal) already." These two cases differ from the last in the shape of the skull and the facial appearance, both of which are childish or infantile.

(46). Susanne Kleinstein or Jenal (see Fig. 689), aged 28 years, height 108 cm. "She, it is true, is of somewhat simple disposition, but grosser defects of intelligence are not to be observed in her either. She has a very big cranium, the bridge of the nose is depressed and broadened, the eyes are wide apart, the skin of the face is wrinkled, the neck very short, there is no enlargement of the thyroid gland. The extremities are well proportioned, movements in every way normal."

(47). (*a*) Julius Kleinstein, aged 30 years, height 108 cm. in shoes. "A large quadrate skull, forehead bulges somewhat forwards, bridge of nose depressed, slight growth of hair upon upper lip, neck short, skin of face wrinkled, no enlargement of thyroid gland, deep, somewhat peculiar voice. All limbs well proportioned, movements quick and accurate. Friendly disposition, normal intelligence. His trade is that of tailoring and he conducts a business of his own in this. (*b*) Marie Kleinstein, sister of the last, aged 26 years, height 93 cm. "Very large angular skull, forehead bulging forwards, bridge of nose depressed, neck very short, skin of neck wrinkled. No enlargement of thyroid gland. Limbs graceful. Movements very quick and precise. No defect of intelligence can be observed; did very well at school. Occupation housework." (*c*) Julie Kleinstein, sister of the two last, aged 14 years, height 86 cm. "She shows just the same features as her sister Marie, except that the cranium is proportionately even bigger than in the latter. She is still at school where she is doing very well." In the last three cases the quadrate form of the skull, with its bulging forehead and depressed nasal bridge, is more clearly shown than in the other cases. These features are also very well shown in the next case and in his brother. (44) to (47) are from photographs kindly provided by Dr Schmolck.

Plate CC (48). Ernesto Magri, a native of Italy, aged 62 years, height in shoes 45″ (see Fig. 690). A large quadrate skull with bulging forehead; bridge of nose somewhat depressed; a considerable growth of hair on upper lip. Neck very short; skin of face and neck much wrinkled but not dry, cracked or in any way abnormal. A peculiar "waxy" colour much like that seen in pernicious anaemia. Thyroid gland felt, not enlarged. Pomum Adami hardly palpable. Voice high pitched and squeaky; "thin" or "piping." Proportions those of early childhood, *i.e.* head relatively large, neck short and limbs relatively short for the body, all well formed. Movements accurate and precise and certainly not slow, but all performed with a curious air of deliberation. Hands proportionately neither larger nor smaller than normal, but they have the shape of those of the infant or young child (compare case (41)), this is shown in the comparative shortness of the fingers, breadth of the hand, its absence of muscularity and "character" or "expression," and the shape of the finger nails; the skin of the hands is much wrinkled but not dry, cracked, or in any way abnormal. Teeth normal and all are still present except the third molars, which have never erupted. A music hall artist. Intelligence good; a man of some education; speaks English fluently with only a trace of accent, writes it idiomatically (in reply to negotiations with a view to this photograph) in a clear, firm hand showing individuality and which only an expert could distinguish from that of any ordinary man of his age. He is somewhat nervous or timid but shows no other peculiarities of disposition. A brother aged 60 years is of the same height and shows the same features in all respects except that there is no trace of hair about the face. Neither of these individuals has suffered at all generally from ill health and has had no sickness of note except measles and chickenpox in infancy. Family history of disease negative.

(49). Ateleiotic male, aged 22 years, height 3′ 3″ approximately. Shows the same features as (47) and (48).

(50). Heinrich Glauer, aged 24 years, and Bruno his brother, aged 20 years, both of height approximately 3′ 2″. Both show the same features as (47) and (48) with modifications. Large quadrate skull with prominent eminences and bulging forehead; bridge of nose depressed. The facial appearance of early childhood but showing the markings of age. There is, however, no increase of subcutaneous tissues or marked wrinkling of skin. Neck short. Relatively short extremities (the proportions of early childhood); hands of the same period. No trace of hair about the face. Voice higher pitched, squeaky and "thin" or "piping." Both have a peculiar "waxy" complexion much like that of pernicious anaemia. Thyroid gland palpable, not enlarged. Pomum Adami very little developed. All movements quick and precise. Intelligence shows no defects. Both read and write well in their own language (German), but speak no English or French. Answer questions promptly, quickly and clearly and convey the impression that they are particularly "cute" (if the expression may be employed). Show no timidity or other peculiarities of disposition. Both are more muscular than most cases of this class. Neither has suffered from general ill health and neither remembers having had any "illness."

Plate DD (51). Ludwig Ulpts, a German dwarf, "the smallest man in the world," aged 18 years. Height 34″. Head very large, quadrate; frontal and parietal eminences very prominent. Forehead bulging, bridge of nose depressed. Neck short. Thyroid gland felt, not enlarged. Pomum Adami not

developed. Proportions and facial appearance those of infancy, as are the hands. Limbs and body very thin and weak. No trace of hair about face. Voice high pitched, "thin" or "piping" and squeaky. Intelligence good. Reads and writes well and speaks English slightly. Cephalic index 77·9; *i.e.* skull is mesaticephalic; height index 77·9. The mother of this individual is a typical achondroplasic woman of height about 3′ 3″. Her sister and niece, the daughter of the last, are achondroplasic and of the same height (3′ 3″). See Boeckh's Case, Figs. 608 and 620. The father of Ludwig Ulpts is said to have been a typical ateleiotic dwarf of height about 3′ 6″. His photograph is shown, with these other individuals, in the section on achondroplasia. The depressed nasal bridge which Ludwig shows is somewhat like that of achondroplasia, but there are no other symptoms of that condition; he presents no other resemblance whatever to an achondroplasic individual. See Plate FF (62) where father and mother are also shown.

(52). Otto (?Bottcher, see p. 406), a German dwarf, aged 21 years, height in shoes 36″ approximately. Head large[1], cranium quadrate, forehead bulging, bridge of nose depressed; facial appearance as well as shape of head and its proportional size to rest of body those of early childhood, but face shows the markings of age. The colour of the face is peculiar; it is of a "waxy" lemon-yellowish appearance, much like that seen in pernicious anaemia. Neck short as in infancy. Trunk and extremities well formed, but the proportions are those of infancy, not of adult age (*i.e.* the extremities are relatively rather short). The hands are also of childish proportions (compare case (41)). Voice childish, high pitched, "thin" or "piping" and rather squeaky. Thyroid gland palpable, not enlarged. Pomum Adami hardly developed at all. Movements certainly not slow and quite precise but are carried out with a curious air of deliberation difficult to describe, but quite obvious at a glance. Music hall singer. Intelligence good. Reads and writes well and speaks English very well. Dentition normal, teeth sound but no third molars (age, however, only 21 years).

(53). Forgéres, a Frenchman, aged 35 years; another example of this condition, height 3′ 9″. Large square head, with prominence of frontal and parietal eminences. Short neck and childish proportions. Skin of face wrinkled. Slight growth of hair on upper lip, etc. His facial appearance is not childish but is that of a young adult. Cephalic index 80·0, *i.e.* he is verging on the brachycephalic. Height index 80·0 approximately.

(54). Smaun Sing H'poo, Burman, aged 26 years, approximate height 3′ 1″. This case differs considerably from all the others shown above except Case (44) (Josefa Prinz, which it broadly resembles), in that the proportions of the size of the head and length of neck and extremities to trunk, as well as the facial appearance, are those of early adult life (corresponding to an age, however, less than his is), and not of infancy. The shape of the head is markedly different from those cases; it is not quadrate and shows no great prominence of eminences; the forehead is not bulging but, rather receding; the bridge of the nose is not depressed[2]. The size of the head compared with that of (52), of about the same height, is as follows: length is as 8 is to 10; breadth is as 7 is to 8; while height is as 7 is to 8, in (52) and (54) respectively. The neck is not very short and the proportions of length of extremities to total stature are those of an adult and not those of infancy or early childhood. The hands also show the shape and proportions of the adult, the fingers being relatively long. The limbs are well formed but they and indeed the whole figure is slighter than in the above cases. No trace of hair about the face. Pomum Adami very little developed. Thyroid gland palpable, not enlarged. Voice high pitched, "thin" or "piping" and squeaky. When observed he made demonstrations of affection towards a small female dwarf whom he hugged and kissed in public[3]. Movements remarkably quick. Quite intelligent. Speaks English very well for a coloured alien. Juggler by profession. The size of the head, which, compared with other cases, is relatively small, its shape, the receding chin and the quickness of movement shown suggest the possibility of microcephalic dwarf growth here. But there is no idiotcy; the intelligence is not in the least defective in any way. The case is possibly one of the same type as that illustrated by (38) in which the general hypoplasia affects the cranium and cerebrum to the same extent as other parts (though to a less extent than in (38)); in that case there was quickness of movement and other features suggestive of microcephaly but the head is, actually, *bigger* than normal for stature and in the present case it is certainly not less than normal. Many of the differences from the above cases here shown may be due to the occurrence of the disease in a different race (Burmese), but not all can be due to this cause because (42) shows similar features in many ways, *e.g.* adult facial appearance, adult proportions, etc.

Plate EE (55). Four English dwarfs belonging to Group II of the ateleiotic class. The two elder were known as "Mr and Mrs Tom Thumb Junior."

[1] The cephalic index of this skull is 80; *i.e.* it is verging on the brachycephalic. The height index, as far as this could be measured, is also 80, *i.e.* the craninm is a relatively high one (but this measurement is only approximate).

[2] The cephalic index is 87·5; the cranium is thus brachycephalic. The height index is 87·5. [The equality of the breadth/length and height/length cephalic indices in all the cases (51), (52), (53) and (54) is in accordance with Dr Rischbieth's manuscript and is given on his responsibility. Editor.]

[3] The Burmese are Mongols at least in part and their facial and cranial characters are the same as the Southern Chinese, *i.e.* they are either mesaticephalic or brachycephalic. In the difference in the size and shape of the head and some of the other features which mark this case off from the others it seems possible that these may be due to race alone.

Group III. (57). Martin Lane, aged 28 years, height 4′ 9″, and his brother, aged 13 years, and a normal adult. "The ateleiosis (or alteration in growth) began at the age of 14 years. Note absence of sexual hair, childish sexual organs and youthful aspect and proportions combined with the weathering of age" (Hastings Gilford). The musculature is very feeble. The length of lower extremities though proportionately greater than in the last group is, however, shorter than in the average normal adult and is approximately the same as in the boy of 13 years. Thus the proportions are (1) in the ateleiotic dwarf: length of lower extremities/total height = 121/264, (2) in the boy of 13 it is 120/264. The length of the femur compared to length of tibia is relatively greater in the ateleiotic, thus approaching more nearly the condition in the average normal male adult. The proportions are (1) in the ateleiotic dwarf: length of femur to length of tibia as 7 is to 5, (2) in the boy of 13: they are as 6 is to 5. Thus in (40), in (41), and in this case the ratio of length of femur to that of tibia is not the same as in the child of the same approximate stature, but is more nearly that of the adult, *i.e.* the femur becomes relatively longer. As well as this, as will be seen, the lower extremities as a whole become proportionately longer in this group, following the ordinary alterations of proportions as age advances, though not to the full extent.

(56). Reinhold Tschuschke (Tyrol: see p. 406 ftn.), aged 36 years, of about 4′ 9″ height. Quite intelligent, reads and writes well but only in German.

(58). Gustav Geschke (Berlin: see p. 406 ftn.), aged 52 years, height about 4′ 9″. Quite intelligent, reads and writes English and German well, and speaks English well.

(59). Three cases of ateleiosis Group II and two of Group III placed together for contrast. The smaller figures are those of (50) and (52), the larger those of (56) and (57).

Plate FF (60). Large group of ateleiotic dwarfs with four of achondroplasia. These and others were exhibited at Olympia, London, 1909—10.

(61). Ateleiosis in the equine species. Note the foal-like appearance of these ponies; their very small size, thickness and general immaturity. They differ markedly from the ponies of normal growth (such as Shetlands), being little heavier than greyhounds of medium size; they have less than a tithe of their strength and endurance. (The man in the background is 6′ 2″ in height.) The smaller human dwarfs belonging to Group II, are shown in Figs. 50 and 52. The larger human dwarf belongs to Group III and is aged 30 years.

(62). Ludwig Ulpts, shown in (51) and (61) and his father and mother. These are also referred to in the Pedigrees (Achondroplasia, Boeckh's Case, Figs. 608 and 620). The sister of this woman and her daughter are also shown in the illustrations of achondroplasia (Plate Q (9)) and the first also appears on the plate showing achondroplasic types (Plate S (14) F., F.').

Plate GG (63) and (64). Cretinism in a brother and sister, aged 28 and 25 years respectively. Reproduced from photographs kindly sent by Professor G. R. Murray. The first is 4′ 5″ in height; his sister is 4′ ½″. Distinguished from the above condition by mental deficiency (it can be seen that these two are idiots), bodily lethargy and slowness of movement and other features. The skin is coarse, dry, scaly, thickened and wrinkled, the hair coarse, short, brittle, dry and sparse. Note the broad nose and flabby cheeks. The eyelids are swollen, with a solid oedema, and the hands and feet show a similar condition. Both these individuals, but especially the female, show the presence of characteristic fatty swellings on each side of the neck, and the female shows a marked goitrous enlargement of the thyroid gland. Note in the male the wide open mouth, with cracked and fissured lips, dribbling saliva (in many cases the tongue is held protruded). Note in the female the flabby pendulous mammae and in both the tumid adipose abdomen. Their proportions are those of children, as in ateleiosis; but the features enumerated render the two easily distinguishable. This condition is due to (*a*) congenital absence, partial or complete, of the thyroid gland, with consequent defect of its secretion, (*b*) to its atrophy or operative removal in childhood with the same result, or (*c*) to the advent of a goitrous enlargement of the gland (goitre, "Derbyshire neck" or bronchocele), with the same result[1]. Cretinism is sporadic as in these cases, or endemic, as in certain parts of Europe, Asia and America which are mountainous, *e.g.* Switzerland, Savoy, Tyrol, Pyrenees, Himalayas, etc.

[1] It must be stated however that the advent of goitrous enlargement of the thyroid gland, if this occurs in early childhood, does not necessarily produce cretinism. Most cases of cretinism show no goitre and most cases of goitre show no cretinism. It is defect of the internal secretion of this gland during the period of growth that determines the occurrence of cretinism. It is only when a goitre produces or is associated with this defect at this time that it is associated with cretinism.

D. Portraits of Famous Exhibition and Historical Dwarfs[1].

It has not been considered necessary to discuss the majority of these individuals at length. We give in general only references to their mention in the text of the *Treasury*.

Plate HH (65). Mademoiselle Anita. A Hungarian dwarf, said to be 25″ tall and 25 years of age. Exhibited throughout England and Scotland in 1911. She belongs to Group II of the ateleiotic dwarfs, but has the adult face of Josefa Prinz (44) and Boruwlaski (67): see p. 363.

(66). Jeffrey Hudson, from the portrait by Daniel Mytens in the National Portrait Gallery: see *Iconography* (45). If the portrait was painted in 1637, Hudson was then 18 years of age. See p. 360. Hudson belongs to Group II of the ateleiotic dwarfs.

Plate II (67) and (68). Count Joseph Boruwlaski from Bonomi's life-size cast taken from life, in the Museum at Durham: see *Iconography* (122). Boruwlaski was said to be 98 when this statue was made. He shows, however, the same adult face in earlier portraits[2]. He belongs to Group II of the ateleiotic dwarfs: see pp. 360, 404 and Fig. 693. He was 3′ 3″ in height.

(69). Nannette Stocker and Johann Hauptmann, famous show dwarfs of the late 18th and early 19th century (London, in 1815), and belonged to Group II of the ateleiotic dwarfs. Nannette Stocker was born about 1782 at Kammer in Upper Austria; she was a very fine child at birth, but ceased to grow at the age of 4 years. At the age of 33, she was said to be 33″ high, and to weigh 33 pounds. Her mother and brother were of normal height. Johann Hauptmann was born about 1778, of normal parents, at Ringendorff, Lower Rhine. He met Nannette at Strassburg in 1798, and from this time the two of them travelled about Europe together, earning their living by performing, Nannette on the piano and Johann on the violin. Both were well proportioned: see Kirby's *Wonderful Museum*, Vol. v. pp. 228—9 and *Iconography* (154).

(70). Thérèse Souvray, the betrothed of Bébé. She called herself Madame Bébé. She and a dwarf sister were born of normal parents. She was 73 years of age, when Virey made the sketch of her from which our cut is reproduced, and her height was 86·4 cm. She appears to have been healthy, active and well proportioned, and probably she as well as her sister belonged to Group II of ateleiotic dwarfs. Her face is not infantile: see *Iconography* (140 and 141).

Plate JJ. See below in Section E.

Plate KK (73). George Romondo, Jewish rickety dwarf. Raymondo or Romondo was born about 1765 of Jewish parents in Lisbon. He was about 3′ 6″ in height. He was a very clever eccentric mimic and obtained his living in England by imitating in public places the cries of different animals or the sounds of musical instruments. See Kirby's *Wonderful Museum*, Vol. III. pp. 113—6. See also *Iconography* (156[a]).

(74). Owen Farrel, the Irish dwarf, born in County Cavan, Ireland, 1716. He acted as a footman for a time, but subsequently begged in the streets of London. He was 3′ 9″ high. He sold his body to Mr Omerod a surgeon, and later "Dr Hunter" became possessed of one of his thigh bones, which measured only 9·5″ (?which Hunter). He was remarkably strong and could carry four men, two sitting astride on each arm. We have been unable to find any trace of either skeleton or femur in the Hunterian museums in London and Glasgow. The prints of Farrel seem to indicate a special form of achondroplasia, but this has been questioned: see Kirby's *Wonderful Museum*, Vol. v. p. 364. There are many prints: see *Iconography* (155, 158).

(75). The family Kostesky, probably ateleiotic in Group II. The four eldest children are said to have ceased growth in their fourth year. For a further account see Bibl. No. 232 and Pedigree, Fig. 744.

(76). Wassilievitch, a Russian myxoedematous (?) dwarf, aged 51. He was the youngest of a family of six, all the others of normal build and healthy. He had a wrinkled, beardless face, and was quite intelligent, although he had had little or no education. Achondroplasia and probably cretinism seem excluded: see Bibl. No. 198.

Plates LL—PP. See below in Section E.

Plate QQ. See below in Section G.

Plate RR (97). Simon Paap was born at Landvoorst, in Holland, in 1789. His father was a fisherman, and he had two brothers and two sisters of normal height. Simon ceased to grow at three years of age; his height was 28″ and his weight 27 pounds. He was well proportioned in limbs and body, but his head was rather too large. He probably belonged to Group II of ateleiotic dwarfs, and may be

[1] [While Dr Rischbieth is not responsible for the final form of Sections A, B, C of these Descriptions of Plates—corrections and additions having been made to his manuscript—Sections D, E, F, G are based on material collected and arranged in the Eugenics Laboratory. Editor.]

[2] The facial appearance in this dwarf is much less infantile or childlike than is usual in this condition even in the aged, and is, indeed, that of an adult. The fact that he was perhaps older, at the time the cast was made, than were most of the other cases shown in illustration, does not appear to explain this fully. It appears to be an individual peculiarity.

compared with Mademoiselle Anita (height 25″), and Ludwig Ulpts (height 34″)—"the smallest man in the world": see (51) and (65).

(98). Wax model of Nicholas Ferry Bébé, in his clothes and with a wig of his own hair prepared by Jeanet, his surgeon, and preserved in the Musée Dupuytren, Paris. It probably gives him too dumpy an appearance, and it is possible that parts of the model as it now stands have sunk (note especially the state of the stockings in the photograph). Bébé was 18 when this model was taken, four years before his death. He was born in the Plain of the Vosges, 1741. When 4·5 years old he was said to be 61 cm. long, 72·5 cm. when about 18, and 89 cm. at his death; in the last few years of his life he developed very considerably and had no reserve of strength for this growth. His case is usually spoken of as a case of true dwarfism or ateleiosis, but it has been recently suggested that it was at least complicated by congenital syphilis. See Fig. 745, Bibl. Nos. 20, 33, etc. and *Iconography* (82), (83), and (121). Cf. Plate Z (39) and (40).

Plate WW. This plate illustrates further cases of ateleiotic dwarfism, partly from famous exhibition cases, and partly from reported medical instances. (117) and (120) reproduce four dwarfs famous for many years in the annals of showdom. Charles S. Stratton ("General Tom Thumb") was the son of Sherwood E. Stratton and was born at Bridgeport, Connecticut, Jan. 11, 1832, of parents mentally and physically perfectly normal; he is said to have weighed 9 lbs. 2 ozs. at birth. At about 5 months he weighed 15 lbs. and measured 25″, and Wood states that up to 1845 he did not increase in stature, and not in weight by more than two ounces. His parents had three other children of ordinary size[1]. When Barnum first saw him (in Nov. 1842) "he was a perfectly-formed bright-eyed little fellow, with light hair and ruddy cheeks, and he enjoyed the best of health. He was exceedingly bashful." In 1862 Stratton met in connection with Barnum "an extraordinary dwarf girl named Lavinia Warren, who was residing with her parents at Middleboro', Massachusetts," and whom Barnum at first exhibited with Commodore Nutt. The latter, according to Barnum, had fallen in love with her, but Tom Thumb, taking a holiday, came to see Barnum, and being introduced to her, succeeded in gaining her consent and married her on Feb. 10, 1863, at Grace Church, New York. In 1864 Tom Thumb and his wife came to England with Commodore Nutt and Mrs Stratton's sister, Minnie Warren. The marriage of Tom Thumb and Lavinia Warren resulted in the birth, on Dec. 5th, 1863, of a female child, Minnie Tom Thumb, who weighed at birth 3 lbs., but was a fine healthy child weighing 7¾ lbs. at a year old; she died from inflammation of the brain at the Norfolk Hotel, Norwich, Sept. 1866, while her parents were on tour in the Eastern Counties. According to Barnum, Tom Thumb erected a tall marble shaft surmounted by a life-size statue of himself in Mountain Grove Cemetery, Bridgeport. Commodore Nutt, otherwise George Washington Morrison Nutt, was the son of Major Rodnia and Maria D. Nutt; his father was a substantial farmer of Manchester, New Hampshire. He was born on April 2, 1844. He came in touch with Barnum in December, 1861, who made what he terms a "palpable hit" with him. Nutt was "a most remarkable dwarf who was a sharp, intelligent little fellow with a deal of drollery and wit. He had a splendid head, was perfectly formed and was very attractive; in short, for a 'showman' he was a perfect treasure." After his failure to win Lavinia Warren, Barnum tried to console him by suggesting that he should marry Minnie Warren. This marriage was several times reported, but never came off, and Nutt about 1876 married "a charming young girl, Miss Lilian Elston of Redow City, California, who tenderly cared for him till his death." There is no reference to any offspring of this marriage. Nutt when born weighed 10 lbs. 2 oz., and when aged 20 is said to have weighed 24 lbs. and to have been 29″ high[2]. His parents were mentally and physically normal and in comfortable circumstances. They had four other children. The first, a brother in California, weighed 165 lbs. when he left home aged 24; the third was also a "very large person." The second, aged 24 years in 1864, weighed only 65 lbs. and was 4′ 1″ in stature; he appears to have escorted the Commodore, who was apparently the fourth child. There are no details of the fifth child. It is said that there is nothing in the history of the Commodore or his family to account in any way for "his miniature features and frame." The appearance of a semi-dwarf brother must, however, be taken into consideration. Lavinia Warren was born Oct. 31, 1842, in Middleboro', Mass., of parents in comfortable circumstances. She is said to have had four brothers, one in Utah and two (in 1864) living with her parents (? the fourth), and three sisters, two of whom were at the same date married. All were of ordinary size except the youngest, Minnie, born June 3, 1846. In 1864 we have:

	Age	Weight	Height
Lavinia Warren	22	29 lbs.	32 in.
Minnie Warren	18	19 lbs.	24 in.

[1] Stratton had a normal sister Mrs Bassett, married and living in New York.

[2] Garnier says Nutt was 30″, when Barnum engaged him in 1860 and 43″ when he died in 1881: see Bibl. No. 205, p. 221.

Until Lavinia was a year old she was of the usual size; from that age she increased slowly in stature and ceased entirely to grow at 10 years of age. "She attended school regularly with the other children in the neighbourhood and found no difficulty whatever in keeping up with them in the classes which she attended." Minnie Warren is said to have been of ordinary size at birth, but to have grown very little afterwards. Both sisters are described as healthy and intelligent. After the death of Tom Thumb, Mrs Stratton married Primo Magri (see Fig. 690); she must, when in London in 1911, have been 69 years of age. The later history of her sister appears to be unrecorded. Some account of the family histories of these dwarfs should have appeared in our pedigree section, but we were unable to discover any record of them at the time except the meagre references in Wood (Bibl. No. 138, pp. 412, 418), Garnier (Bibl. No. 205, pp. 206, 221) and Barnum (Bibl. No. 240, pp. 71, 136, 213, 219, 224, 227). A second work of Barnum (*Struggles and Triumphs, or Sixty Years' Recollections of P. T. Barnum*, London, New York and Melbourne, 1889, pp. 88, 249, 255) repeats with but slight additions the facts stated in the *Life of P. T. Barnum*. The above account, while using these books, is chiefly drawn from an anonymous work: *Sketch of the Lives, Personal Appearance, Character and Manners of Charles S. Stratton, the Man in miniature known as General Tom Thumb, and his Wife Lavinia Warren Stratton, including the History of their Courtship and Marriage, Commodore Nutt and Miss Minnie Warren with some account of remarkable Dwarfs, Giants and other human Phenomena of ancient and modern Times*, London, 1865, Brickhill and Bateman. I am not aware that complete and accurate measurements as adults of any of these four famous ateleiotic dwarfs have ever been published. We have Quetelet's measurements of Tom Thumb taken when he was a boy (before 1850): see Fig. 735.

(118). Of "Prince Mignon," clearly a very interesting case of ateleiosis, we have so far no record beyond the photograph.

(119). The details of this remarkable family are given in our Fig. 731[b]. It is a case of ateleiosis showing heredity in three generations. The adult dwarf on the left is II. 2, aged 39, height 132 cm. The boy is III. 5, aged 12, height 95 cm., and the girl is a normal daughter aged 8. On the left is a man of normal stature. This is the only case where we have actually portraits from two generations of true dwarfs.

E. Illustrations of Sub-types of Dwarf Growth of Pathological Origin.

Plate JJ. The myxoedematous dwarf. We have already in Plate KK (76) seen a probable representative of this class in the show dwarf Wassilievitch.

(71[b]). Photograph of a water-colour drawing of a typical Dutch sample of a myxoedematous dwarf. The colours of the original convey the expression in a manner which can only be weakly reproduced by our photograph, but still our cut illustrates the type more forcibly than direct photography or woodcut can attain to. The reader should compare with (36). The ateleiotic dwarfs present a number of features common to infantile myxoedema, by which ateleiosis and myxoedema seem to merge one into the other, and it appears not impossible that they have relations in common. Absolutely certain differentiation is not in all cases possible: see pp. 365 and 367 above. We owe this illustration to the kindness of Dr W. Bulloch, who has placed the original in the Eugenics Laboratory.

(71[a]). Balthazar Zimmermann, a show dwarf described by Quatrefages. He measured 76 cm. when aged 16, and was thus comparable in stature at that age with Bébé. An account of Zimmermann will be found in Fig. 765, and Bibl. Nos. 187 and 232 may be consulted. He was probably myxoedematous but his physiognomy compares closely with that of a number of ateleiotic dwarfs.

(72). This cut contains four brothers, sons of the same parents, and our information concerning them is conveyed in a letter from Dr A. Marie: see Fig. 837. He considers that the three youngest represent characteristic myxoedematous dwarfism, the youngest with less of oedematous infiltration and having more of a cretinoid aspect. The eldest presents a case of simple infantilism. Whatever be the causes of dwarfism in this family, whether hereditary or toxic, we can hardly doubt the common origin in all four brothers, and thus the differentiation of these types as noted on pp. 365 and 367 becomes more obscure.

Plate KK. See above, Section D.

Plate LL. Types of Indian dwarfism contrasted. Major C. H. James, I.M.S., published in the *Indian Medical Gazette*, November, 1910, an account of three varieties of dwarfs, and most kindly sent excellent original photographs of these and other dwarfs to Karl Pearson.

(77) and (78) show anterior and posterior views of a normal native (*f*), of height 5′ 6·5″, alongside two achondroplasic dwarfs (*d*) and (*e*), a cretinous dwarf (*a*) and two further dwarfs (*b*) and (*c*), whom Major James classes as cases of infantilism, or "true arrests of general development." The following accounts are provided:

(*a*) The cretin Ralho is aged 30 years, Hindu. Her mother is an inmate of Patiala poor-house. Her father, two brothers and a sister, the latter three, at ages 20, 16 and 11 years, all died of the plague,

but were normal in every way. Her mother says she was normal at birth and ceased to grow at 16. There are no other dwarfs in the family and no history of goitre obtainable. She is an idiot and unable to talk, the breasts and external sexual organs are undeveloped. Her height is 2′ 11·5″ and weight 2 stone 11 lbs. No indication of a thyroid gland can be felt in the neck.

(*b*) Piyara Lal, aged 20 years, Hindu goldsmith. Quite intelligent and sharp-witted and quick at grasping new ideas. He has a falsetto voice, penis and scrotum small and undeveloped, no hair on face or pubes. His complexion (see photograph) is very fair for a native. He is active and runs fast. Height 3′ 3″. Weight 2 stone 11 lbs. The thyroid gland can be felt in the neck. He is said to have stopped growing at 10. It would be natural to class him as a case of ateleiosis, Group II. No family history of dwarfism.

(*c*) Sewa Singh, aged 28 years, Sikh. Member of a large family, four elder brothers, three elder sisters and seven younger sisters, but only two younger sisters have survived. All said to be normal, and the two surviving sisters are tall women. His height is 3′ 4·5″, and weight 2 stone 11 lbs. He has good health, is a good rider, roller skater, and plays many games. He has a slight moustache, which began to appear at 25 years, and a little hair on pubes, but his sexual organs are not fully developed. Quick, active and full of fun, sharp as a needle in repartee where banter and pleasantry are concerned; the voice is small and childish. Thyroid gland present and no deformity or bending of bones. It will be seen from the photograph that he is of darker complexion than (*b*) and that his physiognomy is far more adult. It will be obvious that this is a case where development has gone on to adolescence in some characters even if retarded. He possibly belongs to Gifford's ateleiotic Group III, although the sexual organs have remained infantile. The intelligence of both (*b*) and (*c*) seems above that of the average of European ateleiotic dwarfs.

(*d*) and (*e*) are typical cases of achondroplasia.

(*d*) Hamel Singh, aged 27 years, height 3′ 9″, weight 5 stone 8 lbs., has several normal brothers and sisters, and no other dwarfs occur in family. He is said to have grown till he was 12 years of age and then ceased. The micromelia is of rhizomelic type; the hands and feet are said to be those of a normal man. There is hair on face and pubes, the sexual organs are normal. He is quite intelligent, and reads and writes a little English as well as Gurkhali and Urdu. He is strong and active.

(*e*) Wazir Singh, aged 47 years, Sikh barber, height 4″, weight 6 stone 3 lbs., has no other known members of his family like himself. His growth continued, he states, until 22. His intelligence is normal; hair on all normal parts of his body, sexual organs fully developed. Limbs as shewn in photograph typically achondroplasic like those of (*d*). His hands are short and broad, and appear from (77) to be "en trident," so that in this matter he is more typical than (*d*). His feet are large.

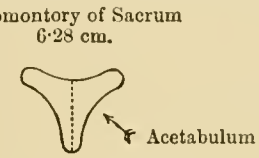
Plate MM (79) and (80). These photographs show a female achondroplasic dwarf, aged 48 years, Lachmi Narain, daughter of Harriji, a Brahman by caste, born at Bindiachal, in the United Provinces. She earns her livelihood as a faqir; has never married. Both parents are dead; she has no brothers, two sisters, who are both normal in size, and have normal children. Lachmi is 36″ in height; weight 3 st. 10 lbs.; chest measurement 26″; circumference of abdomen 24″; total length of spine from nape of neck to tip of coccyx 29″; height of head from vertex to mental point 8″; from shoulder to elbow, when forearm is bent, 8″; from hip to knee 9·5″; from hip to sole of foot 19″; circumference of thigh 13″; circumference at knee 9·5″; at ankle 5·75″: length of foot 6″; elbow to wrist 4·75″; breadth of palm 2·25″; length of index finger 1·5″; length of middle finger 1·62″; length of ring finger 1·62″; length of little finger 1·25″; wrist to knuckles 2″. The hand is thus typically achondroplasic, and from the photograph "en trident." She has all her teeth except third molars, which have never erupted; the breasts are small and not properly developed; vagina infantile and she has never menstruated. Intelligence good, she can read and write Hindi. She walks well, but slowly with a certain amount of waddle, and states that she soon gets tired. [From a letter of Major James to Karl Pearson.]

(81). A case of pituitary giant growth in a Cashmiri aged 23 years, height 7′ 9″. Beside him an English man of medium height. Two Indian dwarfs, probably achondroplasic, but age, race, height and nature of underlying condition are uncertain. The appearance of the bigger certainly suggests achondroplasia by the shortness of the limbs, shape of curvatures as far as these are seen; the smaller may possibly be of a rickety nature, but achondroplasia is not excluded. An ateleiotic dwarf from Patiala, aged 23 years, height 28″, is shown on extreme right. No full anthropometric or medical description with photograph.

Plates NN, OO, and PP give further types of dwarfism in marked contrast. We owe these excellent photographs to Professor Nijhoff of Groningen, and one of the chief reasons for publishing them here is to create if possible a strong public feeling against the legislative laxity which permits in modern states reproduction by such deformed persons. Not only are these cases in which the deformities have been or may be perpetuated—the women in (89) and (92) come of an achondroplasic stock and have had achondroplasic offspring—but in each parturition there is grave danger to the mother, and Caesarian

section has been necessary not once, but *repeatedly* in the case of the same woman. When medical science renders it possible for such mothers and their often deformed offspring to survive, is it not needful for the strong hand of the state to intervene,—since natural repugnance appears no longer to guide the instinct of he male,—and prevent the parenthood of deformity by its segregation before adolescence? The world little realises what racial harm is done, when the multiplication of the unfit is rendered possible by increased surgical skill, or by economic provision for the deformed unaccompanied by stringent segregation.

Plate NN (82)—(84). Rickety dwarf growth in brother and sister, associated with multiple and extreme curvatures of the long bones of both lower extremities on both sides due to bending of softened bone under body weight; and resultant other deformities. The curvatures affect the shafts of these bones



rather than the regions of junction of epiphyses and diaphyses and are actual curves rather than angles (contrast achondroplasia). In the female, M. B., the pelvis is markedly deformed; and the form of its inlet is tri-radiate; it was diagrammatically represented by Professor Nijhoff as shown. It gave the following measurements: inter-spinous 18 cm.; inter-cristal 17 cm.; inter-trochanteric 20·5 cm.; external conjugate 14 cm.; diagonal conjugate 7 cm.; true conjugate 6·25 cm. M. B., who is a single woman, aged 40, shows the scar of the operation of Caesarian section by which means she was delivered by Professor Nijhoff of a normal male child, which was, however, born dead. The condition of the male pelvis may very probably be much the same, but this point being of no obstetric importance is not usually investigated in such cases. In this male, in whom, if anything, the curvatures and deformities are of more extreme grade than in the female, there is considerable rickety deformity of the thorax as well, the sternum is curved forwards and prominent, the antero-posterior diameter of the thorax being increased ("pigeon-breast"); the lateral walls are somewhat depressed or "fallen in" (or, more accurately, "pulled" or "pushed" in by the action of diaphragmatic traction and atmospheric pressure during inspiration on unduly soft bone, "Harrison's sulcus." This latter feature is, however, here not very well marked.) The height of the female, whose age is 40 years, is 86 cm.; that of the male, whose age is uncertain, is 96 cm. They show no other peculiarities than the above. The male dwarf is described as a "merchant."

Plate OO (85)—(86). A. J., aged 35 years, rickety dwarf. Height 143 cm. Married and has had three children. The first was delivered by craniotomy in her own home. The second was by Caesarian section (Jan. 15th, 1907); the mother recovered, the child a male, normal, was born living. (It was 55 cm. in length and 4055 grammes in weight.) The third delivery was also by Caesarian section (March 7th, 1909); the mother recovered, the child, a male, normal, was born living. (It was 52 cm. in length and 3720 grammes in weight.) The pelvic measurements of this dwarf are as follows: inter-spinous 25 cm.; inter-cristal 26 cm.; inter-trochanteric 30 cm.; external conjugate 17 cm.; diagonal conjugate $8\frac{3}{4}$ cm. The proportions of this woman, apart from small stature and pelvic deformity, are about normal for height and afford a marked contrast to those of the achondroplasic women. She shows, however, a condition of genu valgum or "knock knee" which is too marked to be considered normal or physiological even for her sex. This deformity, that of the pelvis, and her small height are doubtless rickety in origin. She also shows marked varicosity of the internal saphenous vein, etc. on both sides ("varicose veins"), an independent condition, and the influence of the gravid uterus in producing this is shown clearly by the series of photographs.

(87)—(88). A. T., achondroplasic woman, aged 27 years. Height 111 cm. Single. Delivered of first child by Caesarian section by Professor Nijhoff, Jan. 17th, 1904. Mother recovered; child, male, normal, born alive. (Its weight was 2800 grammes and its length 51 cm.) Pelvic measurements of A. T.: Inter-spinous 22·5 cm.; inter-cristal 25·5 cm.; inter-trochanteric 27·5 cm.; external conjugate 18·5 cm.; diagonal conjugate 9 cm. All three of Professor Nijhoff's cases (*i.e.* (91), (89) and (87)) show the marked obesity typical of the condition in the female adult (as contrasted with muscularity in the male adult), they show the typical features of the achondroplasic condition, shortness and massiveness of limbs, prominent buttocks, characteristic curvatures of the long bones, short broad hands and feet with characteristic digits, etc. The head and face of (87) are, however, not characteristic (or typical) as they are in (91) and (89). (87) also shows a spinal curvature (scoliosis) of some extent with a primary curve, dorsal, convex to the right (as most commonly occurs in scoliosis); the secondary, compensatory curvatures to the left in the cervical and lumbar regions are only faintly shown. A similar condition of scoliosis, occurring, however, in an adult *male*, was shown by Parhon Shunda and Zalplachta's Roumanian case

described in Fig. 656. She also shows scars which appear suggestive of old ulceration (possibly tertiary syphilitic) on the anterior surface of both legs.

PLATE PP (89)—(90). Aaltje B., the elder of two achondroplasic sisters belonging to a stock with achondroplasia for at least three generations. Stature 122 cm., married, normal child by Caesarian section, which lived to 9 years of age.

(91)—(92) Jauna[1] B., aged 41, height 123 cm. Like her sister a typical achondroplasic woman. Married, Caesarian section, achondroplasic female child. These two cases show the condition typically for adult females. These features have, however, been fully enumerated in the text and do not require repetition here: see for the pedigree of this achondroplasic stock Fig. 664.

PLATE QQ. See below, Section G.

PLATE RR. See above, Section D.

PLATE SS. This plate illustrates special forms of achondroplasia and its associations.

(99)—(101). These cases give a family wherein one child, the girl (100), aged 17, is a typical achondroplasic dwarf; she is mentally normal. Her brother (99), aged 13, is an imbecile. Without having any other achondroplasic character, his hands appear to present something of the form "en trident." The third sibling represented (101) is a boy of age 16 years. He is mentally and physically normal, but Dr Hunter, to whom we owe the case, says that he has an unusual shortening of the lower limbs. He suffered from diabetes for seven years. The family appears to indicate that achondroplasia may be easily associated with other signs of degeneracy.

(102). Chipeta, an achondroplasic negro dwarf. He completes the proof of the widespread character of achondroplasia—Chinese, Hindus, Negros and Europeans are all affected. (The bird under the dwarf's arm might almost lead one to believe that the dwarf or his photographer had heard of Homer's legend (see p. 355 ftn.)!)

(103). Congenital humeral micromelia in a Bantu, Yao tribe, Nyasaland. Dr Hugh S. Stanuus, to whom we owe the photograph, states in a private communication that he has seen six similar cases, which form the material of a memoir by him in conjunction with Dr S. A. Kinnier Wilson, to appear shortly in the *Nouvelle Iconographie de la Salpêtrière*. The memoir discusses the relation of this condition to achondroplasia (Regnault in 1901 used the term "achondroplasie partielle" for a like state), and in three of Dr Stannus' cases there were some other features of achondroplasia. But until the publication of Dr Stannus' memoir all judgment must be suspended.

F. The Obese Dwarfs.

One may perhaps give this name to a group of dwarfs, which does not appear to have been fully examined and classified. Possibly their rarity accounts for this neglect. It is conceivable that they are related to the "myxoedème fruste" of Apert, who under this head (see Bibl. No. 469[b]) has figured an extremely obese, diabetic dwarf. With the exception of Carrie Akers all the dwarfs we have come across of this type in its most marked form are historic.

PLATE TT (104). The most noteworthy sample of it is perhaps the *Ragazza gigantesca*, of whom two portraits exist in Madrid. Both were painted by Carreno di Miranda. The clothed one is in El Prado, and represents a gigantic dwarf woman in a rich brocade dress. This is reproduced in (104). The second is in the Royal Palace, and represents the same female dwarf nude as Silenus. We have not been able to procure a photograph of this picture: see *Iconography* (64[a]) and (64[b]). The Comtesse d'Aulnoy thus describes this gigantic female dwarf whom she saw in 1679: "Une petite naine, grosse comme un tonneau et plus courte qu'un potiron, toute vetue de brocard or et argent, avec de longs cheveux qui lui descendaient presque aux pieds, entra et se vint mettre à genoux devant la Reine pour lui demander s'il lui plaisait de souper": see Bibl. No. 14[b]. The description accords well with Carreno's picture.

(105). Carrie Akers appears to have been of the same type. Her height was 34″, and her weight over 22 stone. Very few facts are unfortunately known with regard to her: see p. 361.

(106). The dwarf Barbino, who occurs in a number of bronzes, probably due to Valerio Cioli (see *Iconography* (118[a–c])), has a body which is also a mass of fat, if not quite so imposing as the *Ragazza gigantesca* or Carrie Akers. Meige looks upon him as a case of myxoedematous infantilism; but probably judgment must at present be suspended. Annibale Caracci in a satirical composition in the National Museum at Naples (see *Iconography* (38)—a photograph of the picture has recently reached us through the courtesy of the Director) has also introduced one of these obese dwarfs, in this case with bandy legs, who is clearly painted from life. We should be very grateful for further references to the type, which would probably find its way to the show booths at fairs.

[1] Erroneously given as G. B. on Plate PP itself.

G. The Dwarf in Art.

It has been impossible, owing to the great expense involved, to represent even in the merest selection the long list of dwarfs included in our *Iconography*. All that has been feasible is to indicate in one or two groups the artistic appreciation of dwarf types at various periods, this appreciation often making a true differentiation, which was not medically expressed till a much later date.

Plate QQ. (94) and (95) seem to suggest that myxoedematous dwarf forms were familiar to the Egyptians. (95) appears to be a dwarf form and Mace in his *El Amra*, Pl. L, 104 and 107, gives a front view, which shows well the curvature of the legs. (96) from the Ashmolean Museum, Oxford, is less certainly a dwarf type, but it indicates that these obese forms were not merely products of primitive and clumsy modelling, but represented something before the eyes of the artist. The much discussed Queen of Punt (93[a]) was certainly a true experience. Suggestions have been made that she was: (i) an achondroplasic dwarf, (ii) merely a steatopygous woman, and that (iii) the peculiarity was acquired. Against (i) several points may be made, for example, she is of the same height as other figures (this might be honour due to royalty or an oversight of an otherwise singularly particular draughtsman); her hands are normal and show no signs of the typical achondroplasic stumpy hand of trident form; the daughter in (93[b]) indicates features somewhat resembling those of her mother, so that the condition would suggest heredity, but parturition for an achondroplasic woman would have been difficult, if not impossible, at that date; the condition does not indicate any great disproportion of radius to humerus, etc. etc. Against (ii) Ruffer in a recent paper[1] has raised objections, the chief, I take it, being that in true steatopygia the buttocks stand out markedly behind the thighs. Ruffer also takes the view that the deformity was an acquired one. On all these counts, and especially in relation to (94)—(96), the question of myxoedema seems of some importance, and it is possible that the Egyptian jug forms may have been as much influenced by the experience of myxoedematous dwarfism as by that of achondroplasia. In that case their relation to the "obese dwarfs" would remain to be determined.

Plate UU. In this plate are collected a number of the life-like antique bronzes of dwarfs, probably largely the product of the Alexandrian school. The best of them are in Paris, either at the Musée de Louvre or at the Bibliothèque Nationale, but some few are in Germany and England. They indicate how well known were the various dwarf types to the ancient world, and how faithfully the artists reproduced their experience.

(107) represents somewhat emaciated negro pygmies, dwarfs of the ethnic type. They may be directly compared with Plate O (1). See *Iconography* (110).

(108) gives us a dwarf achondroplasic gladiator (cf. p. 358), in juxtaposition with a Chinese achondroplasic dwarf in the "cangue," a type of pillory. See *Iconography* (113) and (114).

(111) represents a further achondroplasic dwarf warrior. See *Iconography* (112[a]).

(112) is the figure of Aesop from the South Kensington Museum. He is represented as a rather obese dwarf, with marked lordosis and bandy legs, somewhat of the type of Carraci's dwarf (*Iconography*, No. 38). The bronze has been described as that of a rickety dwarf. See *Iconography* (115).

Finally in (109) and (110), we have life-like dwarf figures, full of action, presumably achondroplasic but less defined in type. See *Iconography* (112[b]).

Plate VV reproduces four of the famous pictures of dwarfs by Velasquez at Madrid.

(113) El Primo, (114) Sebastiano de Morra, (115) Antonio l'Inglese are all achondroplasic, and chiefly of interest as studies in achondroplasic physiognomy.

(116) gives Maria Barbola, an achondroplasic female dwarf, alongside Nicolasino Pertuseno, an ateleiotic male dwarf.

In the text we have referred to the long series of Spanish Court dwarfs. The fashion which collected and painted them, if idle at the time, served at least the scientific ends of a later generation whose dwarfs are not thus skilfully depicted by the great artists of their time for analysis and classification by posterity.

Plate WW. See above, Section D.

[1] *Bulletin de la Société Archéologique d'Alexandrie*, No. 13, 1911.

(1)

Normal or physiological (racial) Dwarfism: "Pigmies"—("Akkas": British Museum of Natural History). Photographed for this work by kind permission of Sir Benjamin Stone.

DWARFISM OF PATHOLOGICAL ORIGIN. ACHONDROPLASIA

(2)

Twins aged 15 months. One is achondroplasic, the other of average normal growth. Photographed for this work. (Dr Robert Hutchison's Case.)

(3)

An achondroplasic girl aged 7 years and her normal sister, aged 5 years, of average height for age. Photographed for this work. (Dr Robert Hutchison's Case.)

(4)

The same achondroplasic girl, aged 7 years. Photographed for this work. (Dr Robert Hutchison's Case.)

(5)

Achondroplasic and normal hands contrasted. They are those of children shown in (3). Photographed for this work. (Dr Robert Hutchison's Case.)

(6)

Achondroplasic male adult, aged 28 years.

(7)

The same adult, profile.

(8)

The same adult, front view.

(6), (7) and (8) were photographed for this work.

(9)

Achondroplasic mother and daughter. Boeckh's Case: see Pedigrees.

(10)

An achondroplasic female, aged 27 years.

The photographs of (9) and (10) are reproduced by kind permission of M. Nicol Gerson, Proprietor of "Tiny Town,"

DWARFISM OF PATHOLOGICAL ORIGIN. ACHONDROPLASIA

(11)

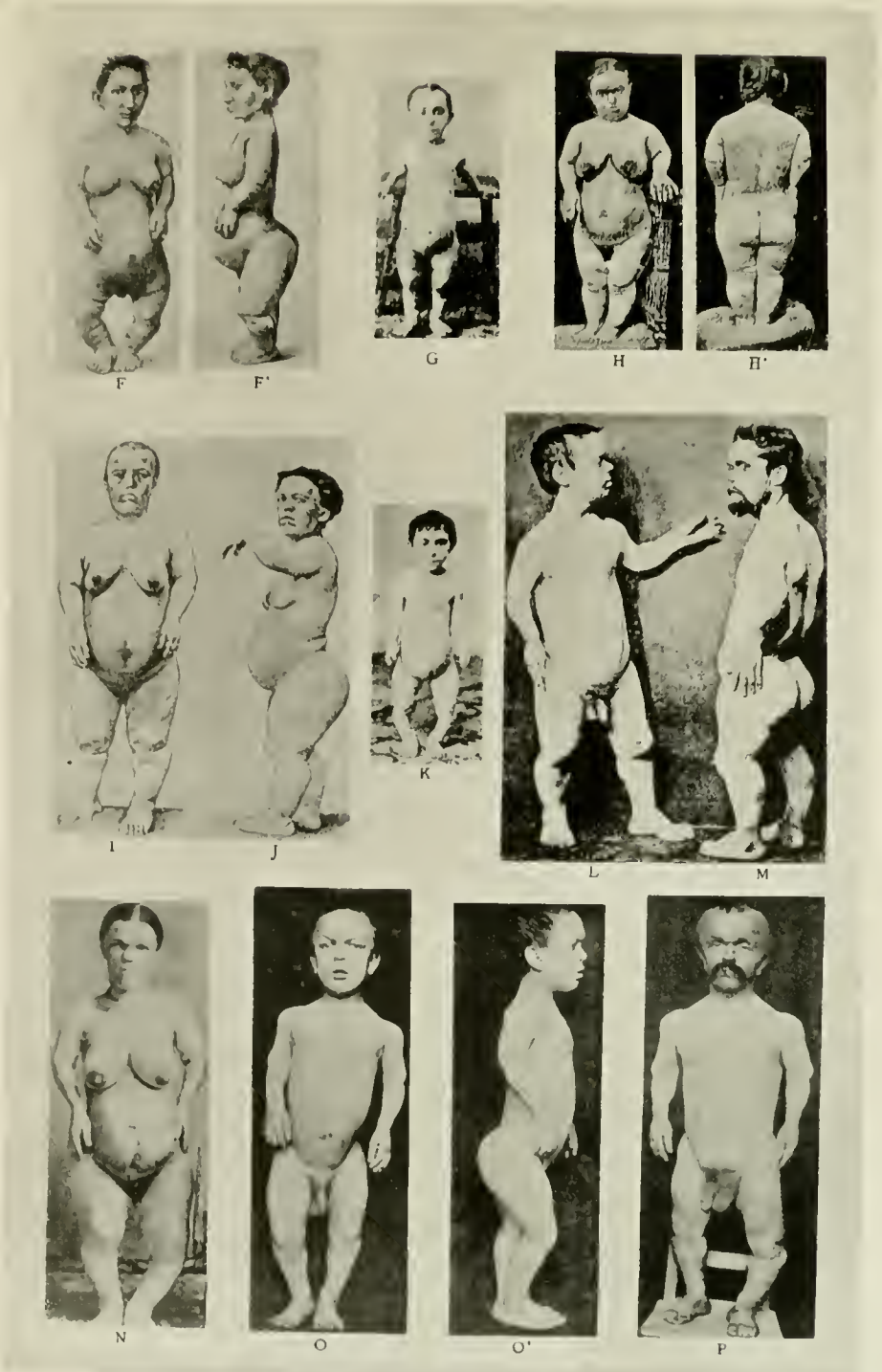
Achondroplasia in a Chinaman, aged 58 years. (Case of Dr Gordon Moir, R.N.)
Photographs (11)—(13) kindly provided by the Editor of the *British Medical Journal*.

(12)

Posterior view of same Chinaman.

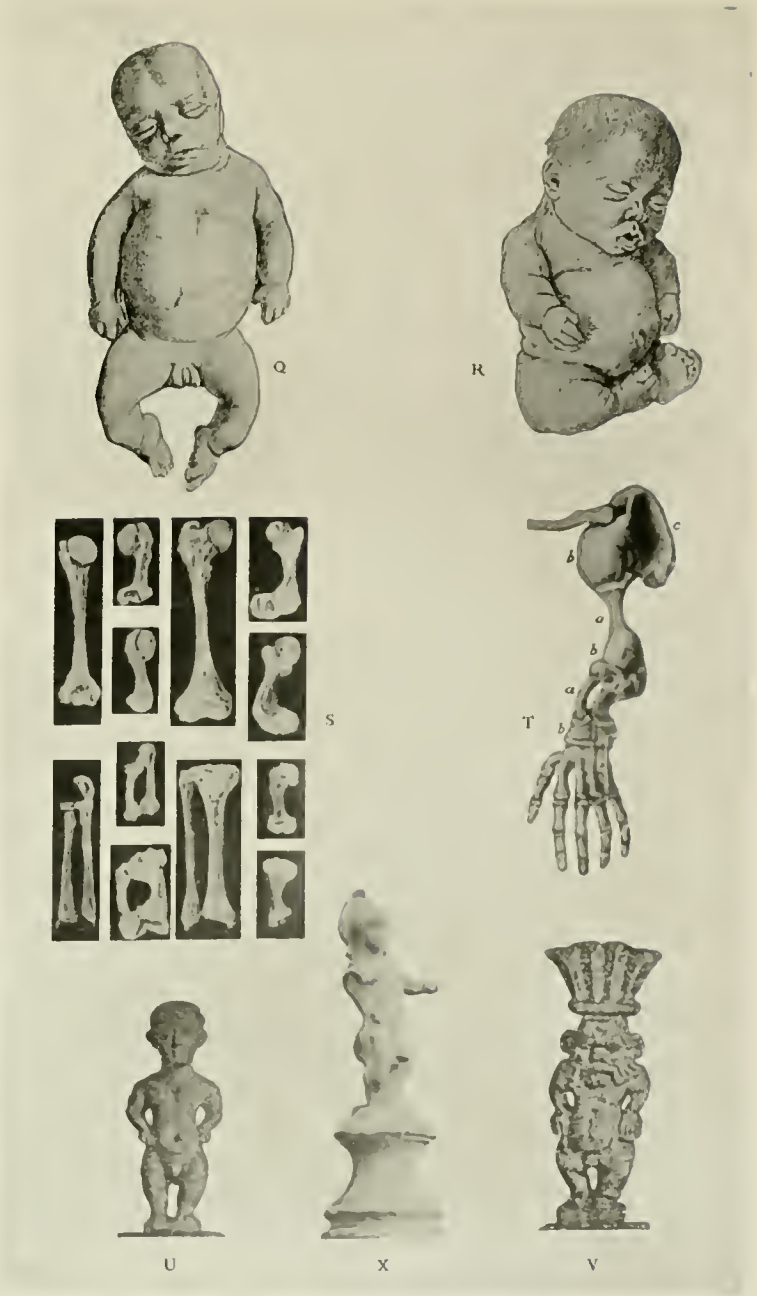
(13)

Anterior view of same Chinaman.



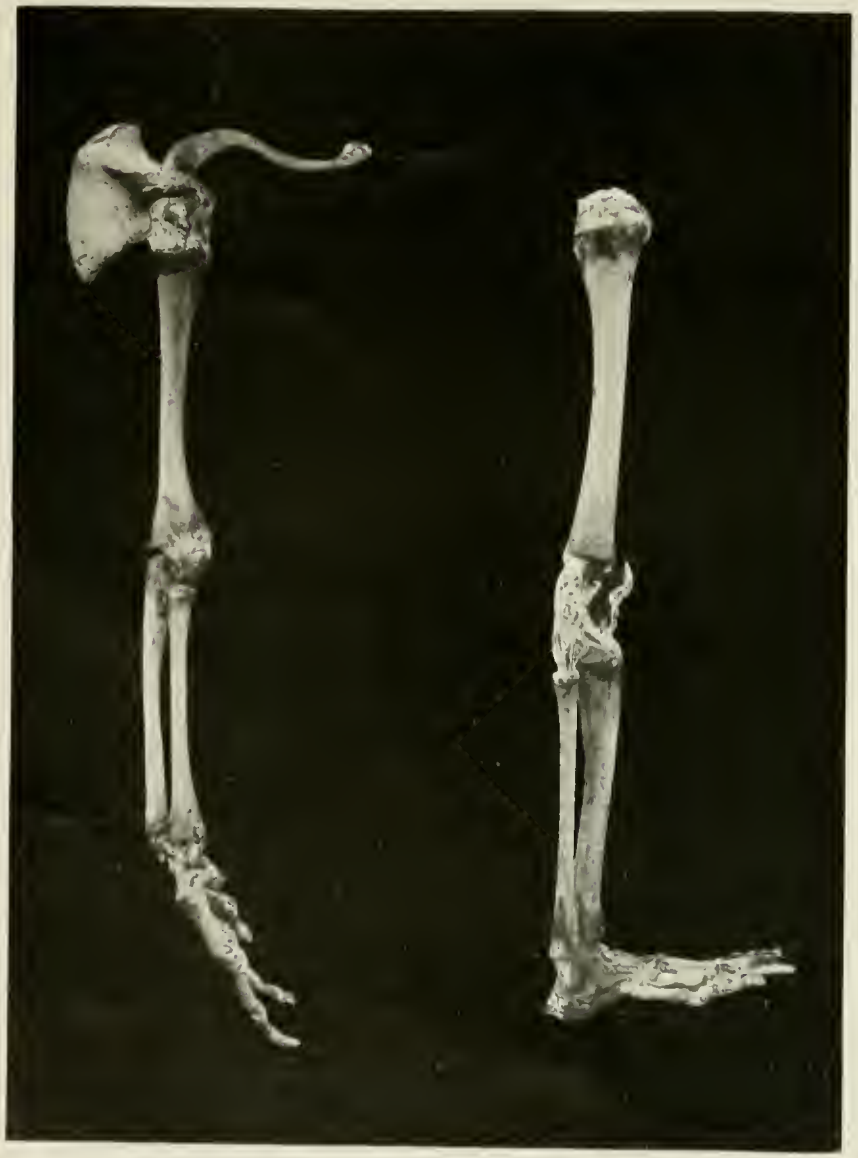
Achondroplasic Types. By kind permission of the Proprietors of the *Nouvelle Iconographie de la Salpêtrière*, Paris.

(14)



Q & *R*, achondroplasic child at birth. *S* & *T*, achondroplasic bones. *U* & *V*, statuettes of the Egyptian gods Ptah-Sokar and Bes, and *X*, statuette of the Roman Emperor Caracella in caricature. These show achondroplasic proportions. By kind permission of the Proprietors of the *Nouvelle Iconographie de la Salpêtrière*, Paris.

(15)



(16)

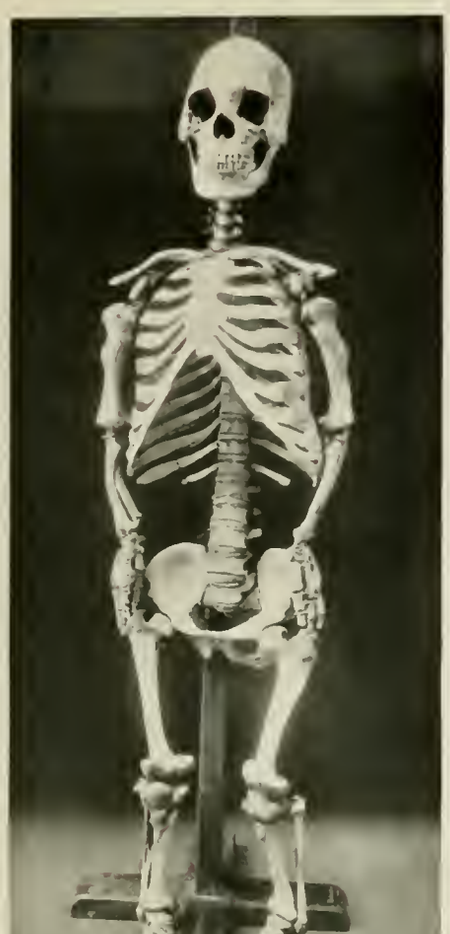
Normal.

(17)

Achondroplasic.

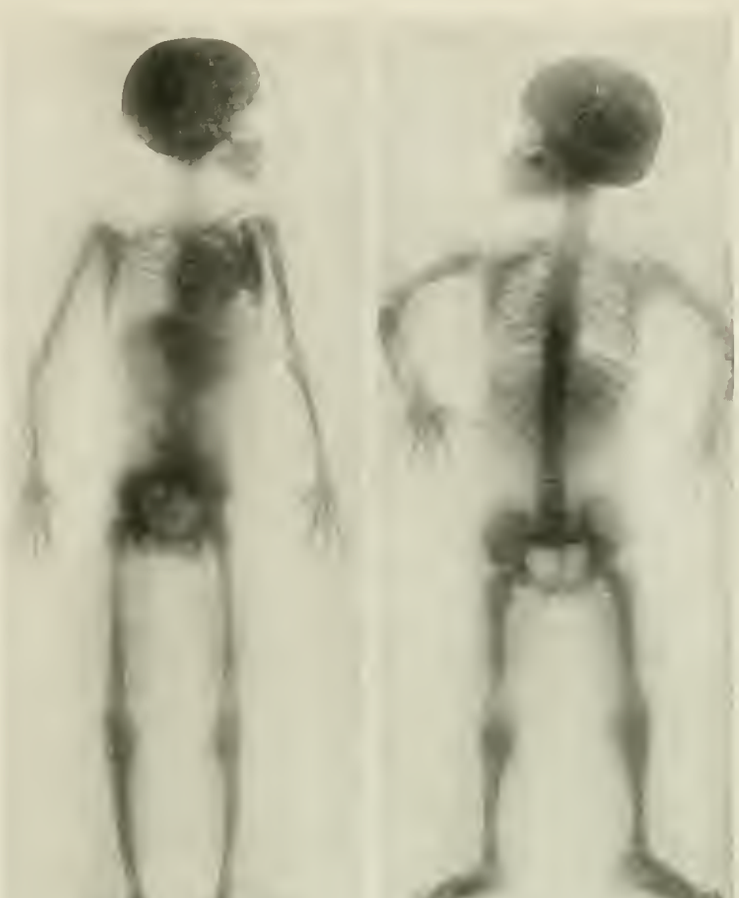
Comparison of the bones of the extremities at birth, normal and achondroplasic. From the Museum of the Royal College of Surgeons. (The first is a dry, the second a wet preparation. Otherwise approximately to scale, $\frac{2}{3}$ natural size.)

DWARFISM OF PATHOLOGICAL ORIGIN. ACHONDROPLASIA



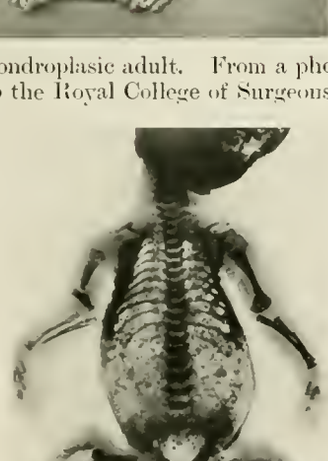
(18)

Skeleton of an achondroplasic adult. From a photograph belonging to the Royal College of Surgeons.



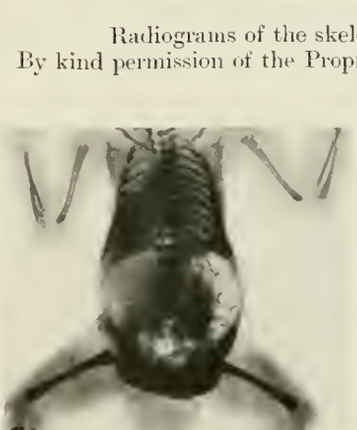
(19)

Radiograms of the skeletons of achondroplasic and normal girls of 9 years.
By kind permission of the Proprietors of the *Nouvelle Iconographie de la Salpêtrière*, Paris.



(20)

Pseudo-achondroplasic, rickety foetus.



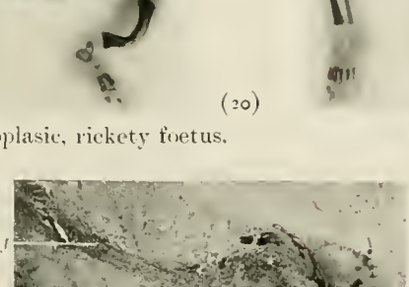
(21)

Normal foetus.

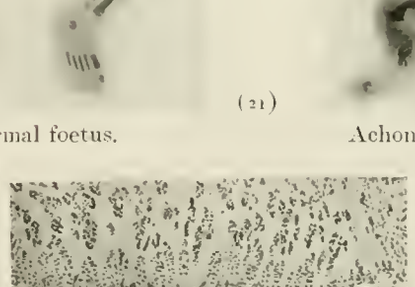
(22)

Achondroplasic foetus.

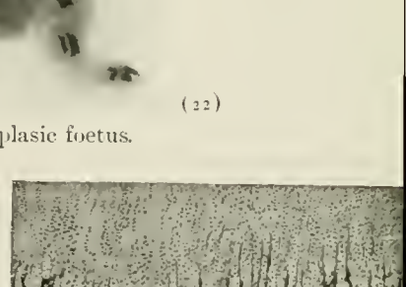
(23)



(24)



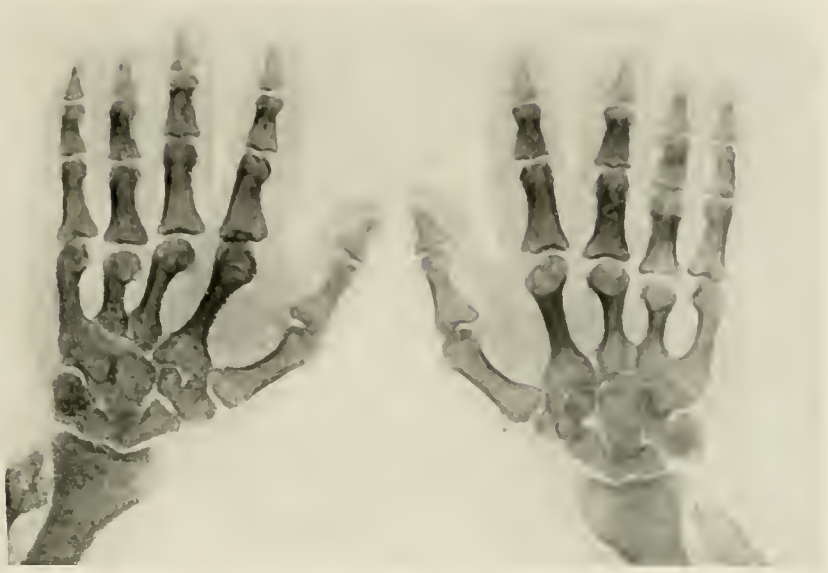
(25)



(26)

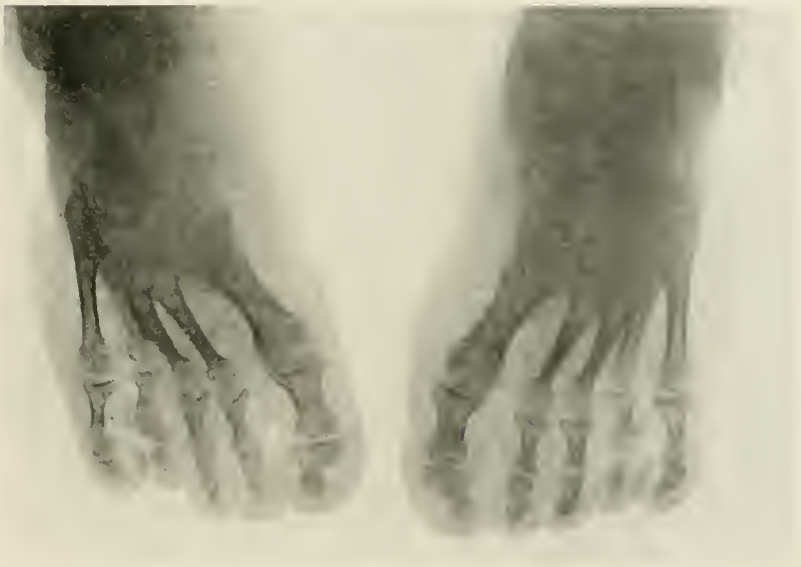
nal chondral ossification. Achondroplasia. Longitudinal section of upper epiphysis of femur. Congenital Rickets (pseudo-achondroplasia). Periosteal Dysplasia.

(20)—(26) are reproduced by kind permission of the Proprietors of the *Nouvelle Iconographie de la Salpêtrière*, Paris.



(27)

Radiograms of hands in the achondroplasic adult.

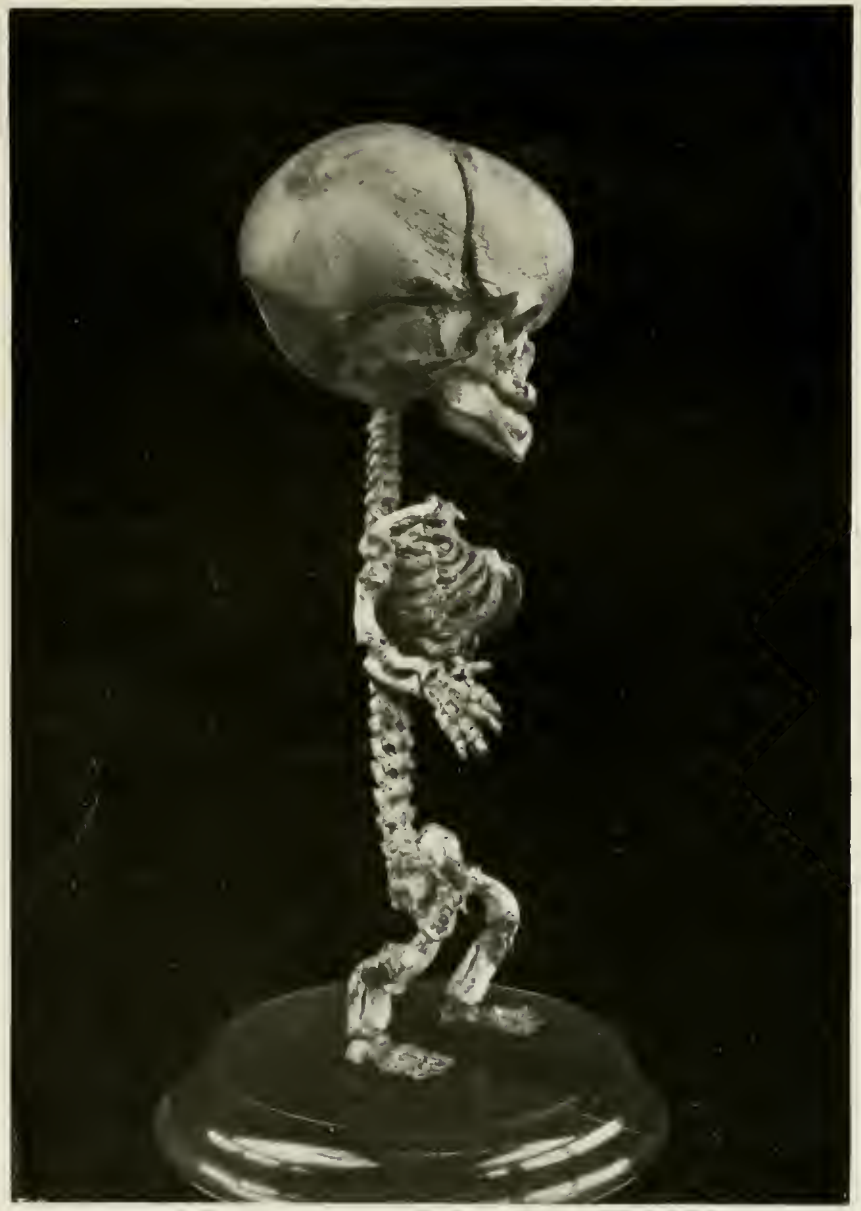


(28)

Radiograms of feet in the achondroplasic adult.

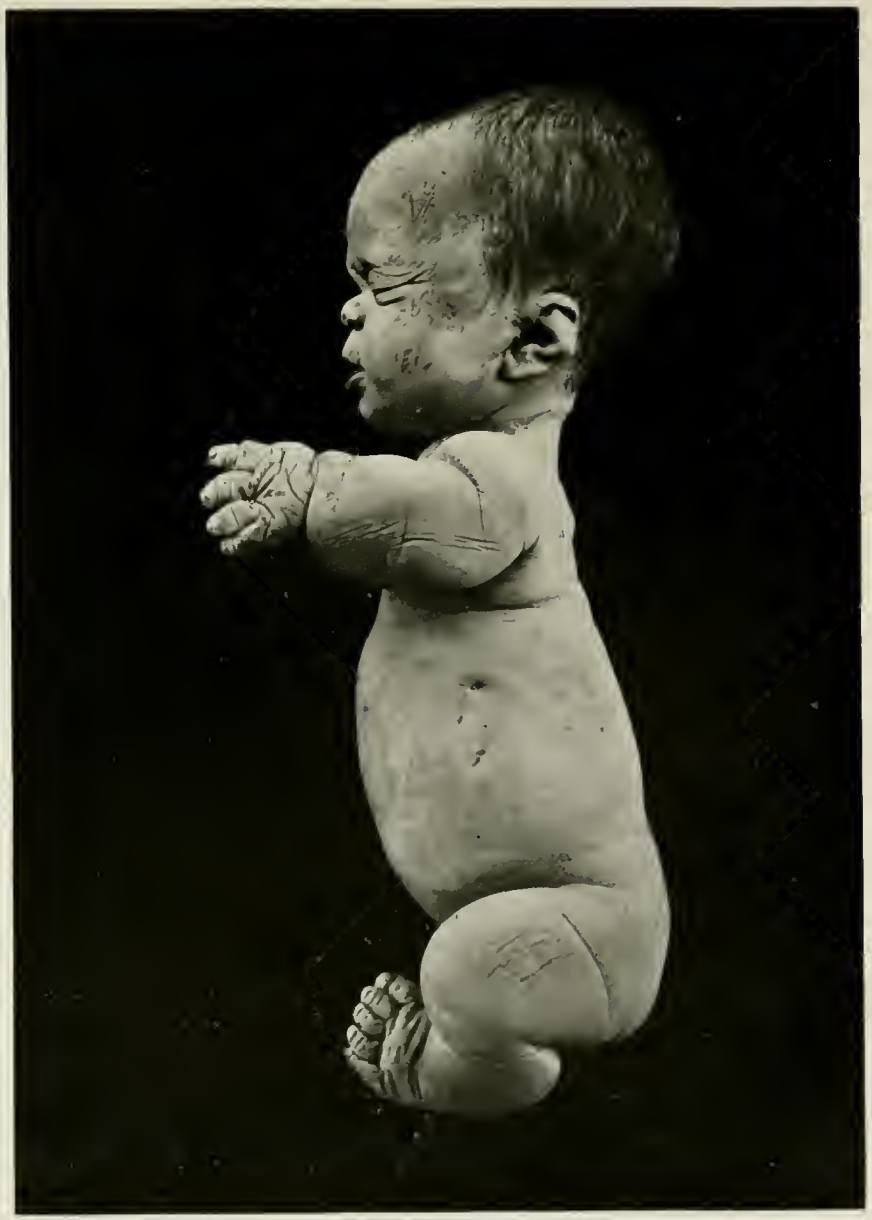
(29)

Radiogram of cranium in the achondroplasic adult.
By kind permission of the Proprietors of the *Nouvelle Iconographie de la Salpêtrière*, Paris.



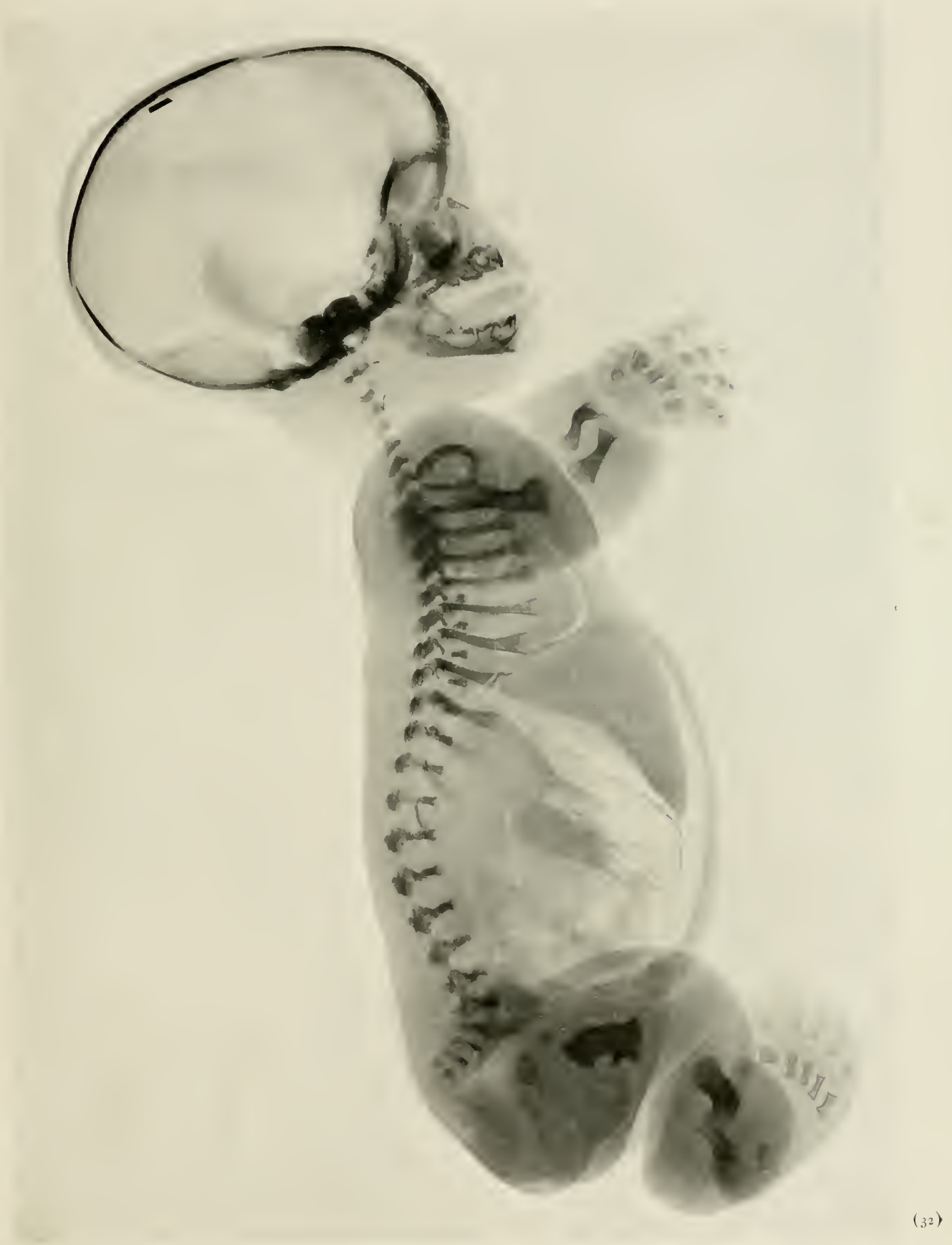
(30)

Skeleton of achondroplasic foetus at full term.
Museum of Royal College of Surgeons.



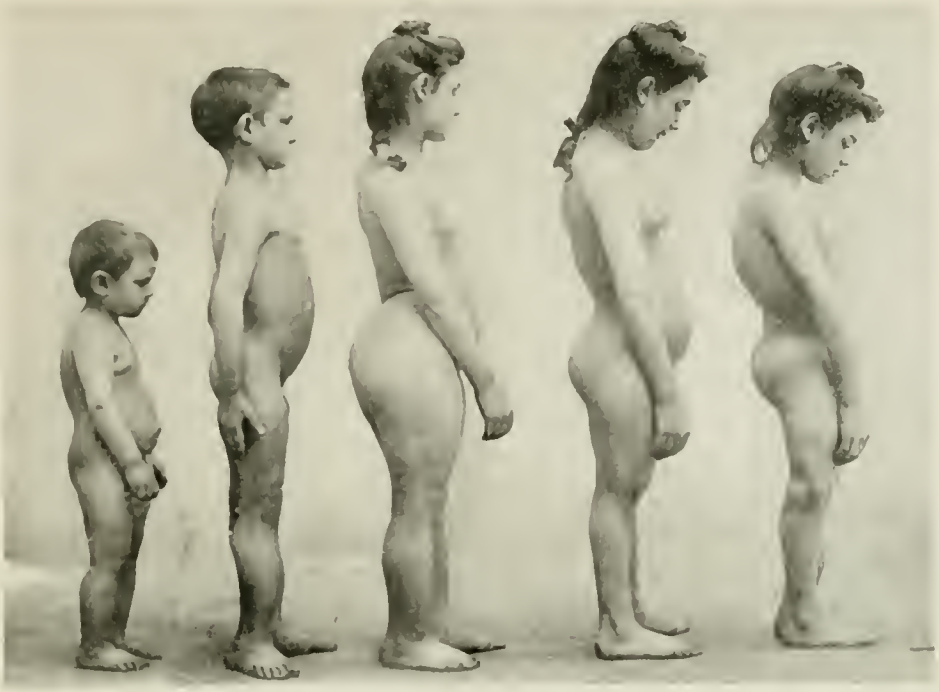
(31)

Achondroplasic foetus at full term (of female sex, as is the rule).
Museum of Royal College of Surgeons.



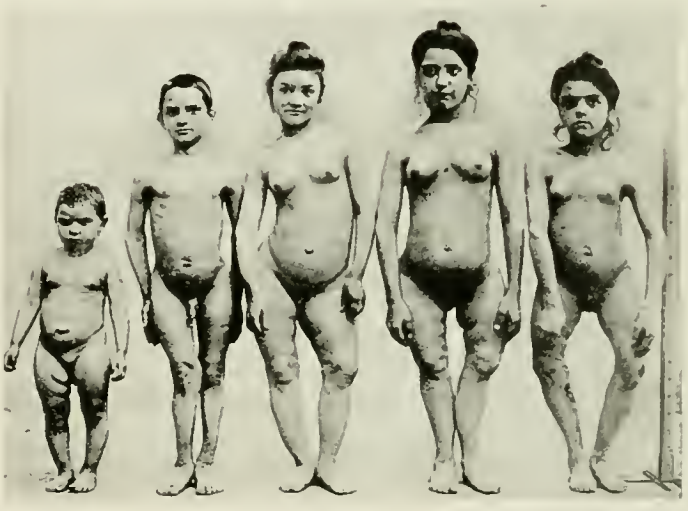
(32)

Radiogram of achondroplasic foetus at full term. From a specimen in the Museum of the Royal College of Surgeons. By kind permission of Professor Arthur Keith.



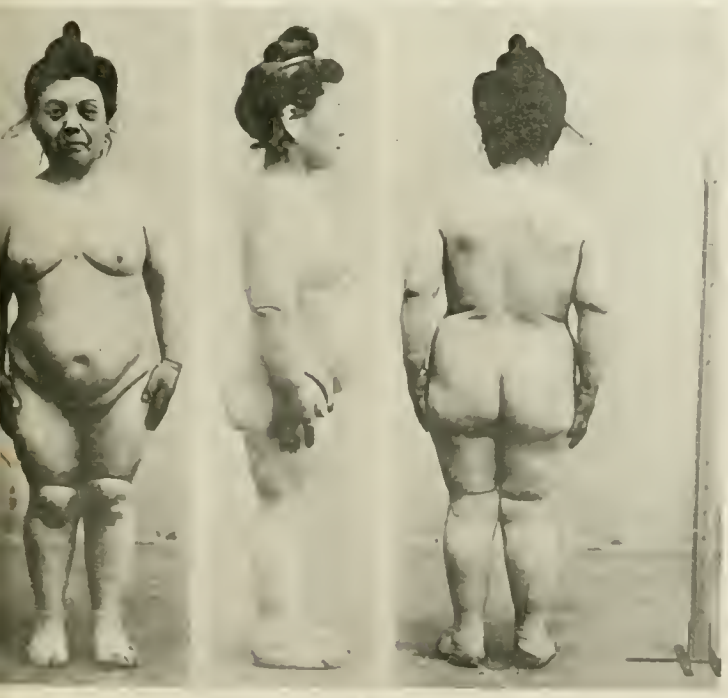
(33)

(34)

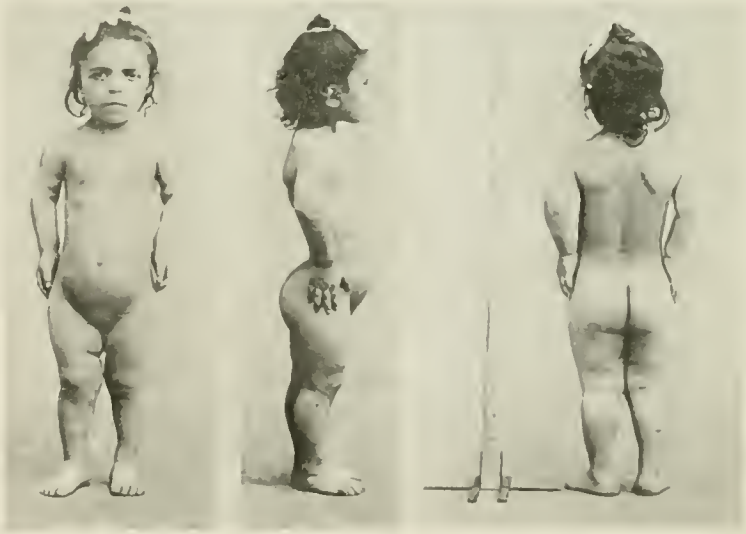


(35)

(33)—(35). The deformity and dwarf growth of rickets shown in a case of family rickets. Bizarre curvatures and deformities of bones due to bending of softened bone under weight. Pseudo—not true micromelia. Contrast (6)—(8), (11)—(13) and (14).



(36)

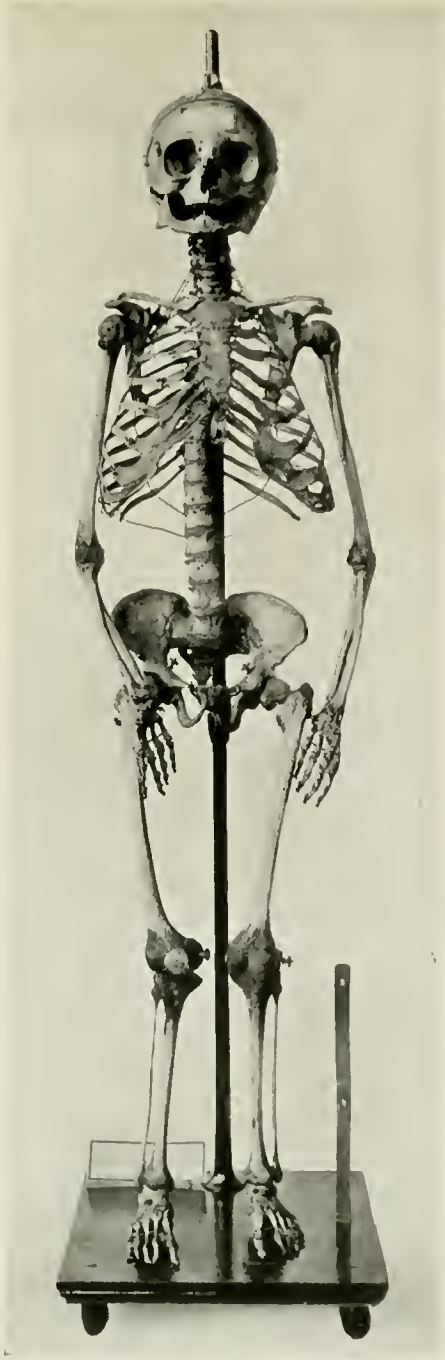


(37)

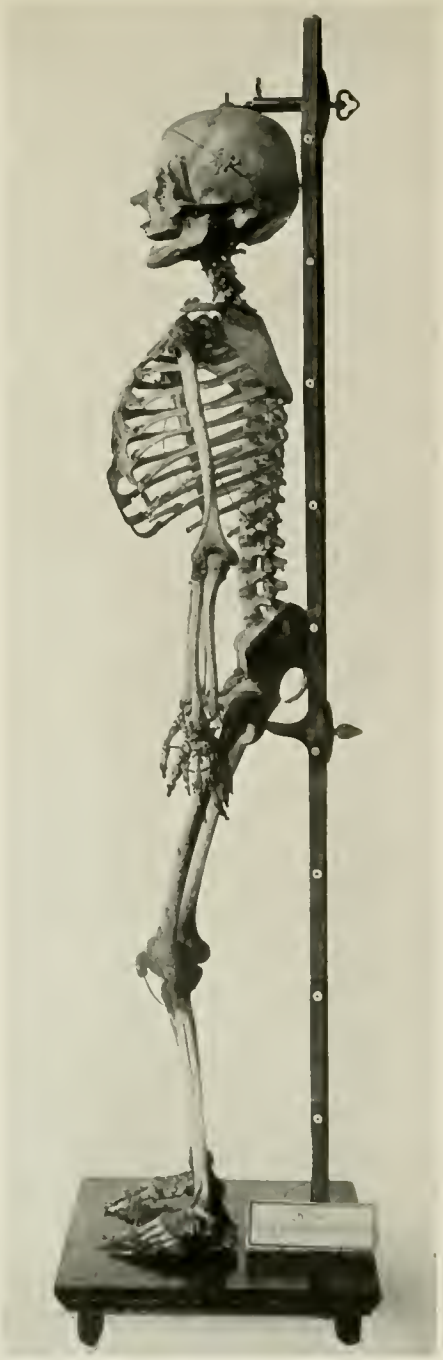
The dwarf growth and proportions of achondroplasia (37) contrasted with those of infantile myxoedema (36).
(33)—(37) are reproduced by kind permission of the Proprietors of the *Nouvelle Iconographie de la Salpêtrière*, Paris.

(38)

Skeleton of a child of 9 years (ateleiosis), height 19½″, with a skeleton of a normal child of 16 months, height 23½″. Photographed for this work from the originals in the Royal College of Surgeons.



(39)



(40)

Skeleton of Bébé, Nicholas Ferry, Dwarf of King Stanislas of Poland. Ateleiosis in a male, who died aged 20 years. Height 92·5 cm. (36·4″). From photographs taken for this work from the original in the Muséum d'Histoire naturelle, Paris, by the kind aid of Professor Dr Verneau

DWARF GROWTH OF PATHOLOGICAL ORIGIN. ATELEIOSIS. GROUP II

(41)

Ateleiotic male, aged 28 years, height 42″, with a normal boy aged 6 years.

(42)

Ateleiotic female aged 18 years, height 23½″.

(43)

Twenty cases of ateleiosis (Group II).

Photographs (41)—(43) are reproduced by kind permission of Mr Hastings Gilford and the Proprietors of the *Transactions of the Medico-Chirurgical Society*, London.

DWARFISM OF PATHOLOGICAL ORIGIN. ATELEIOSIS. GROUP H

(44)

A woman aged 70 years and her ateleiotic daughter aged 26 years, height (in shoes) 45″.

(45)

Three brothers, two ateleiotic, aged 24 and 22 years, height of each of these 40″ (without shoes).

(46)

Ateleiotic female, aged 28 years, height (in shoes) 43·2″.

(47)

Three ateleiotic siblings. Male, aged 30 years, height (in shoes) 43·2″; females aged 26 and 14 years respectively, heights 37·2″ and 34·4″ respectively.

The Tyrolese dwarfs (44)—(47) are reproduced for this work from photographs most kindly sent to K. Pearson by Dr Schmolck. Cf. *Virchows Archiv*, Bd. 187, S. 105, 1906.

(48)

Ateleiotic male, aged 62 years, height in shoes 45″.
Photographed for this work.

(49)

Ateleiotic male, aged 22 years, height in shoes, about 39″.

(50)

Two ateleiotic brothers, aged respectively 24 and 20 years;
of approximate height 36″.

(49) and (50) are reproduced by kind permission of M. Nicol Gerson,
Proprietor of "Tiny Town," Olympia, London, 1909—10.

(51)

Ludwig Ulpts, a German. Ateleiotic male, aged 18 years.
Height in shoes 34″ approximately.

(52)

Otto, a German. Ateleiotic male, aged 21 years.
Height in shoes 36″ approximately.

(53)

Forgéres, a Frenchman. Ateleiotic male, aged 35 years.
Height in shoes 45″ approximately.

(54)

Smaun Sing Hpoo, a Burman. Ateleiotic male, aged 26 years.
Height in shoes 37″ approximately.

(51)—(54) are reproduced from photographs kindly lent by M. Nicol Gerson, Proprietor of "Tiny Town," Olympia, London, 1909—10.

DWARF GROWTH OF PATHOLOGICAL ORIGIN. ATELEIOSIS. GROUPS II AND III

(55)

Four dwarfs belonging to Group II. Females aged 30 and 26, males 28 and 23. Reproduced by kind permission from a photograph by Messrs Porter Brothers of Hampstead.

(56)

Ateleiotic male, aged 36 years. Height (in shoes) 56″ approximately.

(57)

Ateleiotic male, aged 28 years. Height 57·5″; with normal brother aged 13 years.

(58)

Ateleiotic male, aged 52 years. Height 56″ (in shoes) approximately.

(59)

57) is reproduced by kind permission of Mr Hastings Gilford and the Proprietors of the *Transactions of the Medico-Chirurgical Society*, London.

(56), (58) and (59) are reproduced by kind permission of M. Nicol Gerson, Proprietor of "Tiny Town," Olympia, London, 1909—10. (56) to (59) belong to Group III.

Cases of Groups II and III compared, the smaller figures are those of (50) and (52), the larger those of (56) and (58).

DWARF GROWTH OF PATHOLOGICAL ORIGIN. ACHONDROPLASIA AND ATELEIOSIS

(60)

Large group of ateleiotic cases and four cases of achondroplasia. The latter may be recognised by their large heads and adult faces. The man in the background is 76″ in height.

(61)

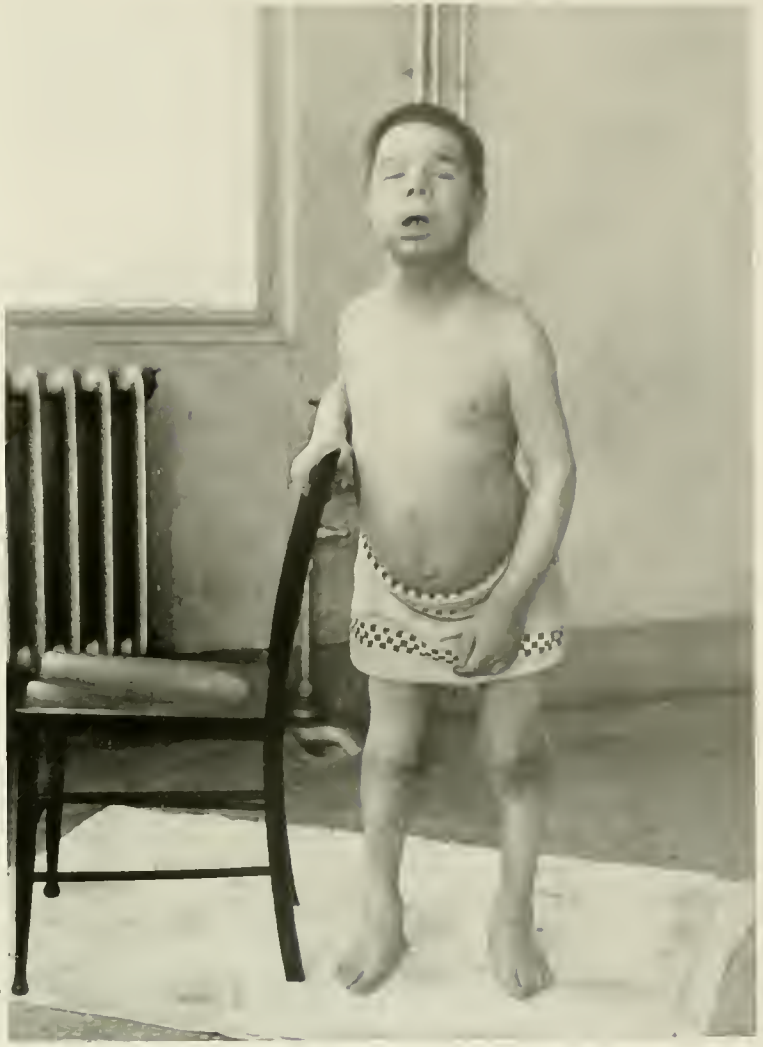
Ateleiosis in the equine species. The two small human dwarfs are figured in (50) and (52). The larger human dwarf belongs to Group III. The man in the background is 74″ in height.

(62)

Ateleiosis in father and son, the latter is shown in (51) and (61). The mother is achondroplasic.

(60) to (62) are reproduced by kind permission of M. Nicol Gerson, Proprietor of "Tiny Town," Olympia, London, 1909—10.

(63)



(64)

The dwarfing of cretinism for contrast with the achondroplasic and ateleiotic cases (particularly the latter) figured in the preceding plates. Note in the female the goitrous enlargement of the thyroid gland. From photographs most kindly provided by Professor G. R. Murray.

(65)

Mademoiselle Anita.
Hungarian Dwarf. From a photograph, by kind permission of Messrs Mendelssohn.

(66)

Jeffrey Hudson.
Queen Henrietta Maria's Dwarf. From a photograph of the picture in the National Portrait Gallery, by kind permission of the London Electrotype Agency.

(67)

(68)

The Boruwlaski Statue by Bonomi in the Museum, Durham. The youth standing beside it is 5 ft. 10 ins. in height. See Iconography No. 122.

(69)

Nannette Stocker and Johann Hauptmann. Reproduced from Kirby's *Wonderful Museum*. See Iconography No. 134.

(70)

Thérèse Souvray, "Madame Bébé." See Iconography No. 141.

(71[a])

Balthazar Zimmerman. According to Regnault he was a myxoedematous Dwarf. Reproduced by kind permission of the Proprietors of *La Nature* (T. XXIX. p. 180): see Bibl. Nos. 232 and 411.

(71[b])

Dutch myxoedematous Dwarf from a water-colour in the possession of W. Bulloch, M.D.

(72)

Four myxoedematous Dwarfs, children of the same parents. From a block most kindly provided by M. le Dr A. Marie: See Pedigree 837.

(73)

George Romondo, Jewish rickety Dwarf and Eccentric Mimic. Reproduced from Kirby's *Wonderful Museum*. See Iconography No. 156[b].

(74)

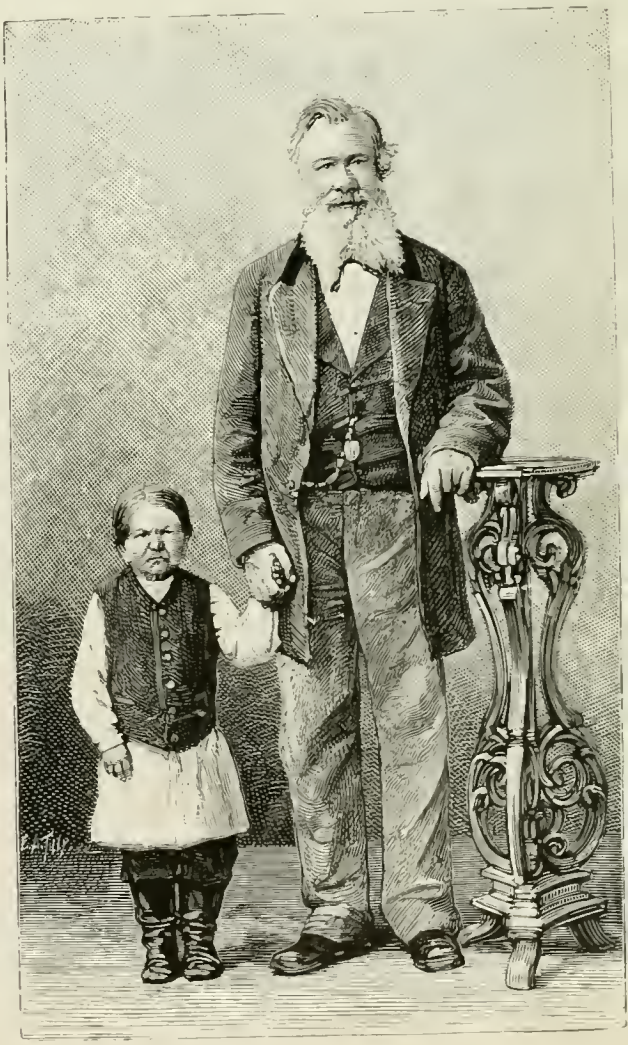
Owen Farrel, the achondroplasic Irish Dwarf. Reproduced from Kirby's *Wonderful Museum*. See Iconography Nos. 155 & 158.

Ages	$16\frac{6}{12}$	14	$11\frac{10}{12}$	9	$4\frac{1}{12}$	$6\frac{10}{12}$	
Statures	97·1	102	95·5	92	100	116	cm.

(75)

The first four ceased to grow in their 4th year.

The family Kostesky. Russian ateleiotic Dwarfs. Reproduced by kind permission of the Proprietors of *La Nature* (T. XXIX. p. 181). See Bibl. No. 232 and Pedigree No. 744.



(76)

Wassilievitch, a Russian myxoedematous Dwarf, aged 51. Reproduced by kind permission of the Proprietors of *La Nature* (T. XXII p. 13). See Bibl. No. 198.

(From photographs most kindly sent by Major C. H. James, I.M.S.)

(77)

a *b* *c* *d* *e* *f*

(*a*) Cretinous Dwarf, (*b*) and (*c*) Ateleiotic Dwarfs, (*d*) and (*e*) Achondroplasic Dwarfs, (*f*) Normal Native.

(78)

a *b* *c* *d* *e* *f*

Back view of the same Figures. Cf. *The Indian Medical Gazette*, Vol. XLV., November, 1910.

(79)

(80)

Major C. H. James' Case. Lachmi Narain, Achondroplasic ♀ Dwarf, aged 48 years, 36 inches. European 75 inches, Native Faqir, 67 inches in height.

(81)

Cashmere Giant (93 inches), normal European, two achondroplasic Dwarfs, and the Patiala midget (ateleiotic, 28 inches). From stereograph, copyright, Messrs Underwood and Underwood, London and New York.

DWARF GROWTH OF PATHOLOGICAL ORIGIN. DWARF GROWTH OF RICKETS

(82)

M.B. Aged 40. Stature 86 cms. Sectio caesarea.

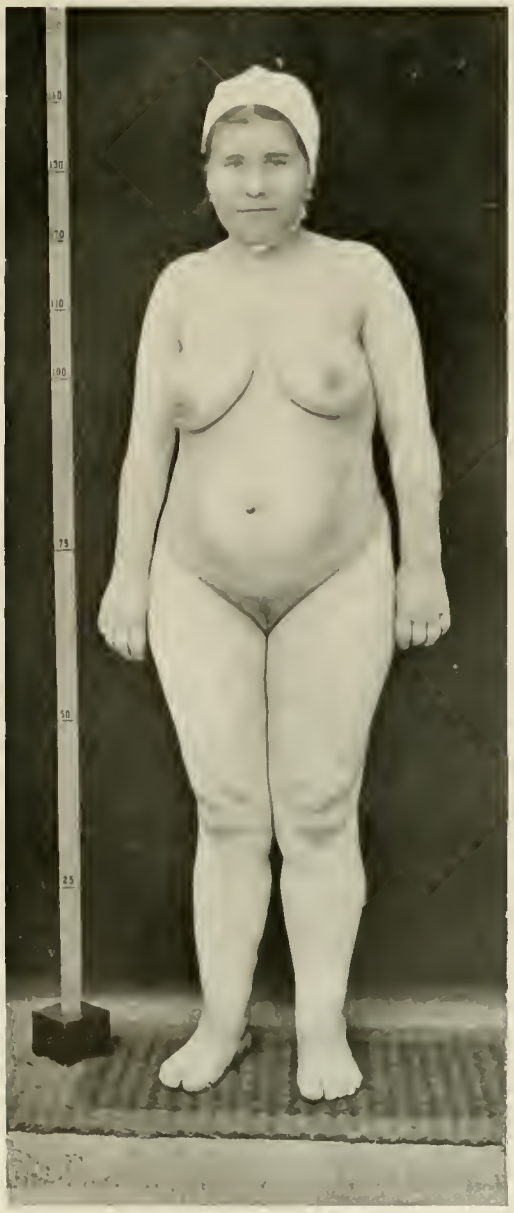
(83)

(84)

Brother to M.B. Stature 96 cms.

The Dwarf Growth of Rickets, associated with multiple rickety curvatures of the long bones of the lower extremities, occurring in sister and brother.
By the kindness of Professor Nijhoff of Groningen.

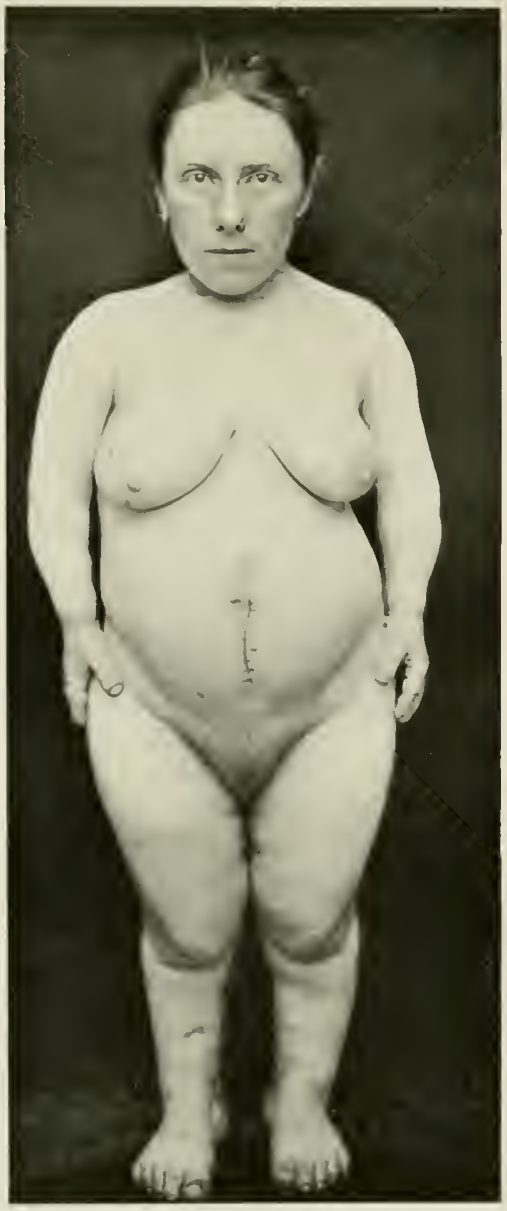
DWARF GROWTH OF PATHOLOGICAL ORIGIN. DWARF GROWTHS OF RICKETS AND ACHONDROPLASIA CONTRASTED



(85)

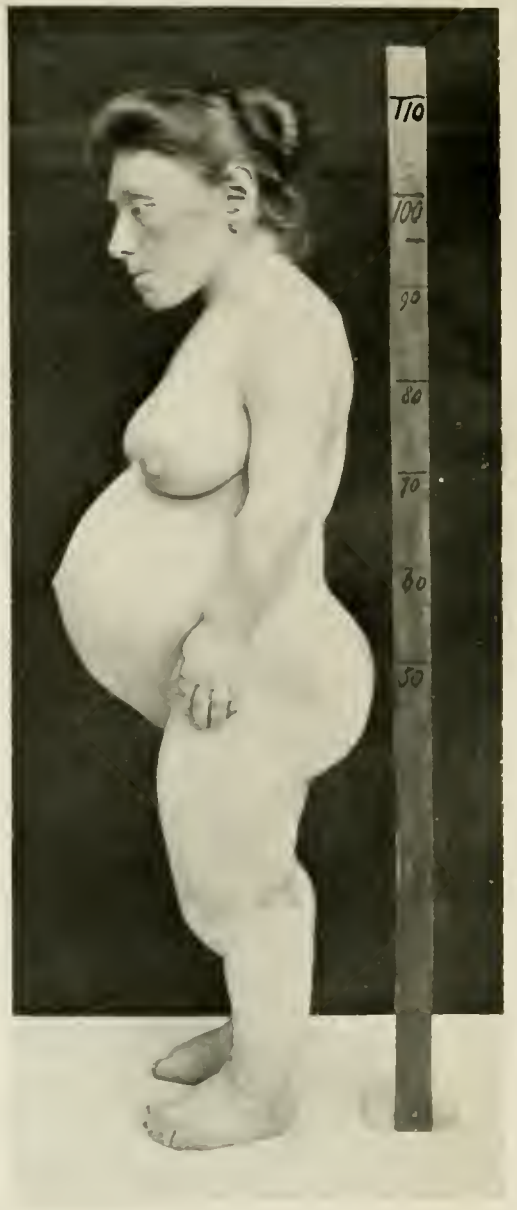
A. J. Rachitic Dwarf. Stature 143 cms. Aged 35. 1st Parturition, craniotomy; 2nd and 3rd, sectio caesarea.

(86)



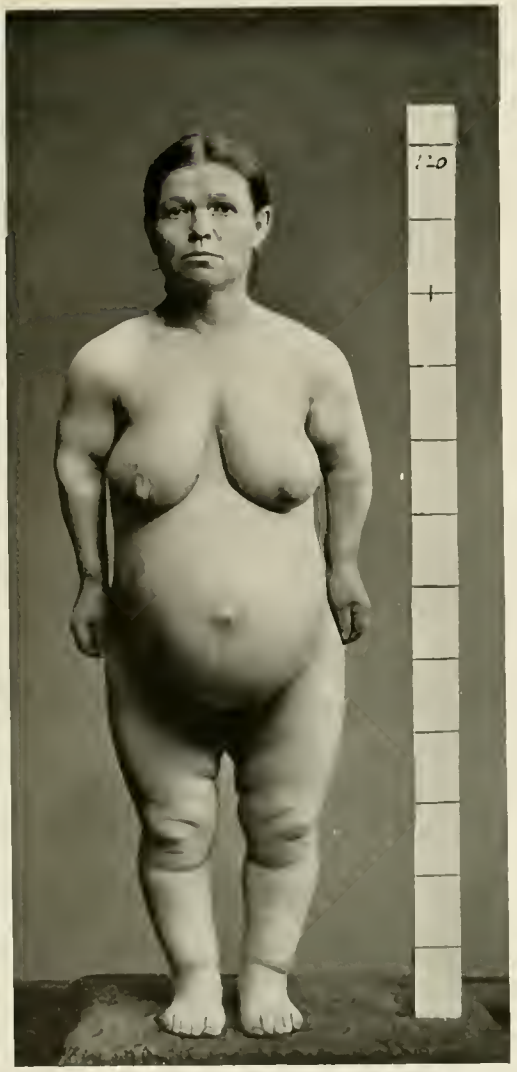
(87)

A. T. Achondroplasic Dwarf. Aged 27. Stature 111 cms. Sectio caesarea. normal ♂ child.

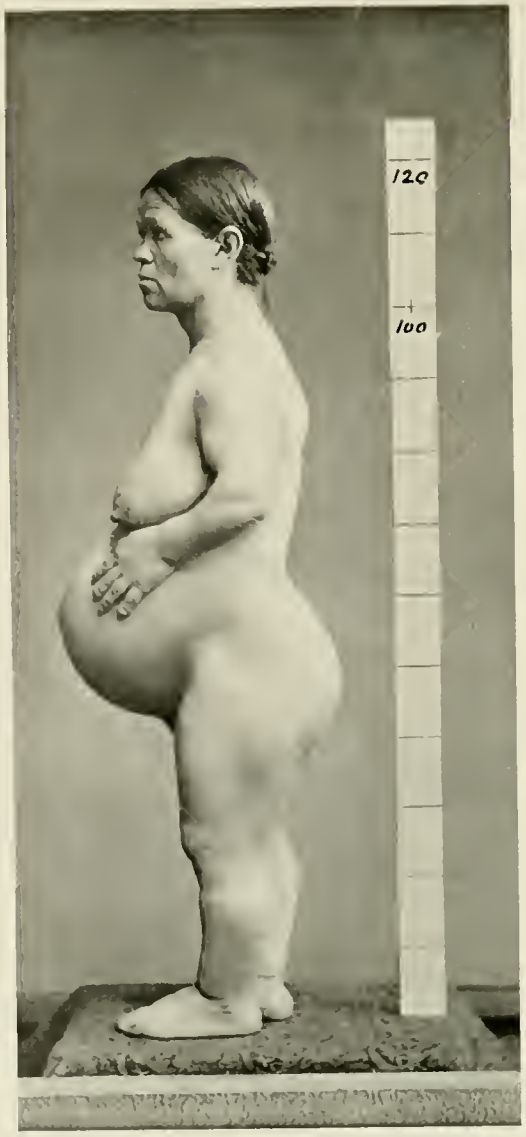


(88)

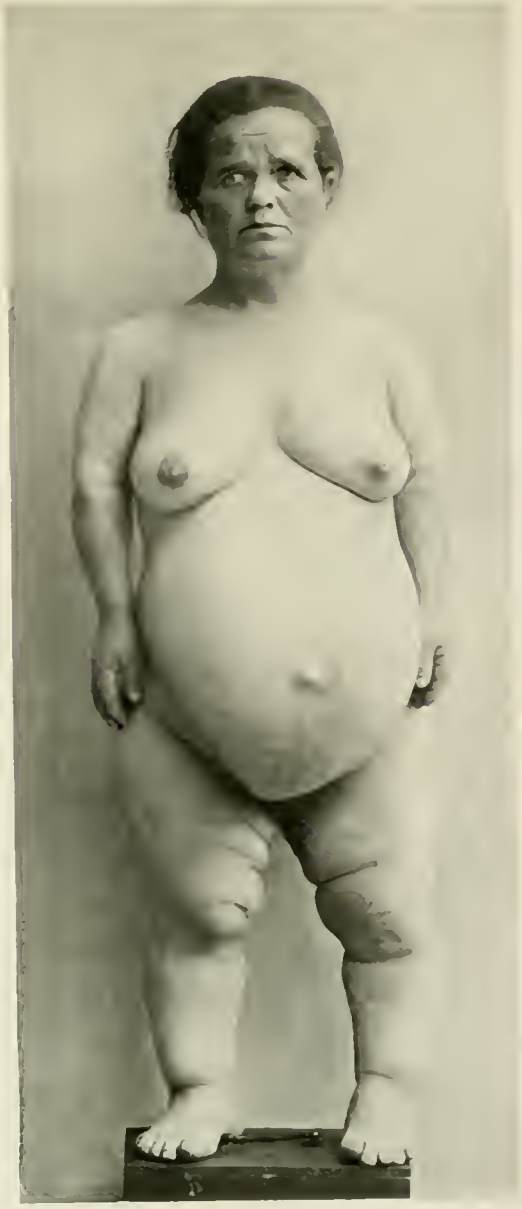
By the kindness of Professor Nijhoff of Groningen.



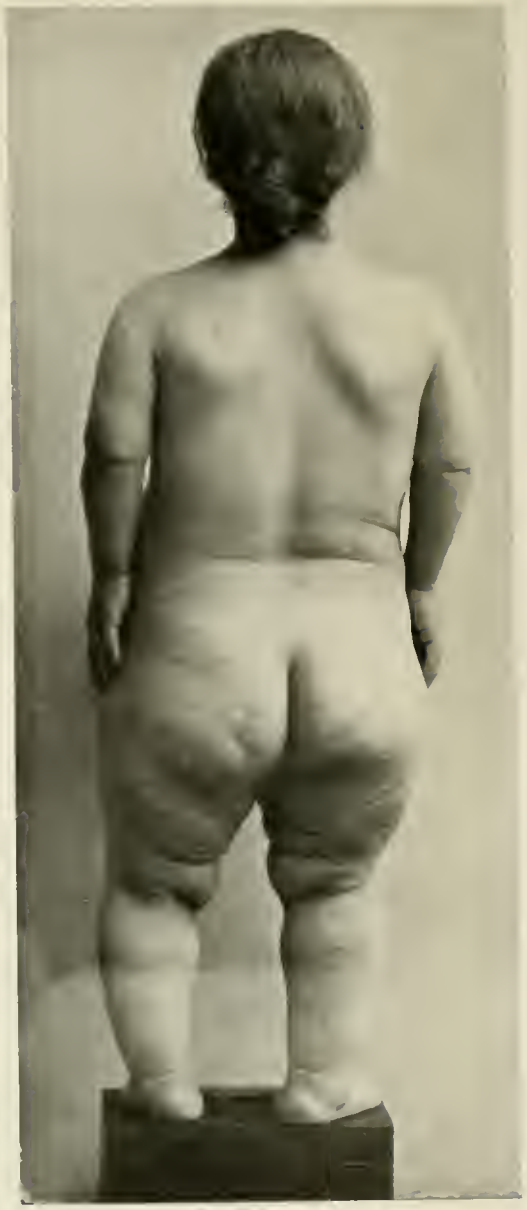
(89)



(90)



(91)



(92)

A. B. Aged ?. Stature 122 cms. Sectio caesarea, normal ♀ child.

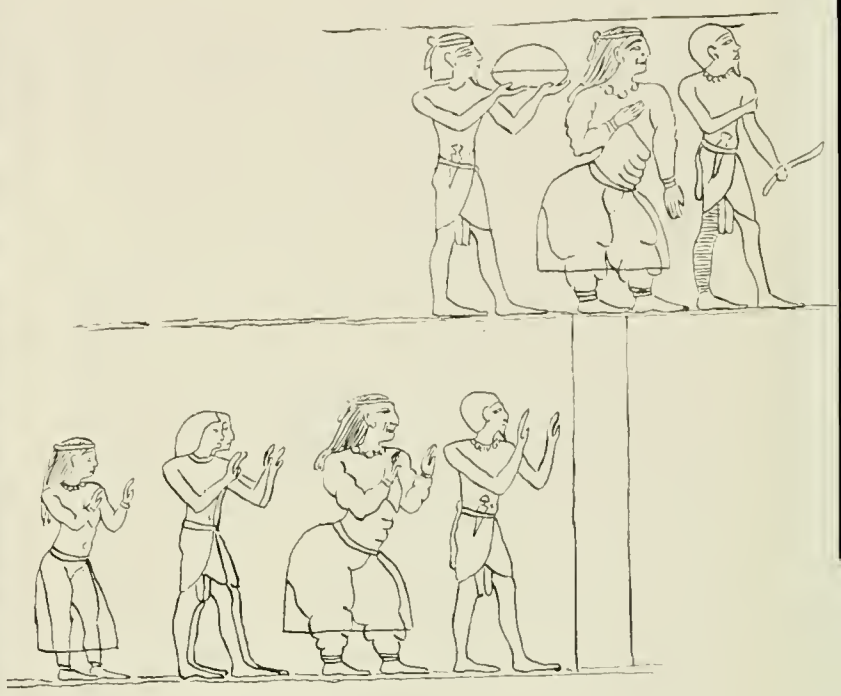
G. B. Aged 41. Stature 123 cms. Sectio caesarea. Achondroplasic ♀ child.

Two achondroplasic sisters, with achondroplasic father and four achondroplasic siblings.

By the kindness of Professor Nijhoff of Groningen.

(93[a])

ieen of Punt from a Bas-Relief, at Deir-el-Bahari B.C. 1516—1481 to be considered in conjunction with jug forms below.

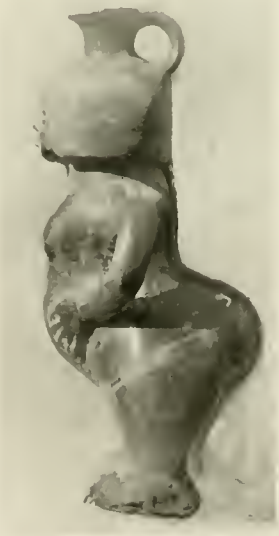


(93[b])

This outline sketch after Mariette of the Bas-Relief shows the daughter of the Queen of Punt with sensible but less marked hereditary characters.

(94)

Jug from British Museum, No. 29935.

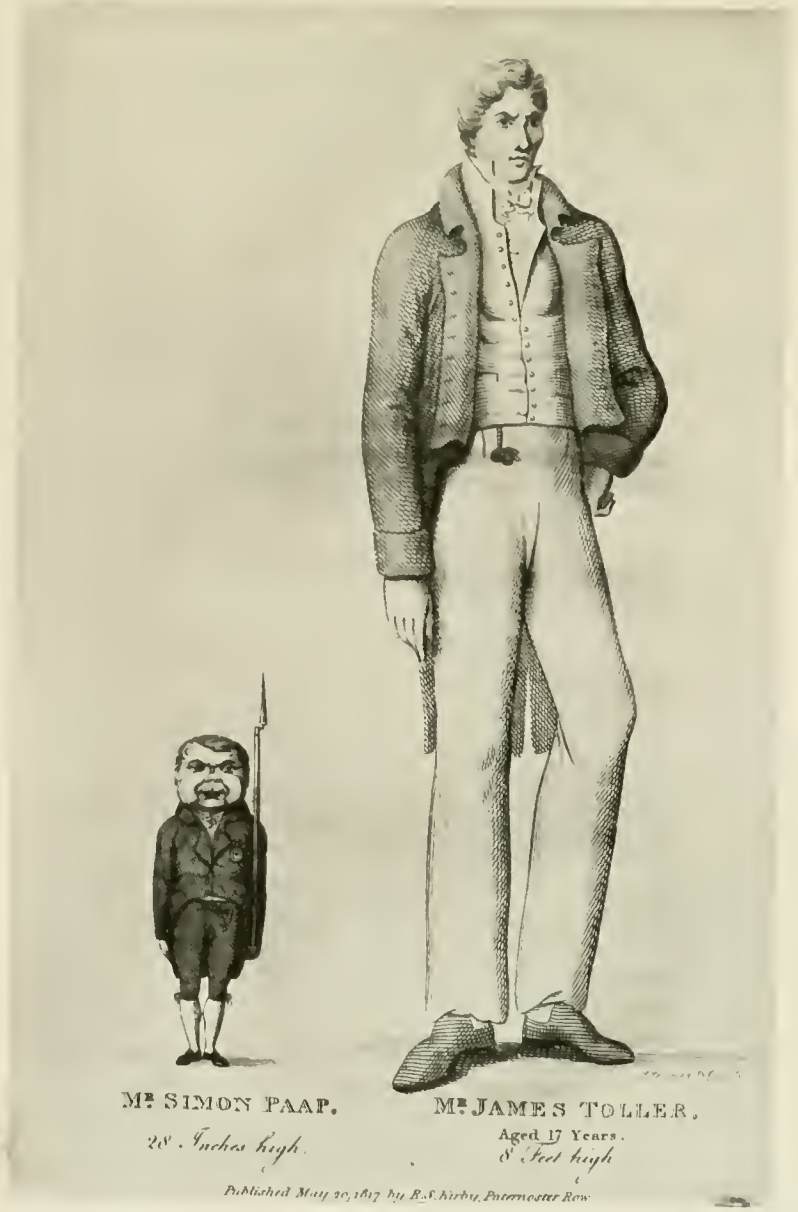


(95)

Jug from Cairo Museum. See Mace, *El Amra*, Plate L.

(96)

Ashmolean Museum, E. 2427. See Garstang, *El Arabah*, Plate XIX.



(97)

The Dwarf Simon Paap, alongside the Giant Toller, reproduced from Kirby's *Wonderful Museum*: see Iconography, No. 156. Unless the drawing is very poor, the macrocephaly of this ateleiotic dwarf must have been a marked feature.

(98)

Nicholas Ferry, Bébé, aged 18. From a wax model in Bébé's clothes with a wig of his own hair, prepared by Jeanet, his surgeon: see Iconography No. 121. Photographed from the original in the Musée Dupuytren, Paris, for this work.

DWARF DEVELOPMENT OF PATHOLOGICAL ORIGIN. ACHONDROPLASIA AND ITS ASSOCIATIONS

(99)

(100)

(101)

Dr D. W. Hunter's Case. The C. Family. Pedigree 632. H. C. (100), aged 17, achondroplasic dwarf, mentally normal. W. C. (99), aged 13, imbecile. Compare his hands with the "mains en trident" of H. C. A. C. (101), aged 16, normal. Suffered from diabetes for 7 years. Dr Hunter draws attention to the shortening of the lower limbs in A. C.

(102)

Chipeta, an achondroplasic dwarf from Nyasaland. Photographed by Dr W. Murray; provided through the kindness of Dr Hugh S. Stannus.

(103)

Congenital humeral micromelia (partial achondroplasia?) in a Bantu, Yao Tribe, Nyasaland. From a photograph most kindly provided by Dr Hugh S. Stannus.

(104)

Ragazza gigantesca by Carreno, Madrid. See Iconography No. 64 and Bibl. No. 14[b]. Anderson Photo.

(105)

Carrie Akers after Gould and Pyle. Height 34″, weight over 22 stone. See p. 361 above.

(106)

The Dwarf Barbino by V. Cioli, Florence. See Iconography No. 119. Alinari photo.

Possibly cases of "Myxoedème fruste" (Apert): see Bibl. No. 469[b]. Cf. also Plate Y (36). A photograph of the Carrache Dwarf, Iconography No. 38, which has just reached us shows that it also belongs to the class of Obese Dwarfs.

(107)

Ethnic Dwarfs. Negro Pygmies. Collection Thiers, Musée de Louvre Photo Giraudon.

(109)

Dwarf Types—? Achondroplasic. Musée de Louvre. Photo Giraudon.

(110)

Dwarf Type—? Achondroplasic. Musée de Louvre. Photo Giraudon.

(108)

Achondroplasic Dwarfs. Dwarf Gladiator and Dwarf in Cangue. Collection Oppenheim, Bibliothèque Nationale, Paris. Photo Giraudon.

(111)

Achondroplasic Dwarf Warrior. Musée de Louvre. Photo Giraudon.

(112)

Aesop as (?) ricketty Dwarf. From the South Kensington Museum. Cf. Plate QQ (95).

(113)

The Dwarf El Primo, by Velasquez, Madrid. See Iconography No. 52. Anderson photo.

(114)

The Dwarf Sebastiano de Morra, by Velasquez, Madrid. See Iconography No. 53. Anderson photo.

(115)

The Dwarf Antonio l'Inglese, by Velasquez, Madrid. See Iconography No. 54. Anderson photo.

(116)

The Dwarfs (to right by hound) Maria Barbola and Nicolasino Pertuseno. See Iconography No. 56. Anderson photo.

All these dwarfs appear to be achondroplasic with the exception of Pertuseno: see p. 359.

(117)

Commodore Nutt and Minnie Warren. Photograph, London Stereoscopic Company.

(118)

Prince Mignon. Photograph, London Stereoscopic Company.

(119)

Hastings Gilford's Case: see Fig. 731[b]. Ateleiotic father and son, with normal daughter. From a photograph most kindly provided by Mr Gilford.

(120)

Charles S. Stratton ("Tom Thumb") and his wife (Lavinia Warren, now Countess Magri). Photograph, London Stereoscopic Company.