The treasury of human inheritance.

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UNIVERSITY OF LONDON
FRANCIS GALTON LABORATORY FOR NATIONAL EUGENICS

THE TREASURY OF HUMAN INHERITANCE

PARTS I AND II

WITH 13 PLATES OF PEDIGREES AND 5 PLATES OF ILLUSTRATIONS

PLATES I—XIII PLATES A—E

PEDIGREES 1—76

LONDON:
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THE FRANCIS GALTON EUGENICS LABORATORY.

University of London, University College, Gower Street, W.C.

The Laboratory is under the supervision of Professor Karl Pearson, F.R.S., in consultation with Mr Francis Galton, F.R.S.

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National Eugenics is the study of agencies under social control that may improve or impair the racial qualities of future generations, either physically or mentally.

It is the intention of the Founder, Mr Francis Galton, that the Laboratory shall act (i) as a storehouse for statistical material bearing on the mental and physical conditions in man and the relation of these conditions to inheritance and environment, (ii) as a centre for the publication or other form of distribution of information concerning National Eugenics. Provision is made in association with the Biometric Laboratory at University College for training in Statistical Method and for assisting research workers in special Eugenic Problems.

Short courses of instruction will be provided for those engaged in social, anthropometric, or medical work and desirous of applying modern methods of analysis to the reduction of their observations.

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CONTENTS OF PARTS I—II.

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

ILLUSTRATIVE PLATES.

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

The Inheritance of Qualities in Families lies at the basis of the Science of Eugenics, and though much is known about it a much fuller inquiry is urgently needed than has hitherto been possible. Goodness and badness of physique, constitution, and abilities are distributed in similar proportions among individuals in successive generations, but the chain-work of hereditary influences through which this is effected has been most inadequately recorded. The facts of Family Inheritance, being unregistered, fall readily into oblivion as generations pass by, and an enormous amount of valuable experience is thereby irrevocably lost. The object of the *Treasury* is to remedy, as far as lies in its power, this deplorable waste of opportunity.

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.

FRANCIS GALTON.

PREFACE.

IT is with much hesitation that the resolution has been formed to bring out this *Treasury* as part of the work of the Francis Galton Laboratory for National Eugenics. There were weighty enough reasons against such an enterprise. The whole of human heredity is not based upon the inheritance of abnormality and of pathological conditions, but their inheritance must always play a large part in eugenic inquiry, it being so much easier to suggest means of eliminating the manifestly unfit as factors of race perpetuation, than to advocate acceptable methods of emphasising the fertility of the socially most valuable members of the community. The inheritance of abnormality and of special diatheses is, however, a subject which requires trained medical knowledge, and lies outside the qualifications of the present staff of this Laboratory. A second reason against the enterprise was the suggestion that the Laboratory might be considered pledged to one or another theory of inheritance, and that the essential condition for a standard collection of hereditary data—an absolutely unbiased gathering, sifting and publication of material—would be lacking in our case. Another important factor also was the great initial expense of a work of the present character and the doubt, whether, supposing we fail to obtain the support we are seeking for, we might not be wasting the funds placed at our disposal for the publication of Eugenics research.

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 issues 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 to be 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 enquiry, 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 unbiased 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

[1] Inquiries on this point should be made to the Editor of the *Treasury*, Eugenics Laboratory, University College, London.

interested in heredity whatever be their theoretical position, and whether they approach the matter from the standpoint of pathologist, biologist or eugenist. Our keenest wish would be 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 the few words he has at my request written to speed us on our way.

KARL PEARSON.

Galton Laboratory,
University of London.
February 15, 1909.

GENERAL SYMBOLS USED IN THE TREASURY.

♂, ♀ Male and female unaffected by characteristic under discussion.

♂, ♀ Male and female possessing characteristic.

♂?, ♀? Male and female probably, but not definitely possessing characteristic.

♂, ♀, ♀, ♂. Individuals possessing the characteristic to an incomplete or partial extent defined in text.

○, Individual of unknown sex.

♂–♂, A belt of this kind marks twins.

③, A numeral inserted in the circle marks a number of children represented by a single symbol.

◍, A hatched circle marks an individual for whom presence or absence of characteristic cannot be asserted.

⊗, marks a deformity, or disease, or special characteristic, which may possibly be associated with that under consideration.

♂ ♀ Descent lines without parents, mark that the individuals were offspring of the same parents, but there is no record or knowledge of these parents.

♂ ♀ ? This arrangement marks a marriage with ignorance as to whether there were or were not offspring.

♂ ♀ ⑦ This arrangement marks a marriage known to have been followed by *normal* offspring but their number is unknown.

♂ ♀ ◍ This arrangement marks a marriage followed by offspring, but neither their number, nor character is known.

S.P. = *sine prole*, under a married couple marks absence of offspring. J. M. = just married, at time of inquiry. † = dead at time of inquiry. The roman figures refer to the generation, the arabic to the individual in that particular generation; thus IV. 13 in the description refers to the thirteenth individual in the fourth generation and he can be at once identified in the pedigree.

Other symbols are employed in dealing with special classes of characteristics, but they will be found defined on the special plates and hold only for those plates.

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], M^{c}Ilraith[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 urine 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 tuberculous 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*

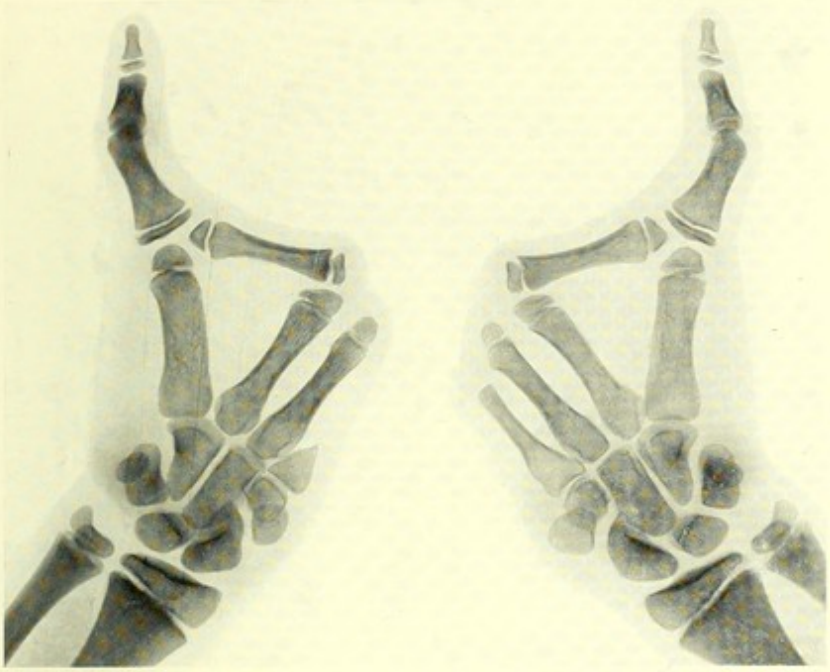

Fig. 1. Skiagram of the hands of R.E.G. (Family No. 15, V. 41). Hereditary Split-foot. The hands are seen from the dorsal aspect.

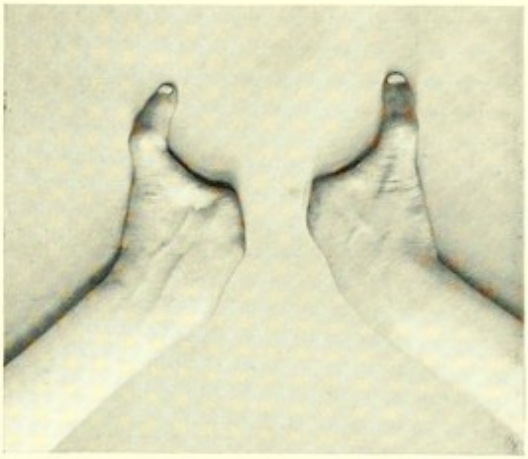

Fig. 1bis. Photograph of hands of R.E.G.

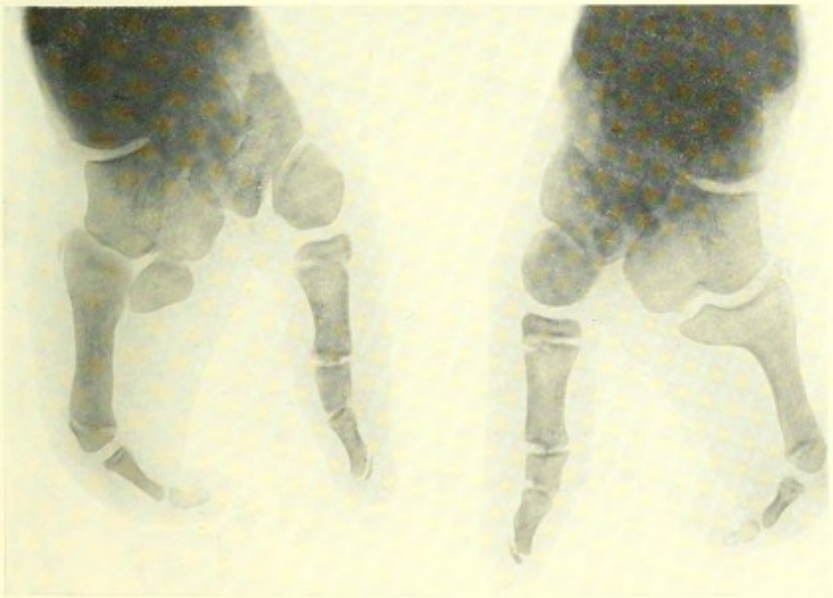

Fig. 2. Feet of R.E.G. (from dorsal aspect).

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

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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 1 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.)

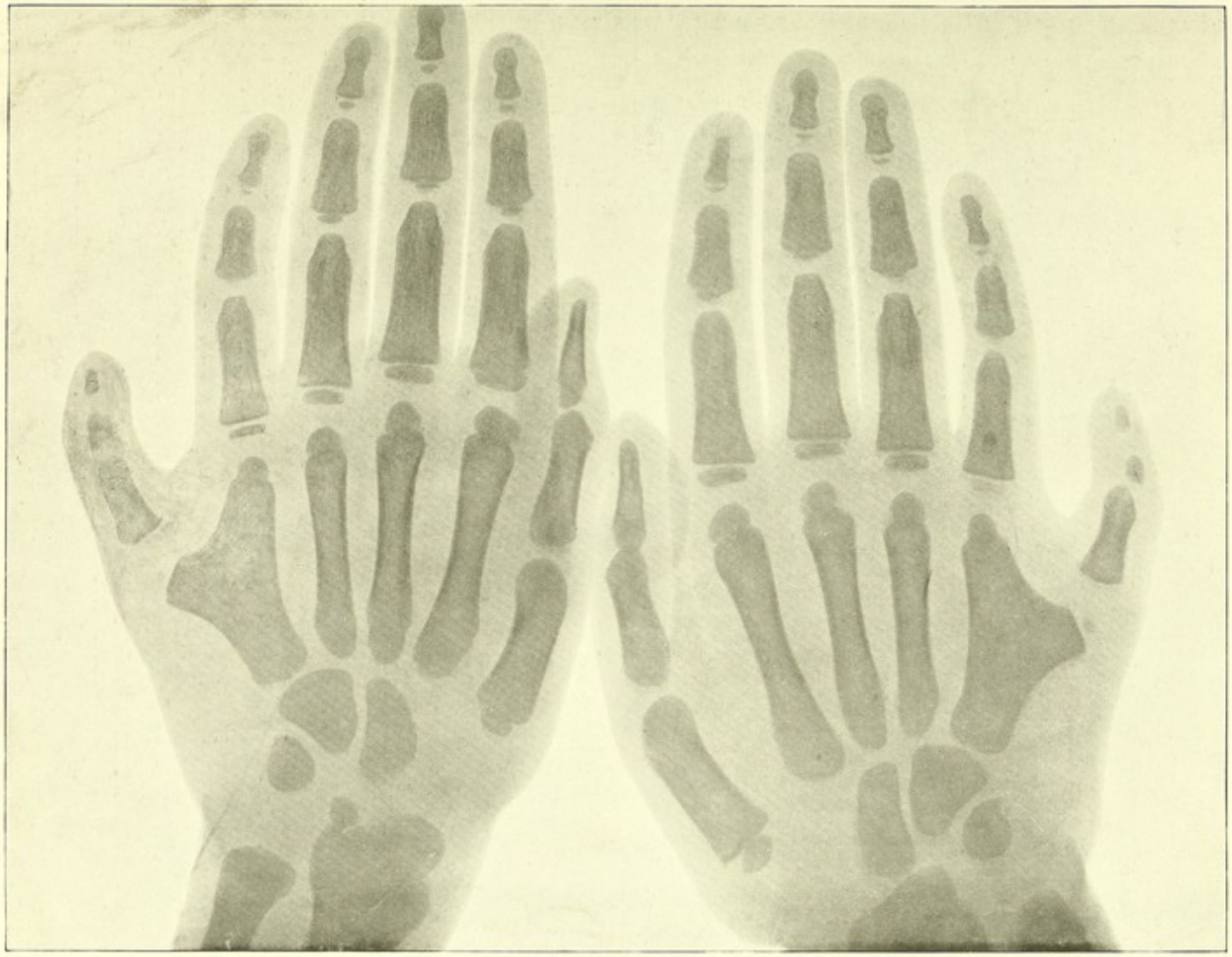
Fig. 37. *Bédart's Case.* II. 1, Auguste Faurie, *hands*, deformed; *feet*, split. III. 2, Louis Faurie, deformed *hands and feet*; III. 5, Elizabeth F., *hands and feet*, deformed. III. 7, Marie Fabre (*née* Faurie), *right hand*, digit 1 almost completely and phalanges of 2 absent, 3, 4 and 5 are syndactylised 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.

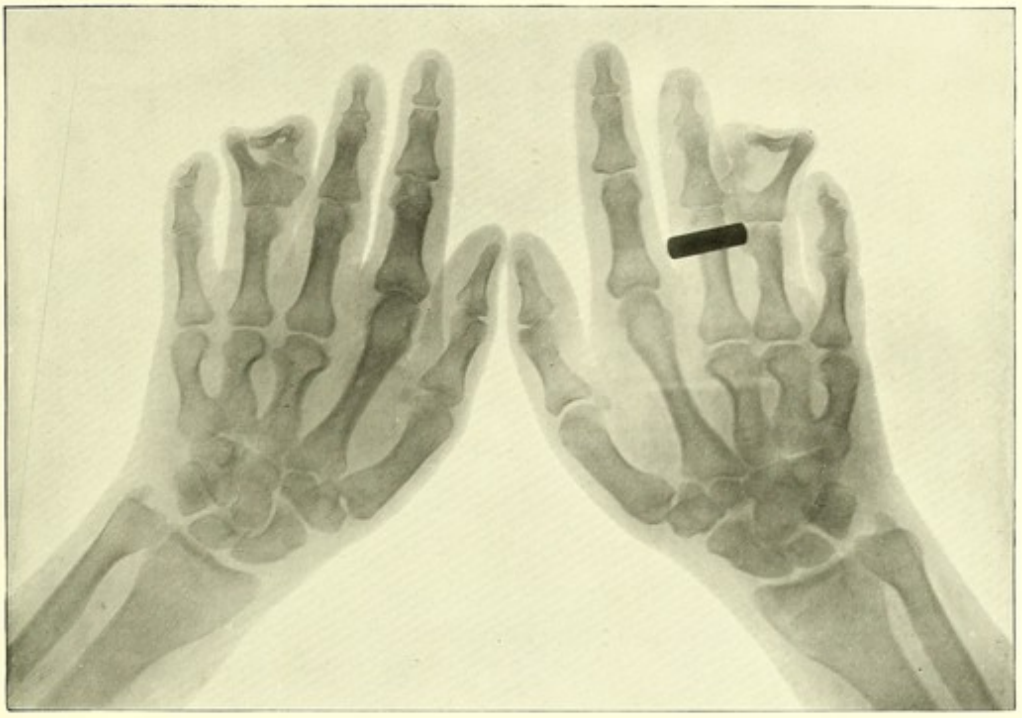


Fig. 1. Skiagram of the hands of VI. 7, Family 19. The figure, for which we are indebted to Mr Mathew of the Middlesex Hospital, shows an exceptional form of polydactylism, in that it is associated with brachydactylism. The right hand is to the left in the figure.

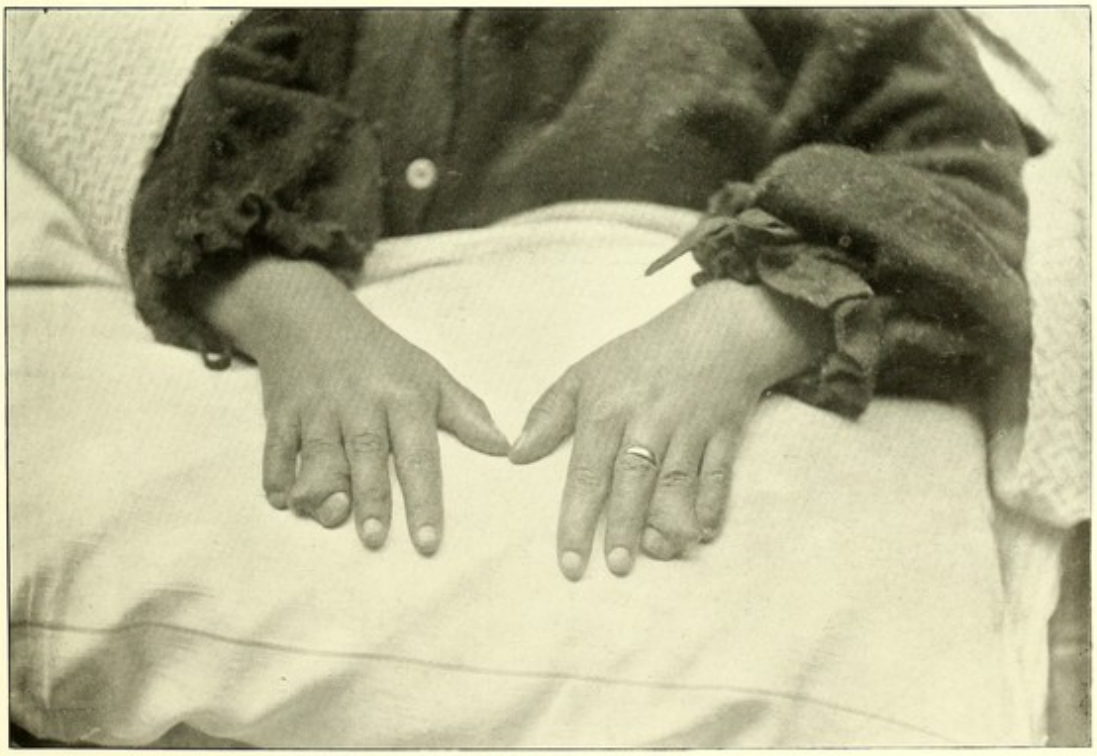


Fig. 2. Photograph of the hands of the same patient.

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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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

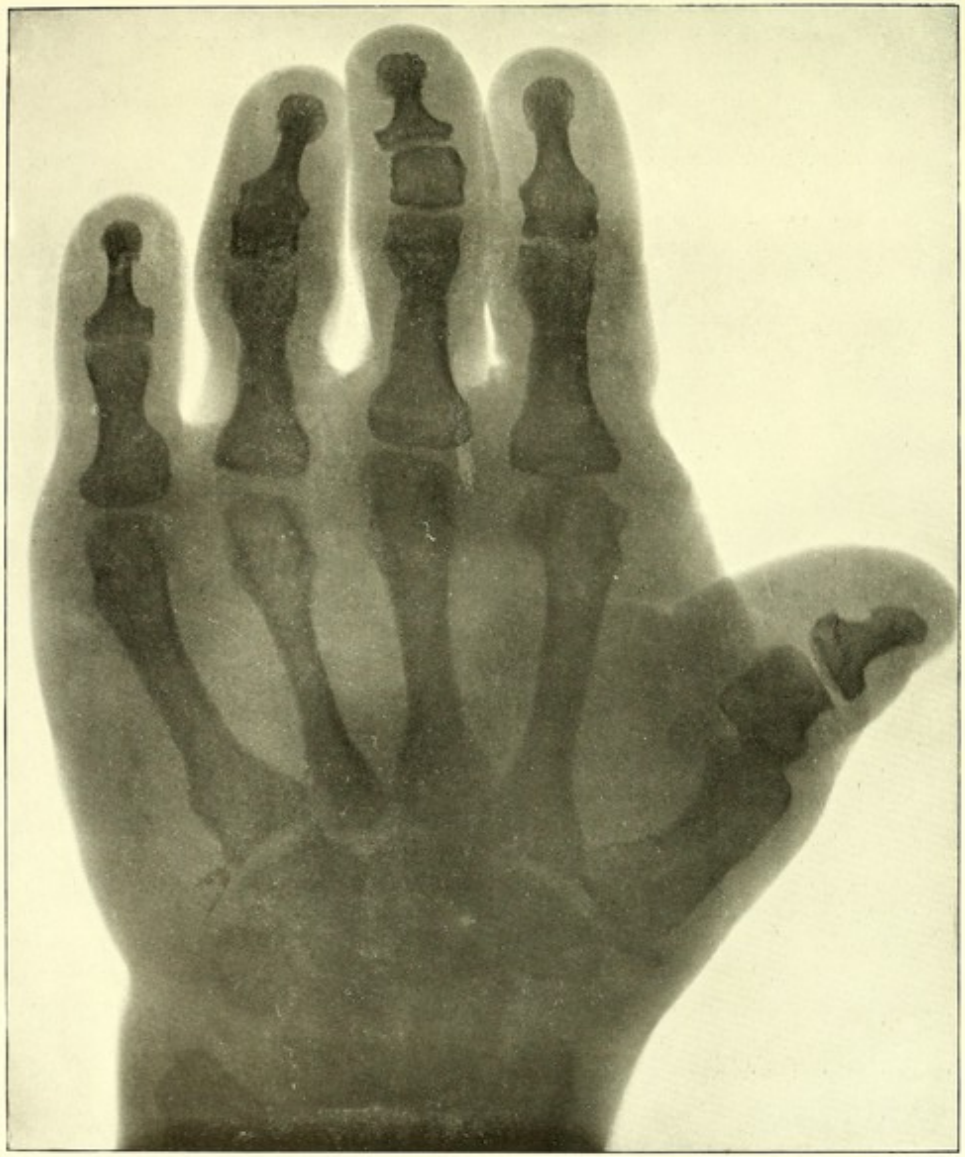

Fig. 1. Skiagram of the hands of a member of the Family 39. It shows brachydactylism in its most complete and most typical form. We are indebted for this as yet unpublished figure to Dr Drinkwater.

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	$10\frac{1}{2}$	$1\frac{7}{16}$	$3\frac{3}{4}$	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	$5\frac{3}{8}$	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	$2\frac{1}{16}$	$3\frac{3}{4}$	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	$4\frac{1}{2}$	$6\frac{3}{4}$	$8\frac{5}{8}$	$11\frac{1}{2}$	14	4	$3\frac{1}{2}$	$1\frac{3}{8}$	$3\frac{1}{2}$	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	$4\frac{1}{2}$	$7\frac{1}{4}$	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	$1\frac{1}{8}$	$2\frac{5}{8}$	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.

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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; 1. 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. $\frac{5}{1\frac{1}{2}}$, 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. $\frac{11}{1\frac{1}{2}}$, 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—$\frac{3}{1\frac{1}{2}}$, 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 α. 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.

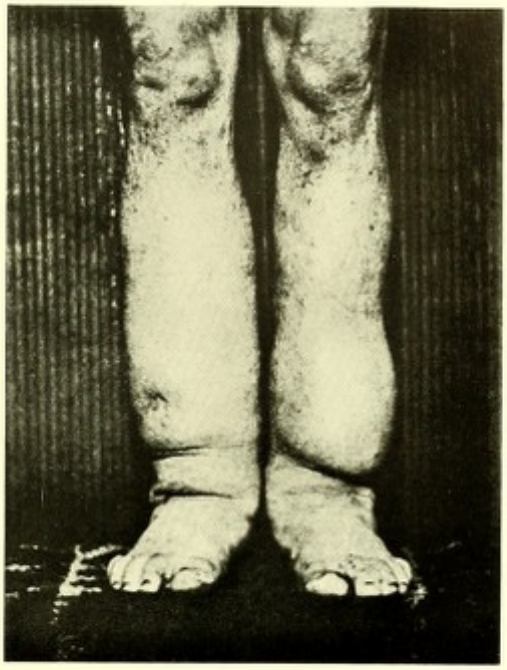

Fig. 2. Father.

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

Fig. 3. Daughter.

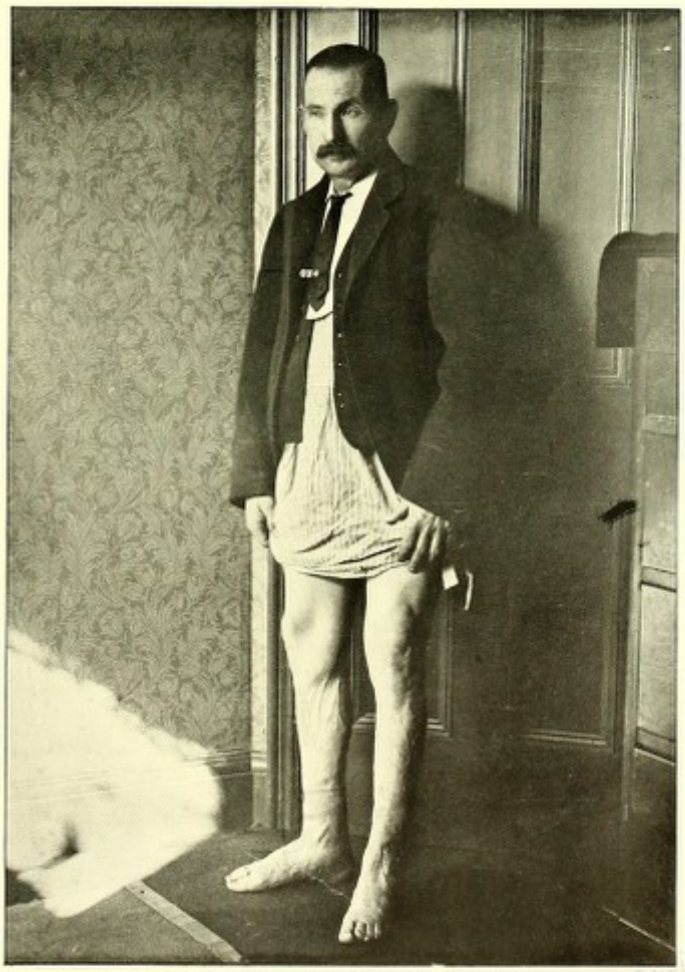

Fig. 4. Father.

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

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 a letter of enquiry sent to Dr Moyer. (See Bibl. No. 19.)

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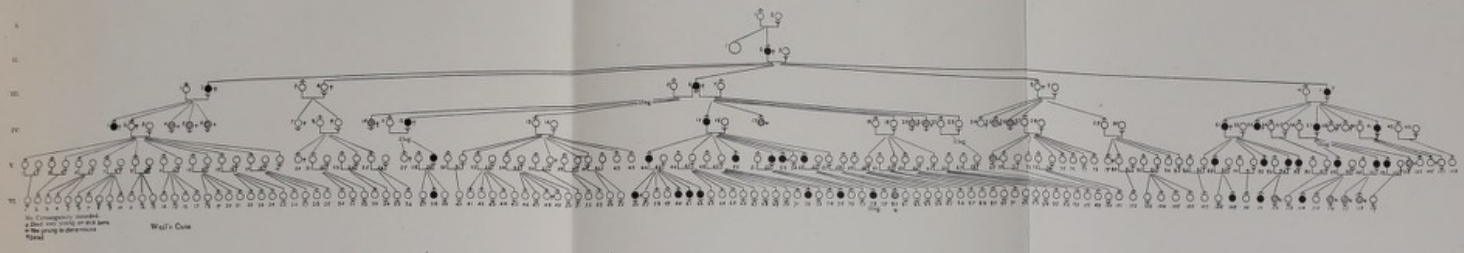
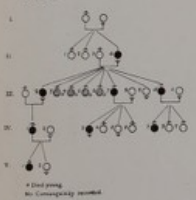
FIG. 1



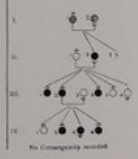
Weil's Case.

FIG. 2



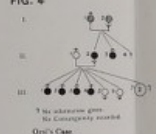
Lazarus's Case.

FIG. 3



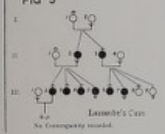
Pain's Case.

FIG. 4



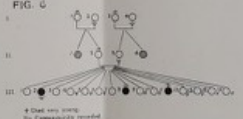
Ord's Case.

FIG. 5



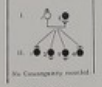
Lacombe's Case.

FIG. 6



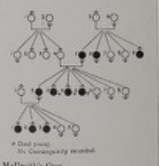
Clay's Case

FIG. 7



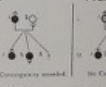
Dorbesy's Case.

FIG. 8



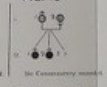
McIlraith's Case.

FIG. 9



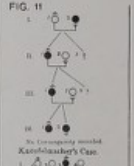
Reith's Case

FIG. 10



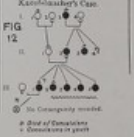
Wachsmuth's Case.

FIG. 11



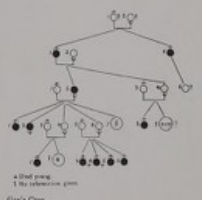
Kassowitzer's Case.

FIG 12.



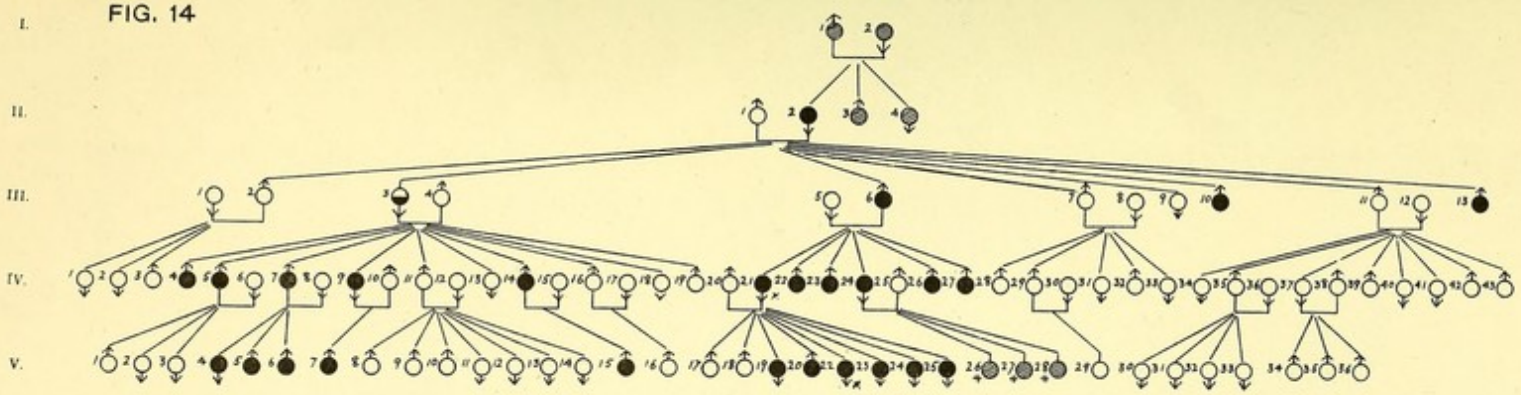
Sano's Case.

FIG. 13



Gee's Case.

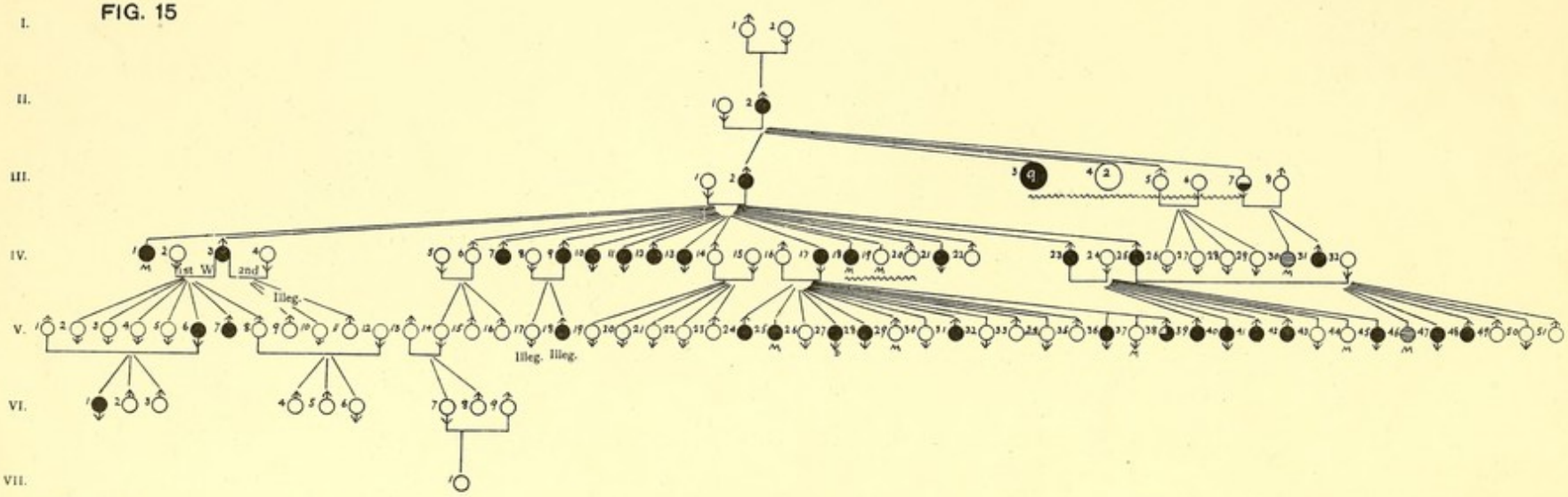
FIG. 14



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

Pearson's Case.

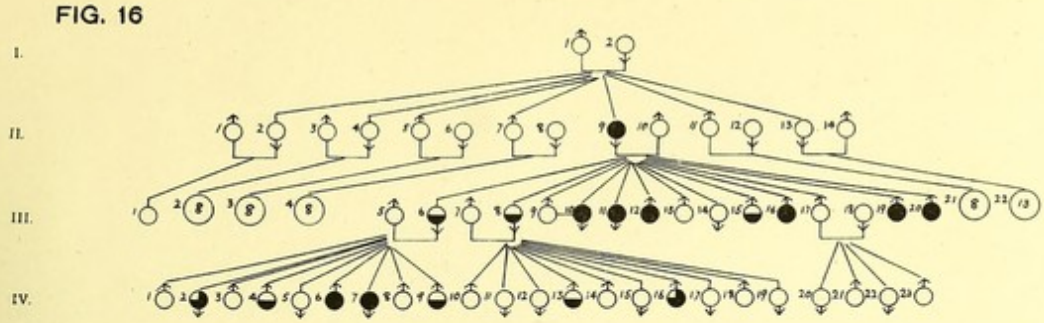
FIG. 15



No Consanguinity.
M = Miscarriage.

Lewis and Embleton's Case.

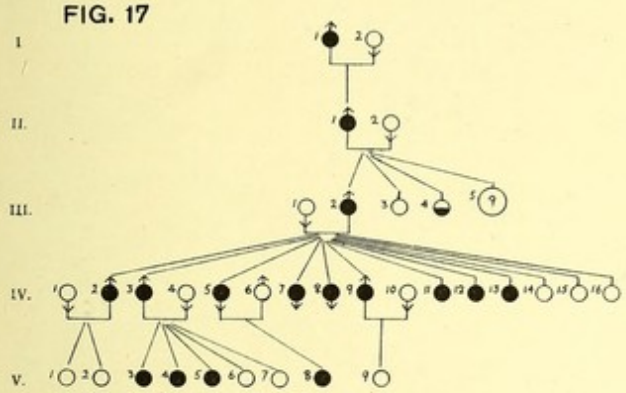
FIG. 16



No Consanguinity recorded.

Parker and Robinson's Case.

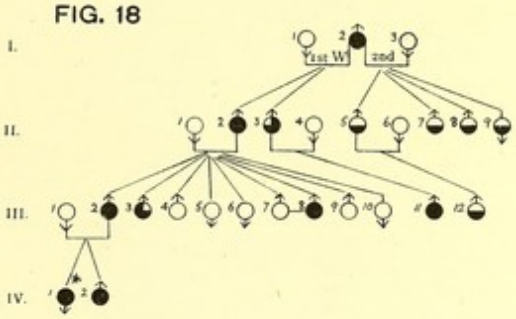
FIG. 17



No Consanguinity recorded.

Fotherby's Case.

FIG. 18



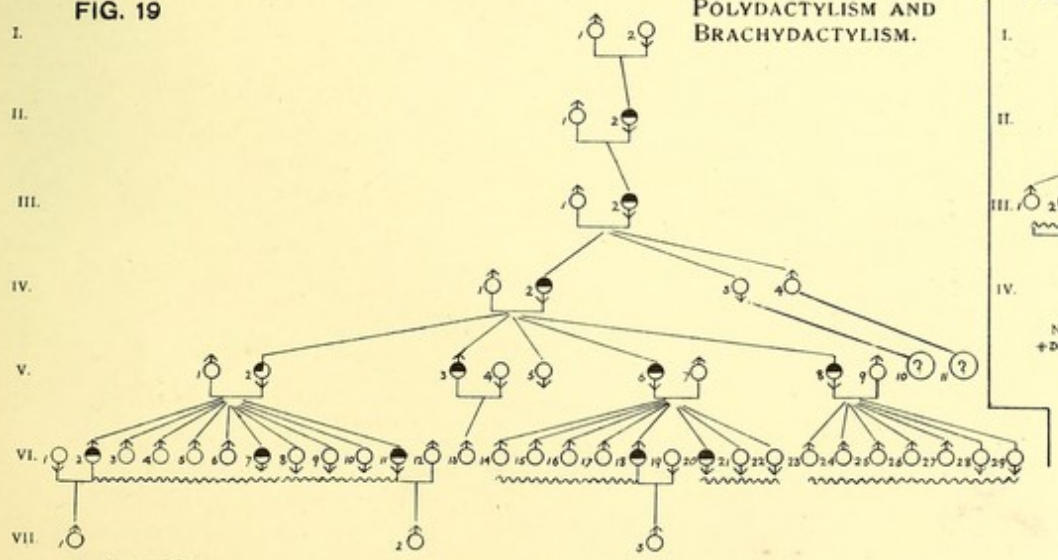
No Consanguinity recorded.

Mayer's Case.

* *Syndactylism alone*

FIG. 19

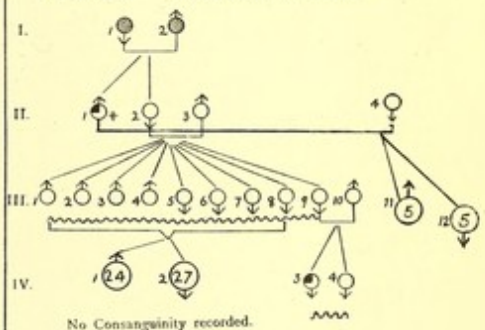
POLYDACTYLISM AND BRACHYDACTYLISM.



No Consanguinity.

Mathews' Case.

FIG. 20 POLYDACTYLISM.

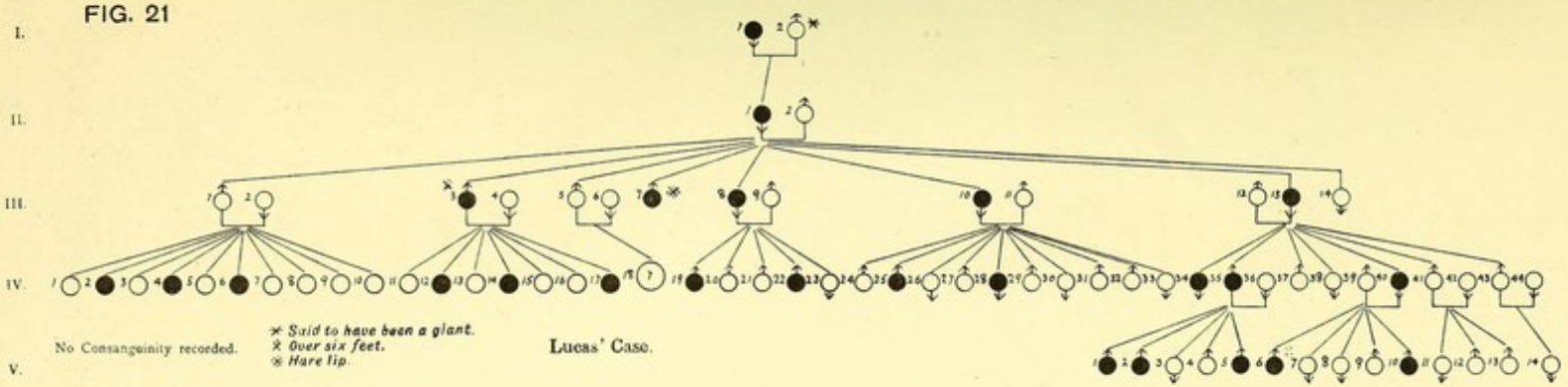


No Consanguinity recorded.

+ Doubtful left or right

Struther's Case.

FIG. 21

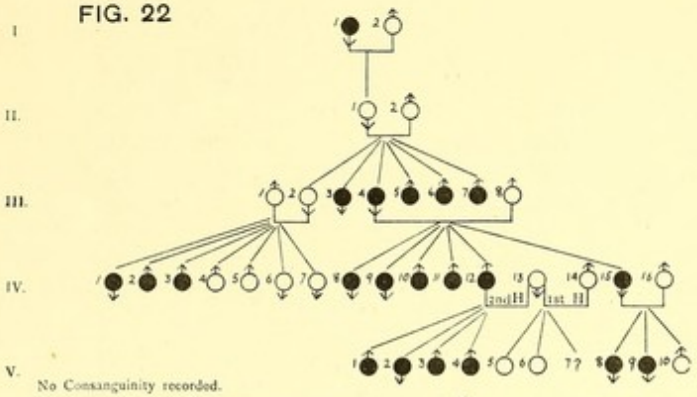


No Consanguinity recorded.

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

Lucas' Case.

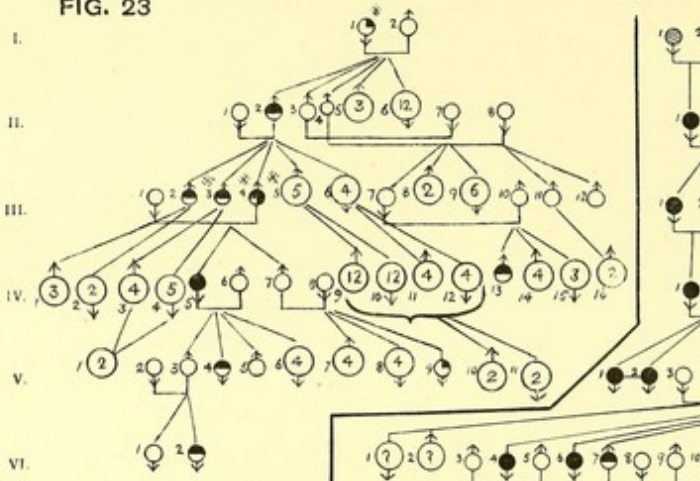
FIG. 22



No Consanguinity recorded.

Smith and Norwell's Case.

FIG. 23

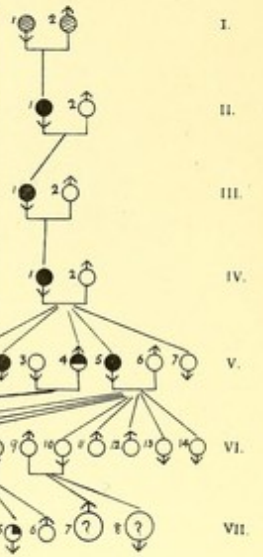


No Consanguinity recorded.

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

Struther and Wilson's Case.

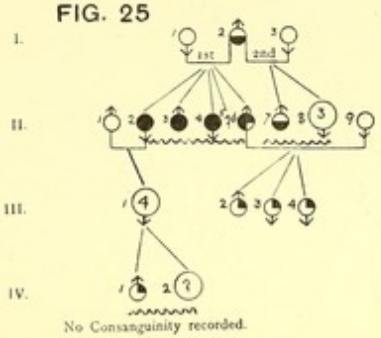
FIG. 24



No Consanguinity recorded.

McKellar's Case.

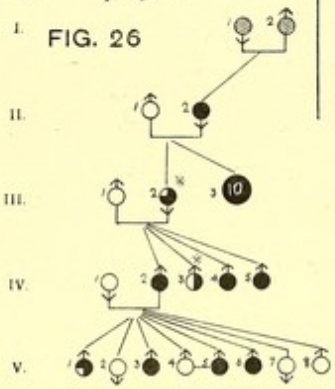
FIG. 25



No Consanguinity recorded.

Wilson's Case.

FIG. 26



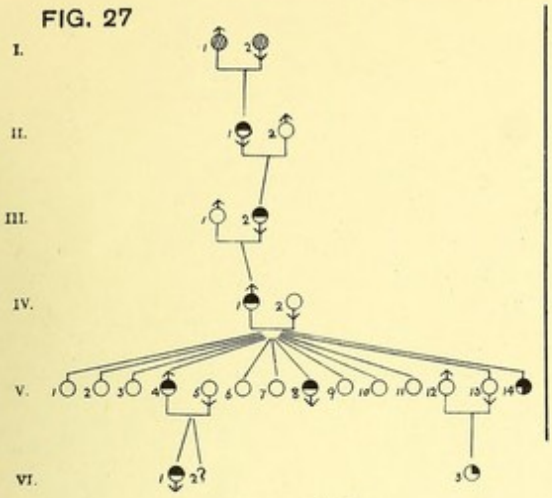
No Consanguinity recorded.

* Unaffected side unknown.

Carlisle's Case.

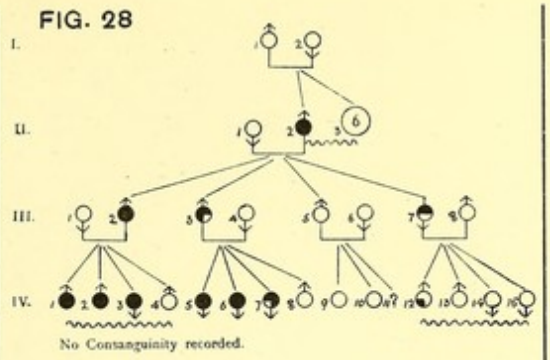
Issued by the Francis Galton Laboratory for National Eugenics.

FIG. 27



No Consanguinity recorded. Greene's Case.

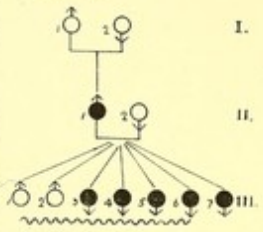
FIG. 28



No Consanguinity recorded.

Riville's Case.

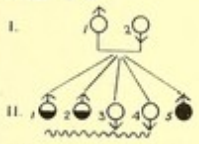
FIG. 29



No Consanguinity.

Morrish's Case.

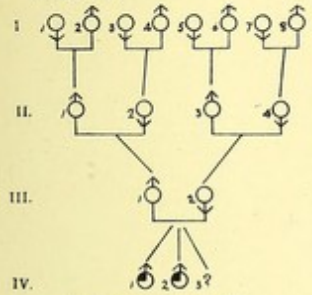
FIG. 30



No Consanguinity.

Withrow's Case.

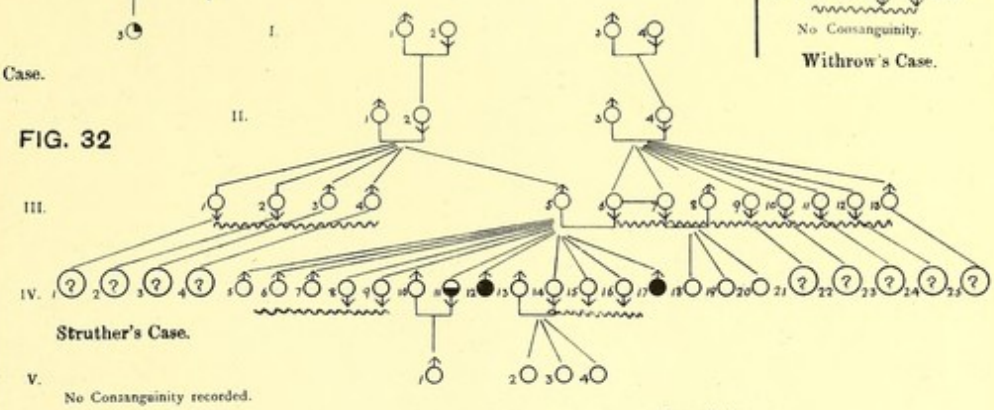
FIG. 31



No Consanguinity recorded.

Struther's Case

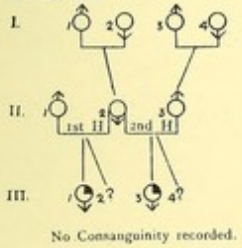
FIG. 32



Struther's Case.

No Consanguinity recorded.

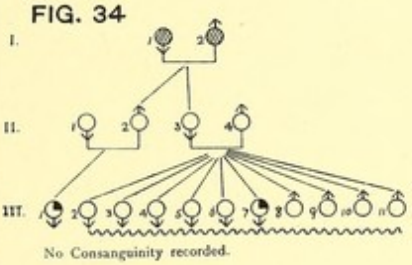
FIG. 33



No Consanguinity recorded.

Struther's Case

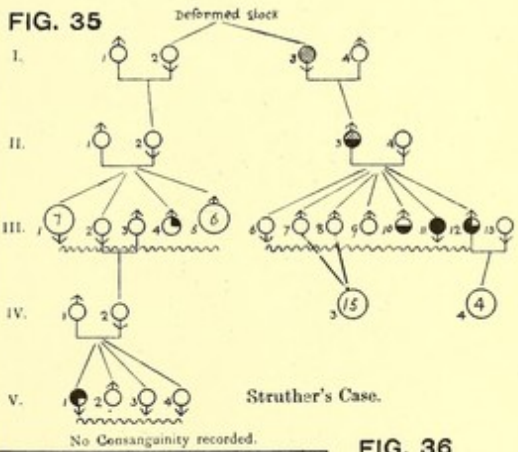
FIG. 34



No Consanguinity recorded.

Struther's Case.

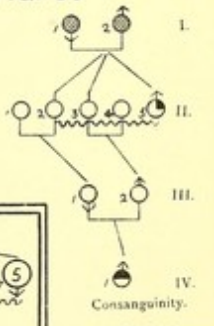
FIG. 35



Struther's Case.

No Consanguinity recorded.

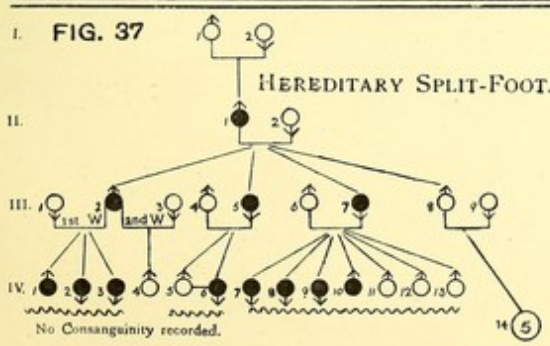
FIG. 36



Consanguinity.

Attlee's Case.

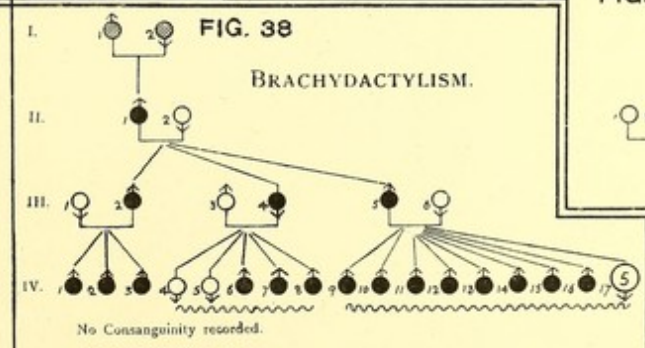
FIG. 37 HEREDITARY SPLIT-FOOT.



No Consanguinity recorded.

Bédart's Case.

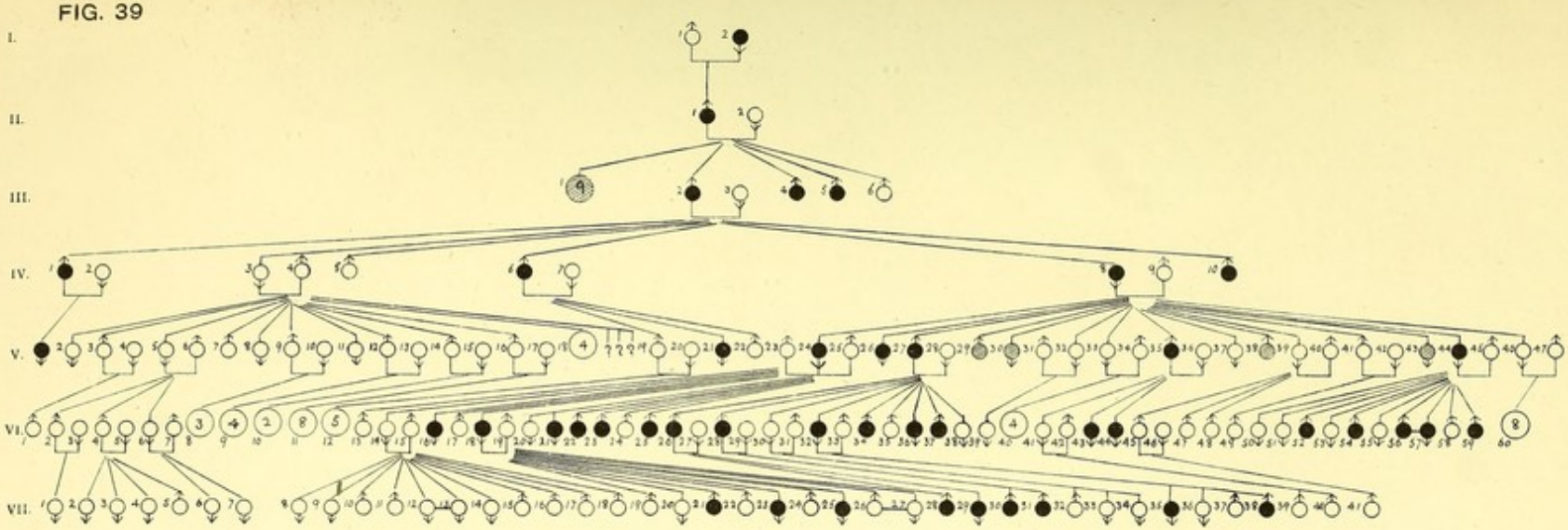
FIG. 38 BRACHYDACTYLISM.



No Consanguinity recorded.

Mercier's Case.

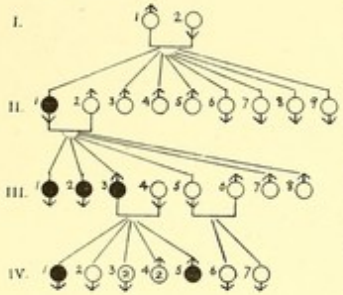
FIG. 39



No Consanguinity recorded. *Dwarfism present*

Drinkwater's Case.

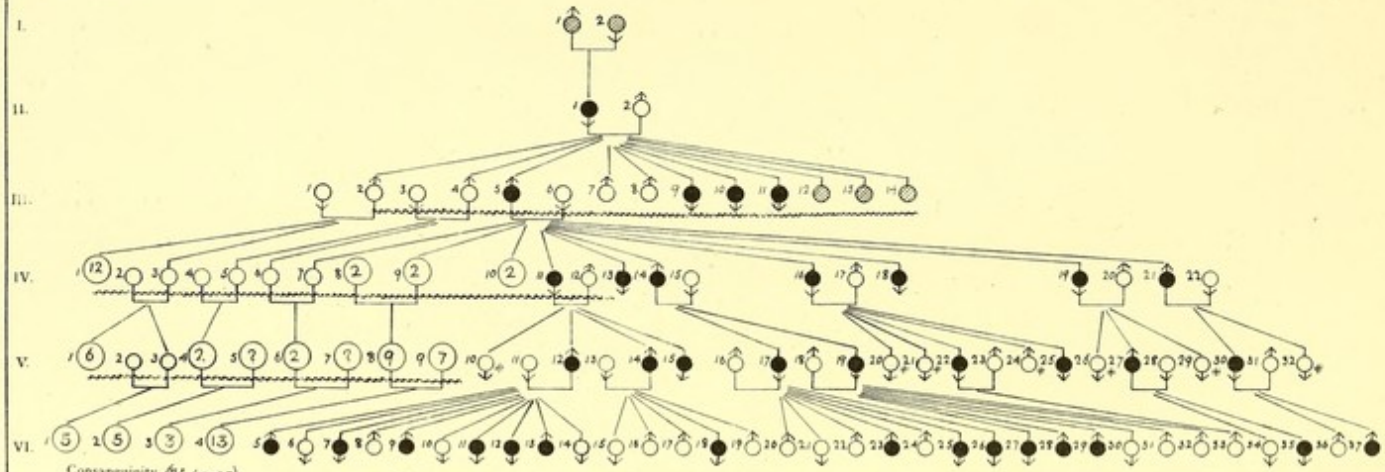
FIG. 40



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.)

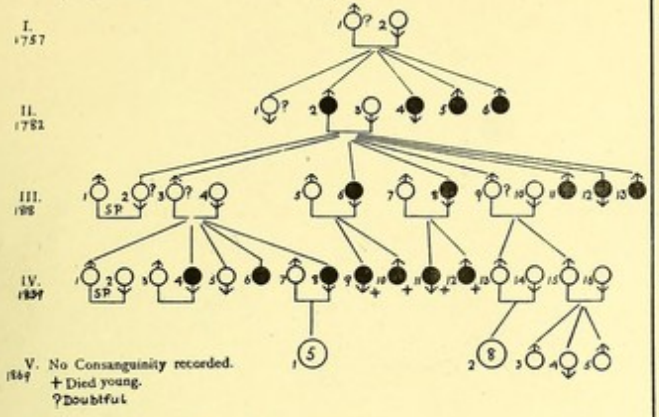


FIG. 43 Klebs' Case (ii.)

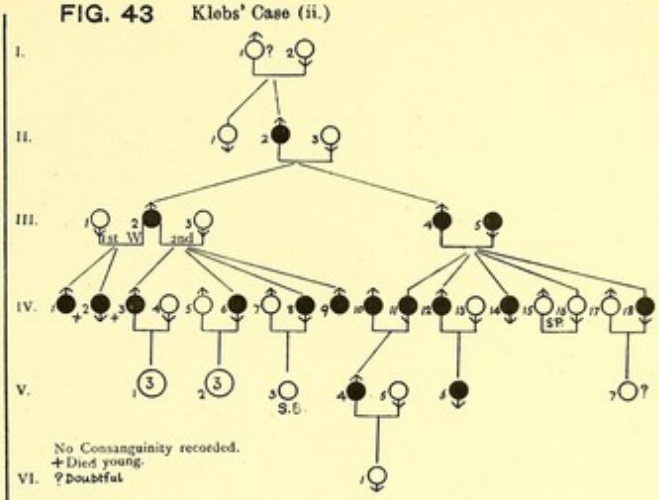


FIG. 44 Rivers' Case (i.)

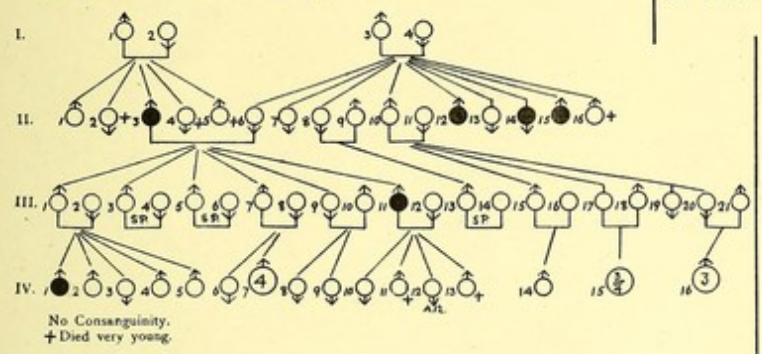


FIG. 45 Rivers' Case (ii.)

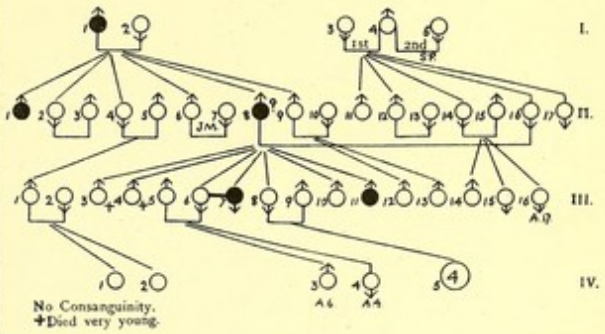


FIG. 46 Rivers' Case (iii.)

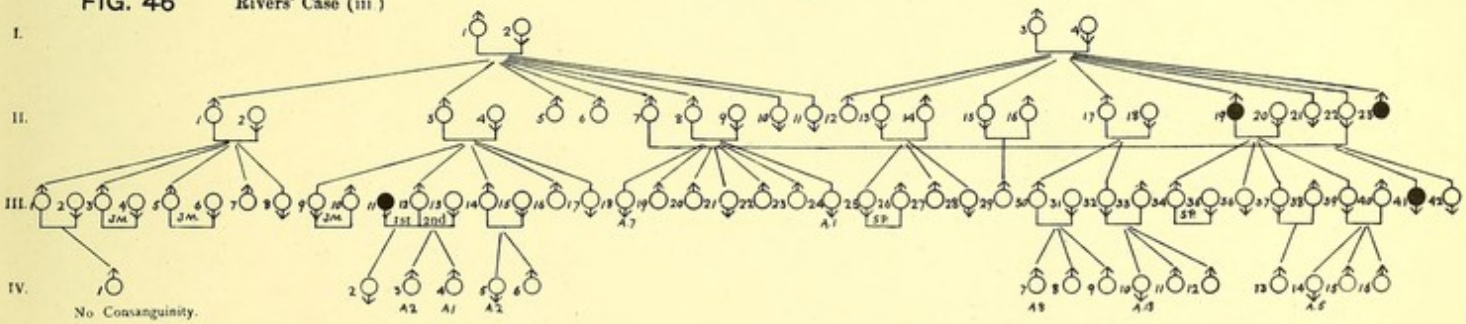


FIG. 47 Rivers' Case (iv.)

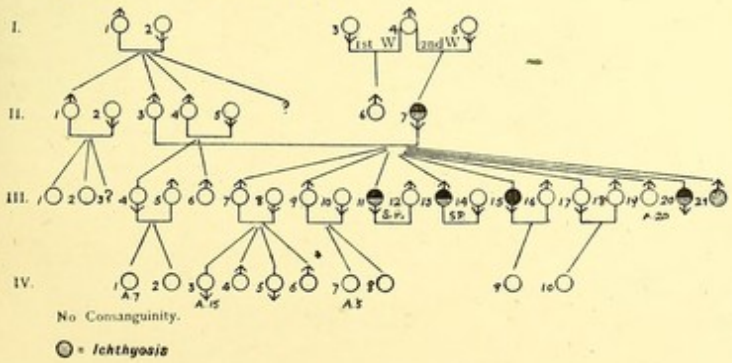


FIG. 48 Rivers' Case (v.)

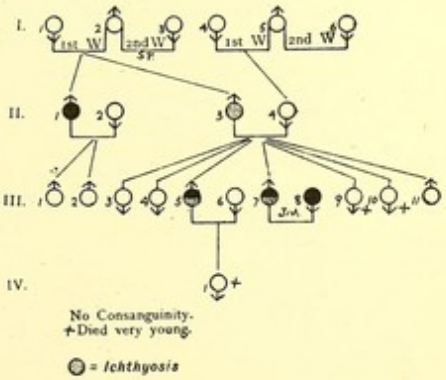


FIG. 49 Rivers' Case (vi.)

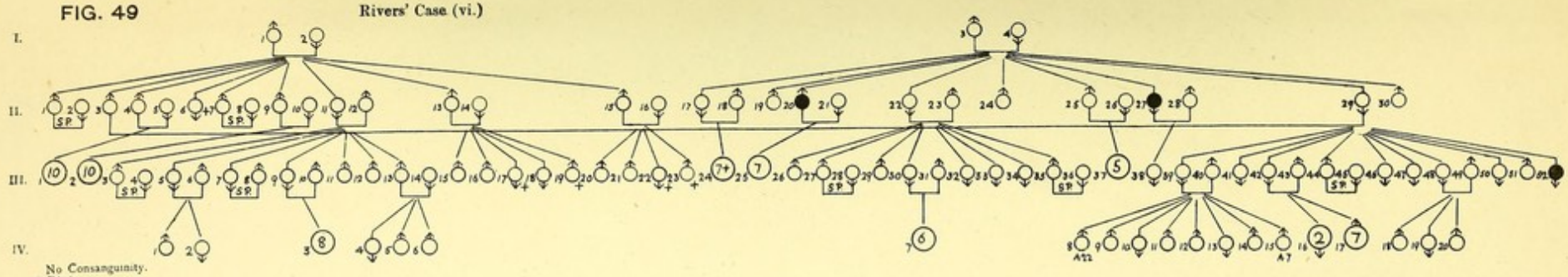


FIG. 50 Rivers' Case (vii.)

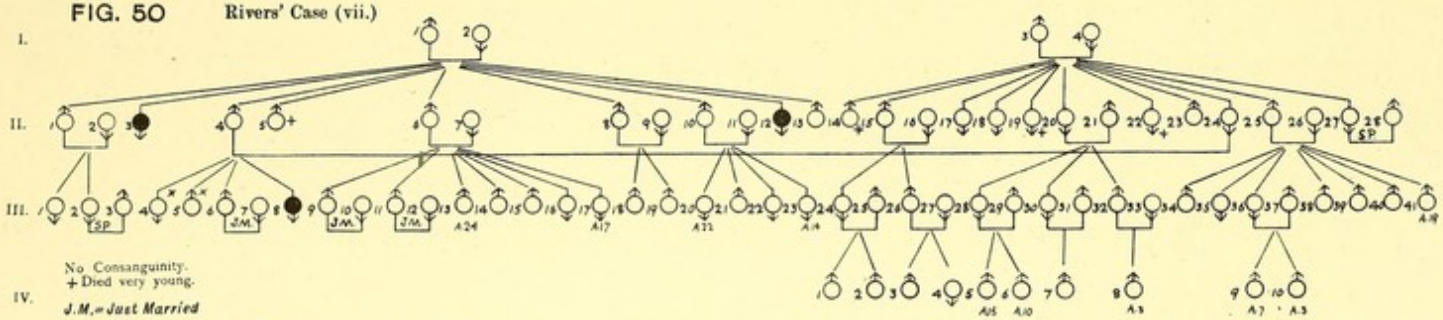


FIG. 51 Pearson's Case (i.)

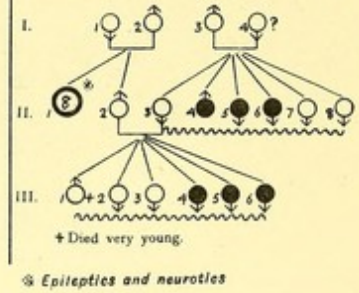


FIG. 52 Rivers' Case (viii.)

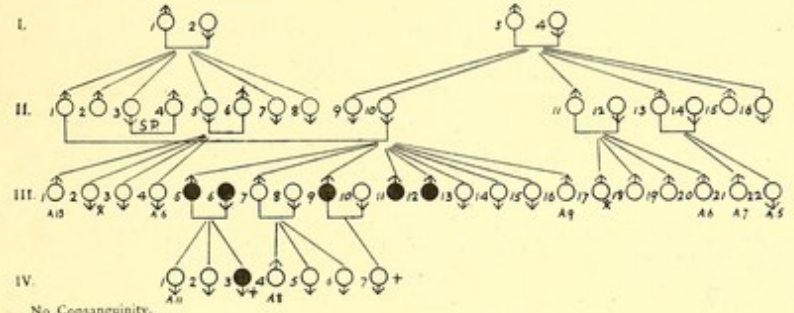


FIG. 53 Rivers' Case (ix.)

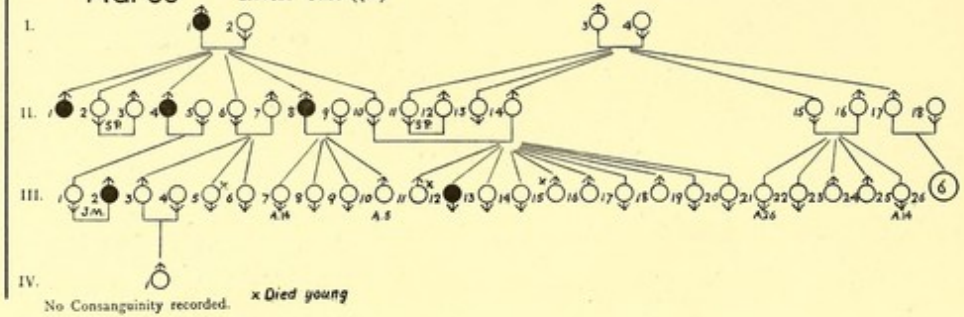


FIG. 54 Rivers' Case (x.)

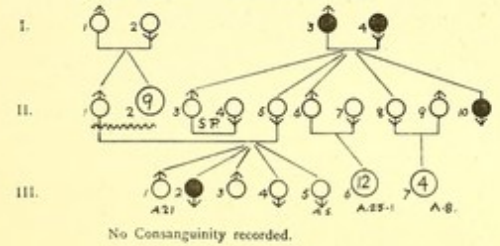


FIG. 55 Rivers' Case (xi.)

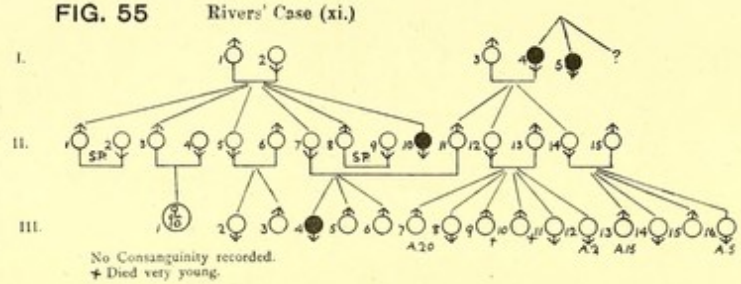


FIG. 56 Rivers' Case (xii.)

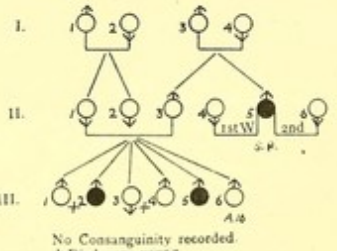


FIG. 57 Stephenson's Case.

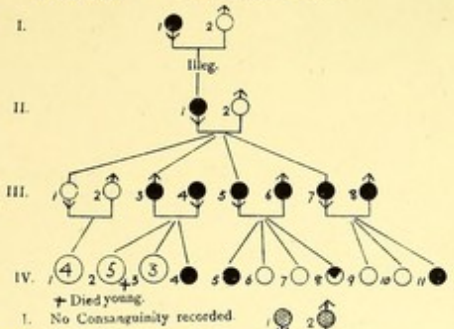


FIG. 58 Moos' Case.

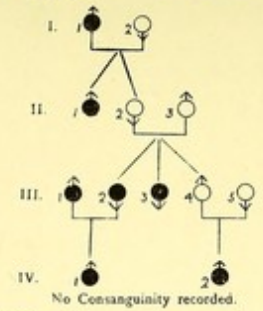


FIG. 59 Dahl's Case.

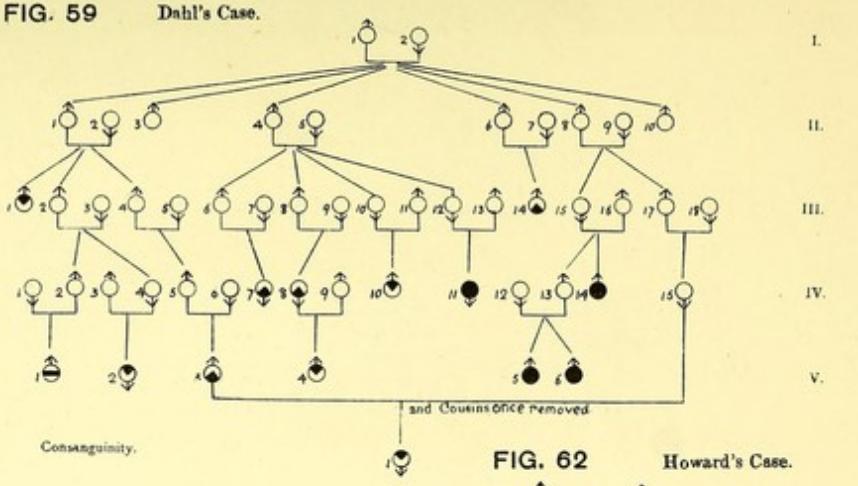


FIG. 60 Townsend's Case.

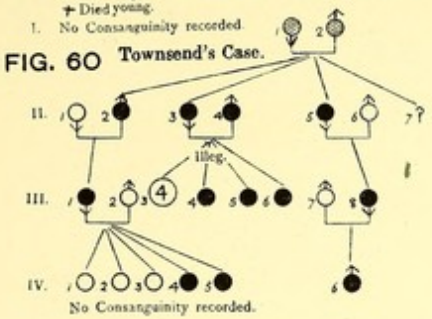


FIG. 61 Hartmann's Case.

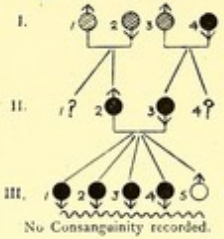


FIG. 62 Howard's Case.

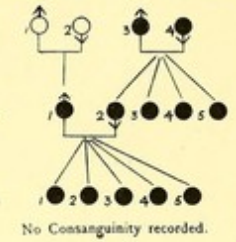


FIG. 63 Dahl's Case.

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

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FIG. 64 LEGAL AND ADMINISTRATIVE.

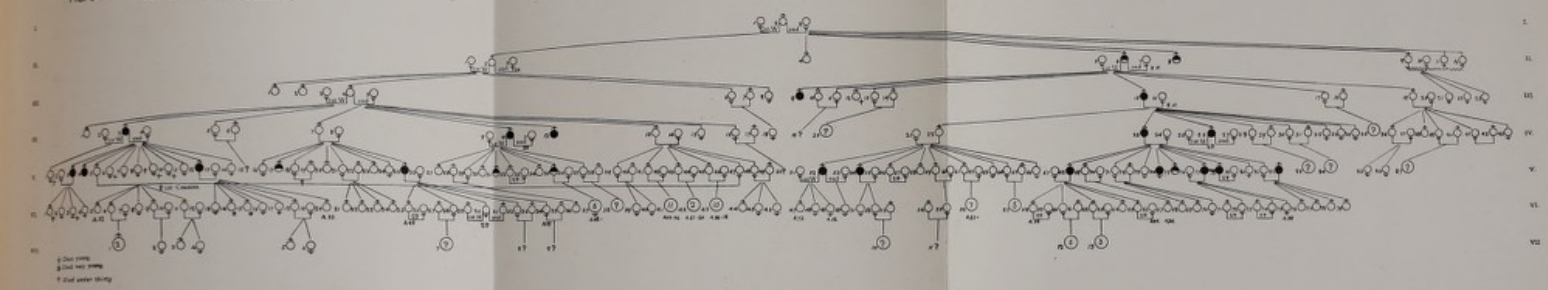

FIG. 65 LEGAL AND LITERARY.

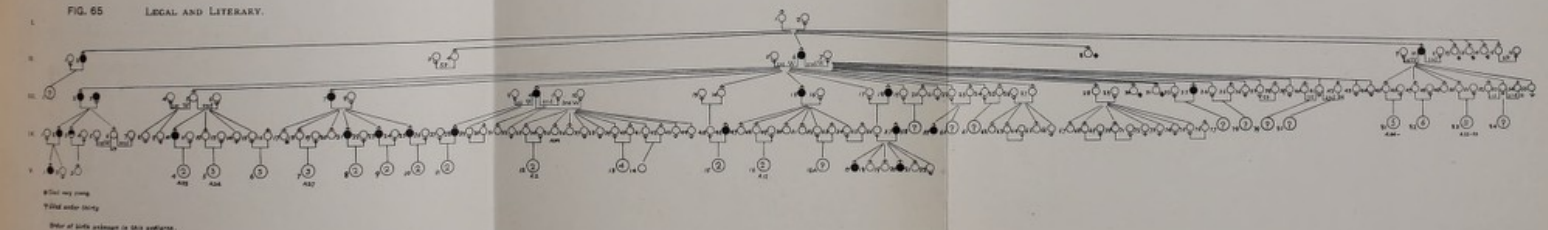

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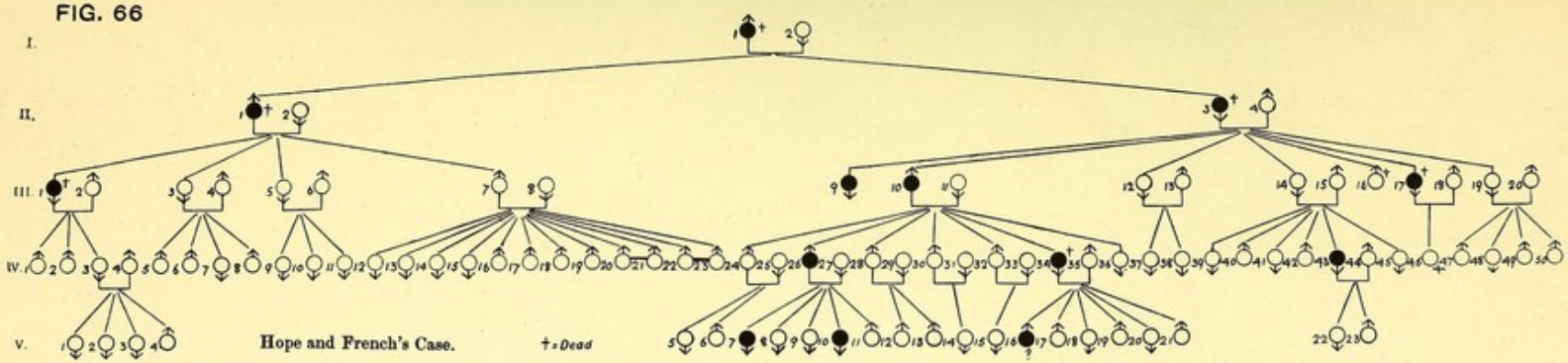
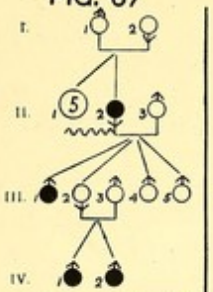
FIG. 66



Hope and French's Case. †=Dead

No Consanguinity recorded.

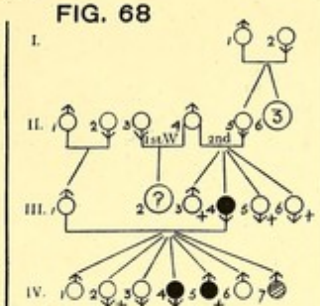
FIG. 67



No Consanguinity recorded.

Tobiesen's Case.

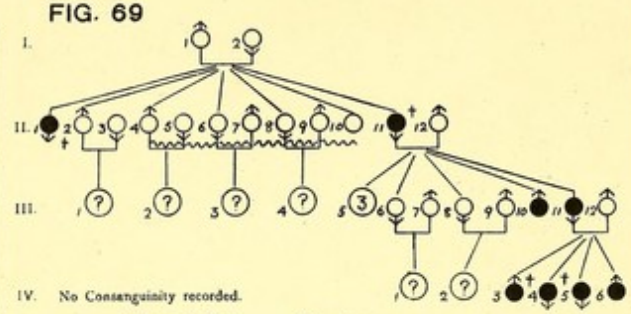
FIG. 68



No Consanguinity recorded.
+ Died young.

Rolleston's Case.

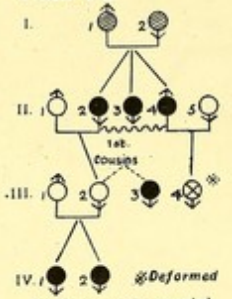
FIG. 69



IV. No Consanguinity recorded.

Nonne's Case. †=Dead

FIG. 70

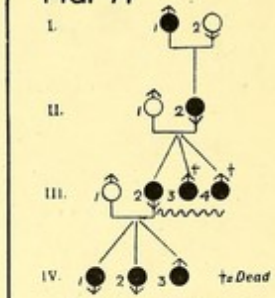


IV. ※=Deformed

No Consanguinity recorded.

Lortat-Jacob's Case.

FIG. 71

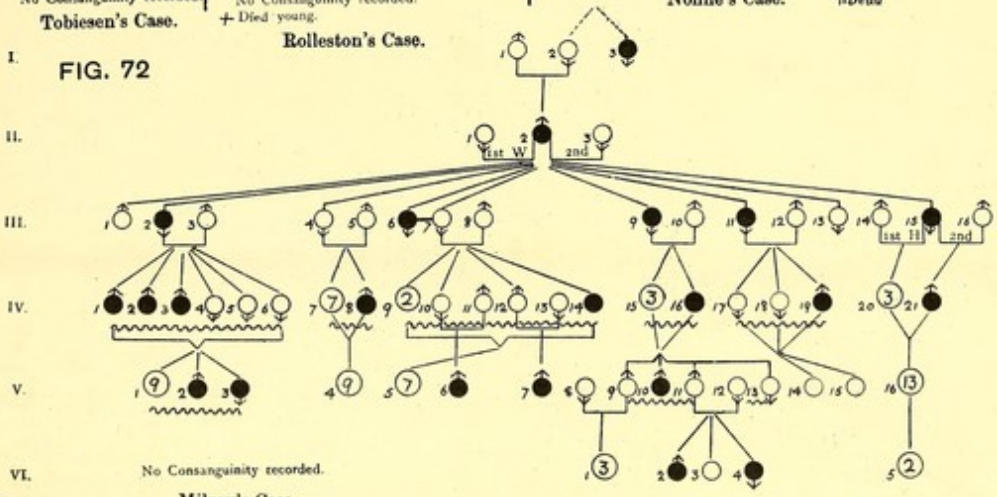


IV. †=Dead

No Consanguinity recorded.

Meige's Case.

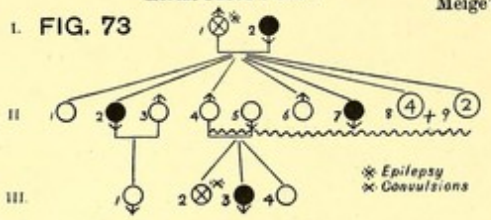
FIG. 72



No Consanguinity recorded.

Milroy's Case.

FIG. 73

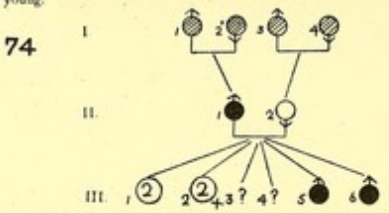


※ Epilepsy
× Convulsions

No Consanguinity recorded.
+ Died young.

Lannois' Case.

FIG. 74



No Consanguinity recorded
+ Died young

Jopson's Case.

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