Mental defect / by Lionel S. Penrose.

TEXT-BOOKS OF SOCIAL BIOLOGY

Edited by LANCELOT HOGBEN, D.Sc., M.A.

Professor of Social Biology in the University of London

MENTAL DEFECT

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MENTAL DEFECT

BY

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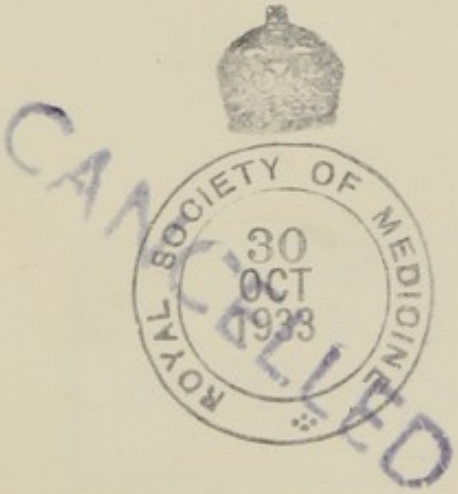

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TO

DR. F. DOUGLAS TURNER

PREFACE

THIS book is intended for the use of medical or lay persons who desire to obtain information on some of the scientific problems associated with the study of mental deficiency. The subject is wide and can be treated from many different points of view, and I have here treated at greatest length the aspects of the subject in which I, personally, am most interested.

The historical, legal and administrative sides are described in the introductory chapter with a view to making these parts of the subject intelligible to readers hitherto unacquainted with them. Less prominence is given to these topics than to questions arising directly out of the scientific study of the nature and causes of mental deficiency. In Chapters II, III, IV, V, VI and VII, the methods of investigation applicable to this branch of study are set forth. Instances of the results which can be obtained are described and some worked examples are given. The various possible ways of classifying mentally defective individuals are discussed in Chapter VIII. The next six chapters (IX, X, XI, XII, XIII and XIV) are devoted to the description of the different types of persons who are liable to be certified mentally defective under the existing laws. Treatment—radical, palliative and preventive —is considered in Chapter XV.

I consider the study of mental deficiency to be a branch of human biology. It provides a fruitful field for research when approached from this angle. In the past, however, research has been impeded by the widespread acceptance of ideas which are not in keeping with recent biological discoveries. I have, therefore, subjected the current theories of the nature and origin of mental deficiency to critical

examination. A glossary is appended for the use of those readers who are not familiar with the technicalities of the subject matter.

In preparing the book for publication, I have received helpful criticism from a great number of people. I thank them all sincerely for their kind cooperation, and, particularly, I wish to express my obligation to Dr. Margaret Penrose, Miss Lang Brown, Dr. E. O. Lewis and Dr. R. M. Stewart.

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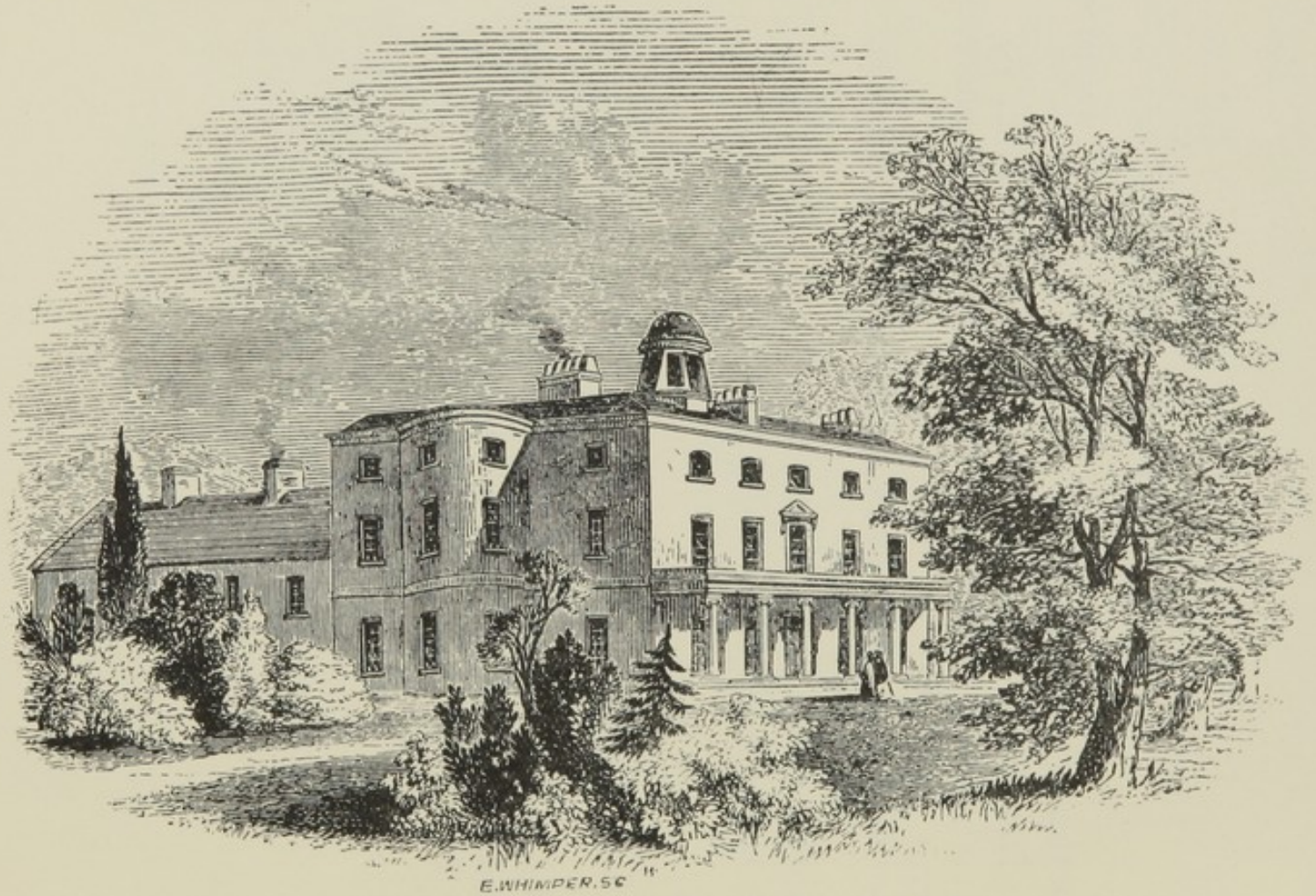


PARK HOUSE, HIGHGATE, IN 1847. (See p. 2.)

MENTAL DEFECT

CHAPTER I

INTRODUCTION

Early History—Recent History—Legal Distinction between Different Types of Mental Abnormality—Mental Deficiency Acts.

Early History.—We live in an age of scientific advances so rapid that a subject which could formerly be understood by every educated person becomes the exclusive province of the expert in the course of a few decades. In the study of mental deficiency there is no exception to this rule. The actual acquaintance of most people with the facts of mental deficiency is confined to the knowledge that some children are more backward at school than others and that there are in the community individuals of the "village idiot" type who are obviously weak-minded. The remainder of their ideas, usually derived from newspapers, are often inaccurate and frequently influenced by some political or sociological bias. Furthermore, the distinction between mental defect and mental derangement has not been fully appreciated, and, even at the present time, this division is by no means clear, practically or theoretically. Institutions for the insane still contain a proportion of defectives and many persons certified mentally defective are mentally deranged. And the average medical practitioner is content with the word "mental," which describes the whole class and exonerates him from having to probe further into the matter.

Apart from speculative views concerning the treatment of defectives in ancient times and in the Middle Ages, history has little to tell us before the beginning of the nineteenth century, when the serious study of the problem of mental deficiency

began. The attempt made by Itard to educate the " wild boy " of Aveyron, which began in 1798, attracted a good deal of attention.[1] His example was followed out on a larger scale by his pupil, Séguin, who, in 1837, founded a centre for the training of idiots in Paris. Soon afterwards the need for providing special institutions for those persons who were obviously idiots was recognized in England. The training and care of these individuals was considered an eminently worthy object of charity. For example, the philanthropist, Andrew Reed, expressed, in 1840, the hope that he might be allowed to do something for fellow creatures who were separate[2] and alone, but " the Divine image stamped upon all." Largely owing to his efforts the first asylum for idiots in this country was founded at Park House, Highgate. It was a charitable institution under the patronage of Her Majesty Queen Victoria and the Prince Consort. " The principle which rules," said Reed, " is charity—Divine Charity." The success of the institution was so great that it was soon found necessary to expand it and, in 1849, Essex Hall, Colchester, was started as an annex for nearly a hundred patients, with a view to teaching them simple mechanical employments. During the next few years the directors began to build a " model asylum " at Earlswood, Surrey. In 1855 all the patients at Park House were transferred to Earlswood, and three years later Essex Hall became the Eastern Counties Asylum. Meanwhile parallel developments were taking place in America with the founding of a private school and also a State school for the teaching of idiots in Massachusetts. The first German schools for mental defectives were started in 1866–7 at Halle and Dresden.

It is important to remember that most of these early institutions were supported voluntarily. Although interest in the subject was to some extent maintained by the prospect of curing patients by training, there were strong humanitarian and religious motives for subscribing to these ventures.

[1] *The Wild Boy of Aveyron*, tr. by Humphrey, G. and M., New York, The Century Company, 1932.

[2] The Greek word from which " idiot " is derived means " apart, separate," and also " lay."

The following extract is from one of the earliest annual reports of Park House.[1]

> Those who make this appeal do it with confidence—the confidence of those who have before challenged public benevolence, and not in vain. Can it be in vain now? It is for the poor, poor Idiot they plead!—for the Idiot, the lowest of all the objects of Christian sympathy—for the Idiot, most needing charity, and for whom charity has done nothing. We ask that he may be elevated from existence to life—from animal being to manhood—from vacancy and unconsciousness to reason and reflection. We ask that his soul may be disimprisoned; that he may look forth from the body with meaning and intelligence on a world full of expression; that he may, as a fellow, discourse with his fellows; that he may cease to be a burden on society, and become a blessing; that he may be qualified to know his Maker, and look beyond our present imperfect modes of being to perfected life in a glorious and everlasting future!

Another extract from the same source[2] a few years later shows how the interest, already stimulated, was reinforced.

> . . . The early friends of the poor Idiot will not forsake the work of their own hands; they will retain their first love; . . . they will stand by us, in seeking to obtain a much greater benefit to the PATIENT, and to SCIENCE, and to the WORLD, than has yet been secured; and this they will do for those who can make no return,—for those who, because they are so helpless, so innocent, and yet so suffering, have been truly called, in the language of benevolence and piety, GOD'S CHILDREN.

Recent History.—Since those days there has been an extraordinary change of attitude among members of the general public and the medical profession towards the unfortunate idiots. In speaking of "Absolute, Complete or Profound Idiocy," Tredgold[3] writes: "Although these unfortunate creatures are, indeed, the veritable offspring of *Homo sapiens*, the depth of their degeneration is such that existence—for it can hardly be called life—is on a lower plane even than the beasts of the field. . . . They have eyes, but they see not; ears, but they hear not; they have no

[1] The Asylum for Idiots Annual Report, 1857.
[2] The Asylum for Idiots Annual Report, 1859.
[3] Tredgold, A. F., *Mental Deficiency*, 5th ed., p. 156, 1929, Baillière, Tindall & Cox, London.

intelligence and no consciousness of pleasure and pain; in fact, their mental state is one of entire negation. . . . In their life and death are revealed the culminating and final manifestation of the neuropathic diathesis." Although this description only applies strictly to the severest grade of mental defect, the attitude implied is quite typical of views which are widely held at the present time, the word "idiot" having become associated with the ideas of sterilization and the lethal chamber.

This change of attitude seems to be partly due to two factors which are not entirely unconnected. One is the popularization of ideas concerning the influence of heredity or genetic determinants upon diseases and defects, and the other is the growth of an agnostic materialistic outlook. To-day addresses such as those quoted above would only appeal to a very restricted proportion of the population. Most people would rather keep their money and have the idiots removed from the face of the earth. The popular attitude has also been influenced by the failure of the early hopes of curing idiots by training. While it became clear after some years of experience that training could provide no complete cure and would, in many cases, produce only a very slight improvement, this, in itself, was considered worth while. Such a view was held, for example, by Montessori and her followers. The modern western world is, however, becoming intolerant of improvements of this kind, and impatiently demands the elimination of causes and the removal of effects.

Not only has the general attitude towards mental deficiency changed in the course of a century, but there has been a parallel alteration in the concept itself. In the earliest times, only the "Idiot" was referred to and the term connoted what we now regard as idiots and imbeciles together with a certain number of feeble-minded persons. At Earlswood, in fact, five of the first cases admitted were, at the end of their period of training, engaged as servants in the institution. The division of defectives into three classes was first proposed by Duncan[1]. He wrote as follows.

[1] Duncan, P. M., Report, Eastern Counties' Asylum, 1860.

" For all practical purposes, the objects of your Charity may be divided into three classes,—simpletons, imbeciles, and idiots. The first are those feeble-minded, who have not been able to receive instruction in the ordinary manner, who do not possess the experience in life peculiar to those of their age in their social position, and who are said to be " dolts," " stupids," and " fools," by the uncharitable. They have nearly all the faculties to a certain degree, but indicate their alliance to the true idiot by their physiological deficiencies and general inertia of mind. They are to be distinguished from the backward and ill-taught."

The use of the word " simpleton " was fortunate and it is a pity that, at a later date, the double word " feeble-minded " should have replaced it in English legislation, to be followed, in America, by a third term for the same class. The expression " moron," devised by Goddard, has, however, not come into official use in England and it is still reasonable to suggest a return to the use of the word simpleton, which gives an excellent description of members of the class.

The contribution of American workers to the study of mental deficiency has influenced its treatment in several ways. One of the most important developments has been the extensive use of intelligence tests. Before 1900 psychometrical measurements were advocated by Gilbert,[1] Cattell [2] and others. The results of sensory discrimination, strength of grip, coordination and reaction time experiments were shown to be related in some degree to scholastic attainments. In 1905 Binet [3] published his tests and these superseded rapidly many of those formerly used which did not appear to have much relation to intelligence. They gave more attention to strictly psychological abilities, such as memory and simple reasoning. While such tests as these may be said to have originated in France,

[1] Gilbert, J. A., Researches on the Mental and Physical Development of Schoolchildren. *Studies from the Yale Psychological Laboratory*, 1894, Vol. II, pp. 40–100.

[2] Cattell, J. McK., Mental Tests and Measurements. *Mind*, 1890, Vol. XV, pp. 373–80.

[3] Binet, A. and Simon, Th., *L'Année Psychologique*, t. XI, pp. 191-244.

the standardization of methods of scoring for normal people,[1] which is essential for their general applicability, was carried out mainly in America, where facilities for studying both schoolchildren and adults were greater than in other countries.

It is probable that the scientific importance of these methods of testing mental ability as opposed to the earlier perceptual tests has been overrated, since they all involve some degree of educational proficiency on the part of the subject. It is easy to take a class of people differently educated and show that they do not do so well in the tests as the class for which the tests were originally designed. For example, a very large percentage of ordinary soldiers in the American army during the war were found to be morons, *i.e.* feeble-minded, as judged by the Army Alpha tests. For similar reasons, " performance " tests, the results of which are to a great extent independent of education but which require attention, co-ordination and accurate perception, are often preferred, especially when dealing with persons of apparently subnormal mental ability.

At the end of last century, popular interest in the subject was gradually increasing, and many voluntary special schools were started for defective children. Special schools were instituted in England by law in 1896. Interest was further stimulated by the introduction of intelligence tests. The new interest evoked was, however, different in character from the old philanthropic outlook and was coincident in America with the beginning of systematic genetic studies.

The advent of mental tests altered the popular conception of mental defectives. The group now included all who failed to satisfy a certain standard of performance in intelligence tests. This new grouping formed a class of vast and dangerous dimensions and, since it had already been shown that improvement on a large scale was not to be looked for, there was nothing to be done but to blame heredity and advocate methods of extinction. Despite the opinions of critics who have cast

[1] Terman, L. M., *The Measurement of Intelligence*, 1919, Harrap, London.

doubts on their views,[1],[2] the influence of the studies of Davenport [3] and of Goddard [4] both among medical men and among laymen has been such that they are largely responsible for the beliefs prevailing to-day. The most widely read authority in England, Tredgold, does not agree with these views upon the *mechanism* of inheritance, but his general attitude is similar. As early as 1908, in the Royal Commission Report, he had expressed himself as follows: "In 90 per cent. of patients suffering from mental defect, the condition is the result of a morbid state of the ancestors which so impairs the vital powers of the embryo that full and perfect development cannot take place." Public opinion, medical and lay, on this subject, both in England and America, still ascribes nearly all mental deficiency to hereditary causes, even though it is sometimes admitted that the manner of inheritance is not known.

Legal Distinction between Different Types of Mental Abnormality.—One of the chief reasons for the prevalence of modern dogmatic views, as opposed to the scepticism of the middle of last century, is that there have been alterations in the legal provisions for persons of the mentally defective class. While idiots, as defined by the original Idiots Act, were easily distinguishable from their fellows, the new laws brought in a class of defectives who were, for the most part, physically indistinguishable from the rest of the population. As no obvious medical reason could be found for their alleged pathological mental condition, heredity was blamed. This was largely a cloak for ignorance. The scientific insight of legislators would have been remarkable if they had been able to define legally a concept of precise genetic significance. That it should be generally accepted that they have done so seems all the more extraordinary when we study closely the legal definitions of mental defectives used in this country.

[1] Heron, D., *Questions of the Day and of the Fray*, VII, 1913.

[2] Hogben, L., *The Nature of Living Matter*, 1930, pp. 208–11, Kegan Paul, London.

[3] Davenport, C. B., *Heredity in Relation to Eugenics*, 1911, Holt.

[4] Goddard, H. H., *Feeble-mindedness, Its Causes and Consequences*, 1914, The Macmillan Co., New York; *The Kallikak Family*, 1912, The Macmillan Co., New York.

In or about the reign of Edward I a distinction was for the first time made between the 'born fool' or idiot (*fatuus naturalis*) and the lunatic or person who 'hath had understanding, but by disease, grief, or other accident, hath lost the use of his reason.' In the Statute of Prerogatives in the reign of Edward II, a similar distinction was made between the 'born fool' and the person unsound in mind with certain intervals of understanding (*non compos mentis, sicut quidam sunt per lucida intervalla*).[1]

Unfortunately, this simple distinction between idiots and lunatics, recognized by early laws, became clouded in obscurity in later centuries. No distinction was made between persons suffering from amentia [2] and mental diseases or disorders in the Poor Law Amendment Act of 1868, which conferred on Guardians certain powers applying equally to idiots, imbeciles and insane paupers, nor in the Lunacy Acts of 1890 and 1891 (which are still in operation), under which not only lunatics, but idiots and even imbeciles and feeble-minded persons, if certified as of "unsound mind," may be sent to asylums, registered hospitals, licensed houses and Poor Law Institutions. It was often a matter of convenience into which category a given case went. For example, a pregnant woman was admitted into a certain Public Assistance Institution. She had already given birth to one illegitimate child but was certified under the Lunacy Act as an idiot, her description being, "she answers questions satisfactorily, but is mentally and morally weak: . . . would not wash herself unless forced to." This was in 1910, and certificates of this kind are sometimes to be found still in force.

The first clear distinction in modern legislation between lunatics on the one hand and idiots and imbeciles on the other is to be found in the Idiots Act of 1886, but this distinction differs from that of the earlier laws. This Act provides that one who is an idiot or imbecile from birth or from an early age may be placed by his parent or guardians in any registered hospital or institution for the care, education and training of idiots or imbeciles.

[1] This legal retrospect follows closely that given in the Wood Report.

[2] "Amentia" is synonymous with "mental deficiency."

Mental Deficiency Acts.—The Mental Deficiency Act of 1913[1] was the outcome of the recommendations of a Royal Commission which was appointed in 1904 " to consider the existing methods of dealing with idiots and epileptics, and with imbeciles, feeble-minded or defective persons not certified under the Lunacy Laws." After four years (1908) the commission reported that a grave state of affairs existed in the country. There were present in the community large numbers of mentally defective persons whose training was neglected, and over whom insufficient control was exercised. Many were committed to prisons for repeated offences, many who did not require careful hospital treatment were to be found crowding the lunatic asylums, and also many were at large, both adults and children, who, in one way or another, were incapable of self-control and therefore exposed to constant moral danger. The commission recommended the creation of a system whereby these mentally defective persons could at an early age be brought into touch with some friendly authority, trained as far as need be, supervised during their lives in cooperation with their relatives, or detained and treated in some measure as wards of the State. An Act was passed in 1913 embodying the main recommendations of the commission. Definitions were laid down, which were slightly amended in 1927, and which are in force at the present time.

1. Idiots, that is to say, persons in whose case there exists mental defectiveness of such a degree that they are unable to guard themselves against common physical dangers.

2. Imbeciles, that is to say, persons in whose case there exists mental defectiveness which, though not amounting to idiocy, is yet so pronounced that they are incapable of managing themselves or their affairs or, in the case of children, of being taught to do so.

3. Feeble-minded persons, that is to say, persons in whose case there exists mental defectiveness which, though not amounting to imbecility, is yet so pronounced that they require

[1] Davey, H., *The Law Relating to the Mentally Defective*, 2nd ed., 1914, Stevens & Sons, Ltd., London.

care, supervision and control for their own protection or for the protection of others or, in the case of children, that they appear to be permanently incapable by reason of such defectiveness of receiving proper benefit from the instruction in ordinary schools.

4. Moral defectives, that is to say, persons in whose case there exists mental defectiveness coupled with strongly vicious or criminal propensities, and who require care, supervision and control for the protection of others.

The Act further stipulates that " mental defectiveness " here means a condition of arrested or incomplete development of mind existing before the age of eighteen years, whether arising from inherent causes or induced by disease or injury.

It will be seen at once that these definitions are framed for practical administrative purposes and make no pretence at scientific usefulness either from the point of view of human psychology or biology. They must be construed in the light of the relative importance of the deficiency of the person under consideration as compared with the normal. The definition of idiots does not imply that a person who is injured in the street by a motor vehicle necessarily belongs to this class. Falling into the fire or starvation are the types of accident envisaged.

The definition of imbeciles also has peculiarities. Ability to manage one's own affairs is partly a matter of intelligence, but it also depends upon general knowledge and on temperament. And, were it not for the fact that the feeble-minded have their own definition, this description of imbeciles would cover them also. Conversely, the definition of the feeble-minded would cover imbeciles were the latter not expressly excluded.

There is one very important point in regard to the legal status of the feeble-minded. The usual interpretation of the Acts is that no adult mentally defective person can be certified in this class unless circumstances can be shown to exist combining the need for the " care, supervision, and control " with insufficient protection of themselves or of others. Hence those adult mental defectives who are actually certified feeble-

minded are far more likely to be drawn from the poorer social classes where supervision and protection are more difficult to provide.

There are special provisions for feeble-minded children under the Education Acts. Here, again, it is unlikely that children from richer social classes will be certified, since the education authority is not so likely to trouble about cases which do not attend the ordinary schools.

The legal class of *moral* defectives is of purely administrative value, framed in order to make it easier to certify persons of only slightly subnormal intelligence who have anti-social tendencies. The curious nomenclature is liable to suggest to the lay mind that this class of defective is *better* behaved than other classes instead of worse behaved. It was believed, by the commissioners of 1904, that delinquency in respect of the prevailing social code might frequently be due to specific deficiency in moral sense, although the intelligence might be relatively unimpaired. In practice the category is used for detaining those persons who have intelligence quotients within normal limits, *i.e.* 70 to 100, but who persistently offend against the law. Many psychologists and psychiatrists, at the present time, do not agree with the implications of the category of moral deficiency. There is an increasing tendency to regard criminal offences, when they are not due to ignorance or stupidity, as indications of mental disorder. In certain cases of delinquency it has been shown to be probable that desire for punishment may actually be an important motive.[1],[2] When this is so, punitive measures cannot be expected to have any deterrent effect.

Slight emendations have been made since 1913 to facilitate administration. A full account of the working of the Acts has been published recently by Shrubsall and Williams.[3]

[1] Alexander, F., Mental Hygiene and Criminology. *Mental Hygiene*, Vol. XIV, p. 853, 1930.

[2] Alexander, F. and Staub, H., *Der Verbrecher und seine Richter*, 1929, Internationaler Psychoanalytischer Verlag.

[3] Shrubsall, F. C., and Williams, A. C., *Mental Deficiency Practice*, 1932, University of London Press.

While the Board of Control are responsible for all persons certified under the Mental Deficiency Act, mentally defective children, between the ages of 7 and 16, are the responsibility of the Education Authorities. The children were formerly dealt with optionally under the Elementary Education Act of 1899. This Act was repealed in 1921, and a new Act was passed providing for the establishment of special schools where these children may learn useful handicrafts and receive more elementary instruction than in the primary schools. Some people consider it unfortunate that instruction in a special school carries with it the stigma of mental deficiency, since dull children (I.Q. 70 to 85) might gain much if the scope of special schools could be widened.[1]

It is obvious that the closest cooperation is necessary between the Board of Education and the Board of Control on problems of mental deficiency, and one of the most important fruits of this cooperation was the Report of the Joint Committee of the Board of Education and the Board of Control, published in 1929, known as the Wood Report. This committee was appointed in 1924 and its report includes an account of the survey, made by Lewis, of the incidence of mental defectives (within the meaning of the Act) in six different areas in England, three rural and three urban. The estimated average incidence was 8·57 per 1,000 individuals in the general population, the frequency being significantly higher in the rural than in the urban areas. The relative proportions of the number of idiots, imbeciles and feeble-minded ascertained were, roughly, as 5 : 20 : 75. A very complete record was given of the proportions of defectives actually dealt with in various ways—in certified institutions, under guardianship, in Poor Law Institutions, etc. The attitude of the Committee, which represents the most authoritative opinion up to the present time, to the problem of mental deficiency is summed up clearly in the following words :

In the light of all the definitions contained in the Mental Deficiency Acts and of the best scientific opinion, we have taken the view that,

[1] Report of the Consultative Committee on the Primary School, Board of Education, 1931, H.M. Stationery Office.

whatever may be the correct legal interpretation of these definitions, the real criterion of mental deficiency is a social one, and that a mentally defective individual, whether child or adult, is one who by reason of incomplete mental development is incapable of independent social adaptation.

Thus mental defect is fundamentally a social, and not simply a biological or psychological, problem. It is the social and economic aspect which is liable to obscure all others and public opinion varies in its attitude towards mental defect precisely in accordance with its attitude towards other social and economic problems. To investigate the subject satisfactorily from the biological or psychological point of view we must throw aside all social and moral prejudices. In order to solve the problems of the most satisfactory way of dealing with mental defectives, we must also take into full consideration the effects of the present economic conditions and the complex nature of modern civilization.

CHAPTER II

SYSTEMATIC INVESTIGATION : GENERAL PHYSICAL EXAMINATION

Introductory—Nervous System—Vascular and Respiratory Systems —Alimentary System—Sexual Organs—Endocrine System—Skeletal System—Stigmata.

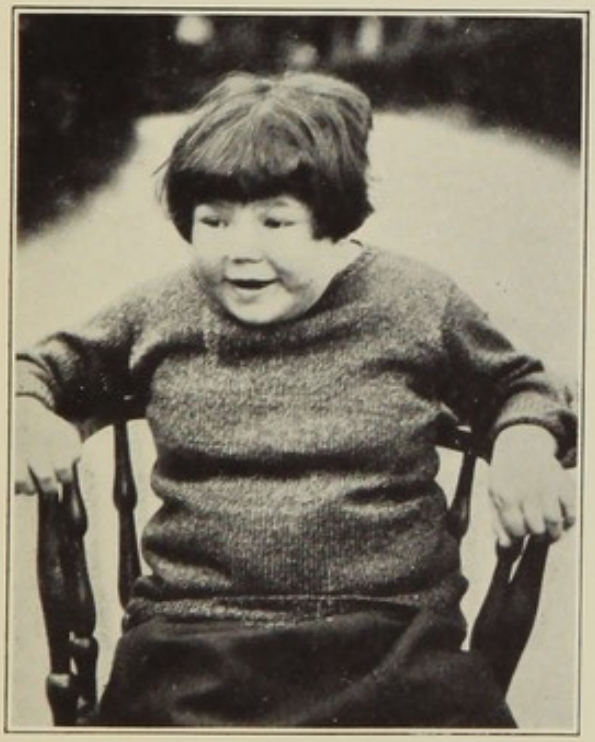
Introductory.—We shall begin the study of mental deficiency by considering the methods available to the investigator. The subject is one in which little headway can be made without a knowledge of medicine, and a physical medical examination of the patient must take precedence over all other investigations. The purely medical part of the investigation will therefore be discussed at once, but as briefly as possible.

In the physical examination of a mentally defective subject there are many things to be noticed which ordinarily it would be considered superfluous to record. On the other hand, certain parts of the routine physical examination are rendered unsatisfactory, especially if the patient is of very low grade.[1] For instance, existing tests of sensation or sense discrimination become impossible at these levels, and examination of the respiratory system is difficult. While the ascertainment of the general state of health of the patient is interesting and useful for administrative purposes, its value in forwarding knowledge of the causes of mental deficiency is limited. The observations which are strictly relevant to this aim are those which give information concerning the pathological conditions associated more frequently with mental deficiency than with normal mentality. Special attention must therefore be given to those signs and symptoms which are supposed to have or actually do have associations of this kind.

[1] *High grade* and *low grade* are used in mental deficiency to mean *mild* and *severe* respectively, and not, as sometimes supposed, in the opposite but perhaps more natural sense.

Imbecile : Mongolism. Male aged 19.
(See p. 96.)

Idiot : No Special Type. Male aged 22.
(See p. 87.)



Idiot : Cretin. Female aged 8.
(See p. 129.)

Imbecile : Pituitary Dystrophy.
Male aged 5. (See p. 132.)

[*To face p.* 14

Nervous System.—Naturally one of the most important examinations to be made in every case is a thorough investigation of the central nervous system, including ophthalmoscopy. Like everything else connected with mental deficiency, the examination is not always a straightforward affair. The majority of high grade defectives, who can cooperate in such an examination, have clinically normal nervous systems, and cases of neurological interest are mostly to be found among the lower grades where cooperation is often impossible. The testing of sensation is of very doubtful diagnostic value from the neurological point of view in mental deficiency although it is of interest psychologically, sensibility to pain (both deep and superficial) being often very much blunted. Deafness is exceedingly difficult to estimate, especially in low grade patients. It is safest to rely on objective signs, such as deep reflexes, which may be brisk.

Neurological lesions associated with mental deficiency are usually of very long standing and may have occurred very early in life, even before birth. Paralytic symptoms are very common and may be due to traumatic, inflammatory or degenerative processes. The resulting clinical picture can be much distorted by subsequent developmental changes, so that only the most competent neurologist is able to make differential diagnoses.

The results obtained from ophthalmological examination are, on the other hand, often very encouraging, and may give us trustworthy evidence on which to make a diagnosis. The clouding of the cornea, characteristic of congenital syphilis (interstitial keratitis), is sometimes the sole clinical manifestation of the disease. Inflammatory and degenerative conditions of the retina (choroiditis, retinitis) and the condition of the optic nerve sometimes give evidence of syphilis, but may sometimes indicate the presence of inherited defects. Especially interesting is the occurrence of progressive optic degeneration (retinitis pigmentosa) giving rise to a wax-like appearance of the retina, which is seen in certain rare cases of mental deficiency. Congenital cataract is supposed to be frequent in mongolism, but it may be found in defectives of

other types. The presence of developmental anomalies of the eye (colobomata) may suggest that the brain, with which the eye is intimately associated, is also affected developmentally. Retinal tumours are seen in rare cases.

Vascular and Respiratory Systems.—The cutaneous vascular system sometimes shows significant peculiarities. Abnormal developments of blood vessels, causing the familiar " port wine " stains or other types of nævi, are occasionally found in association with impaired mental powers. This association has been named *nævoid amentia*, but the term is not a very exact one, since a nævus is not of ætiological importance unless it extends to the deep structures of the nervous system and is associated with brain deformity. The dilated capillaries sometimes seen on the cheeks of young aments are supposed to be indicative of congenital syphilis, and anomalous end-capillaries, concerning which more will be said shortly, are associated with endocrine disorder. Vasomotor abnormalities, due to paralysis of the nervous supply of the blood vessels, are frequently seen in neurological cases, and care has to be taken to avoid such patients suffering severely from chilblains.

Congenital heart lesions should be looked for, though they are by no means particularly common among mental defectives. Some authors have stated that a particular developmental abnormality of the heart (incomplete septum between the ventricles) is very frequently found in mongolism, but the present writer has not found congenital heart lesions at all common even in the mongolian type. The circulation in these cases is poor and their extremities are blue and cold, but this is possibly due to a small heart with flabby musculature. The pulse rate in these and also other cases has been asserted to be slower than normal, but it must be remembered that the patients are often not at all active. Some observers have, in fact, attempted to correlate pulse rate with intelligence without any very marked success.[1] Others have attempted to find out whether the oral temperatures of idiots

[1] Whiting, M. H., A Study in Criminal Anthropometry. *Biometrika*, Vol. XI, 1915, p. 1.

are lower than normal with inconclusive results.[1] Respiration rate has also been studied in the same connection, and Pearson and Goring found correlations of the order of $-0{\cdot}17 \pm 0{\cdot}03$ for intelligence and respiration rate, working with students and criminals respectively.[2]

Although the complete examination of the lungs in aments is subject to difficulties owing to their imperfect cooperation, from the administrative point of view it is very important. In view of the former prevalence of tuberculosis in institutions and the supposedly high susceptibility of aments to the disease, the usual pulmonary examinations of patients are now supplemented by a monthly weight chart for each of the patients. According to Turner the introduction of this routine in 1905, together with attention to diet and ventilation, was responsible for a dramatic reduction in the tuberculosis death rate of the patients in the Royal Eastern Counties Institution.

AVERAGE DEATH RATE OF MENTAL DEFECTIVES FROM ALL FORMS OF TUBERCULOSIS.

1859 to 1905	46·0 per thousand.
1906 to 1925	12·9 per thousand.
1926 to 1932	4·8 per thousand.

When we bear in mind that these figures will probably be still further reduced in future we are left in doubt whether there is any real evidence that mental defectives in general are particularly liable to pulmonary tuberculosis. It remains quite likely that special types, such as mongols and congenital syphilitics, are more prone than the general population to tuberculous infection. The prevalence of tuberculosis, found by some observers, among mental defectives of no special type and among their relatives, may be due to the habits and mode of life of these people and of their families.

Alimentary System.—The clinical examination of the alimentary system is of interest mainly on account of the condition of the mouth which, in congenital syphilis and in

[1] Norsworthy, N., Psychology of Mentally Defective Children. *Arch. Psychology* I, Nov., 1906.

[2] Pearson, K., On our Present Knowledge of the Relationship of Mind and Body. *Annals of Eugenics*, Vol. I, 1925–26, p. 382.

mongolism, may present characteristic features. The diagnostic value of palatal abnormalities has probably, in the past, been overrated. Cleft palate is not commonly found in mental defectives and the high and narrow palate characteristic of many low grade patients must be considered in relation to the opportunities the palate has had for development. Leaving aside the rare cases where there is gross deformity of the cranium, mouth breathing and imperfect use of the lips and jaws for feeding may be important in determining the shape of the palate. Among higher grades, as shown by Channing and Wissler, no significant deviation of this structure from the normal is to be found. Other points of special interest in the alimentary system are the morphology of the tongue, the presence or absence of umbilical hernia and the disposition of fat on the abdomen. Mucous diarrhœa is a common complaint among mongols. In institutions for the defective, as in hospitals for the insane, care has to be taken to avoid epidemics of dysentery spread by patients whose habits are not clean.

Sexual Organs.—The development of the genitals is an important feature to record in any case of amentia. It is sometimes surprising how complete is the sexual development of even the lowest grade defectives, both males and females, if we rule out those cases with obvious endocrine disorder (*e.g.* cretins). The great majority of all female mental defectives menstruate with regularity and this commences at the normal time : the secondary sexual characters are usually developed well in both sexes, though sometimes a little late. But sexual desire, that is to say, an object-love which must be satisfied in other ways than by masturbation, seems to be lacking in idiots and low grade imbeciles : in fact, the intensity of sexual desire seems to be roughly in proportion to the intelligence quotient.

Endocrine System.—The search for evidence of endocrine dystrophy among mental defectives frequently reveals many mild degrees of disorder. Besides these there are the comparatively rare examples of a clear clinical picture of cretinism or pathological obesity with infantilism (dystrophia-adiposo-

genitalis). Cases of mixed endocrine dystrophy are also to be found, such as gigantism with adiposity, and in mongolian and other dwarfs features of cretinism may be present. Of great interest in the study of all these types is the special examination of cutaneous capillaries, which Jaensch has shown to be imperfectly developed not only in cretins but also in cases where endocrine dystrophy is outwardly not very marked.[1] The capillaries studied are those in the fold of skin covering the nail bed and they can easily be seen with the low power of a microscope in bright daylight if a drop of oil of cedar wood or cloves is placed on the skin above them. The fourth digit gives the easiest view, but since one finger may show deformed capillaries and another not, it is well to compare several fingers with one another in the same subject. Jaensch himself claims that perversion of pituitary secretion is the basis of the failure in development of these capillaries and hypothetically connects the condition with disorder of function in the mid-brain.

Skeletal System.—Dwarfs of other types than cretins, such as the ateleiotic and achondroplasic, are sometimes observed among the mentally defective. Ateleiosis, or smallness of body without disturbance of proportion, is the more commonly observed of the two conditions. Their connection with endocrine disorder is at present unknown, but one may often here suspect some anomaly of internal secretion. The true types of dwarfism have to be carefully distinguished from rachitic and renal dwarfs, and congenital syphilis has always to be looked for in disorders of the skeletal system where no obvious cause is discoverable. Deformities affecting the skeletal system, other than those directly attributable to infection, nutrition or paralysis, are the degenerative processes such as the muscular dystrophies and their occasional occurrence among mental defectives must not be overlooked. Muscular dystrophies can be classed with certain other rare conditions of hereditary origin like amaurotic family iodiocy and Friedreich's ataxia. Imperfect development of the parts of the skull and collar bone (cleidocranial dysostosis),

[1] Jaensch, W., *Hb. der biolog. Arbeitsmethoden*, Abt. IX, Teil 3, pp. 865–940, 1930, Berlin.

abnormally fragile bones and split-hand are hereditary developmental abnormalities affecting the skeletal system more directly, but these are very rarely found associated with mental deficiency. Milder types of deformity like syndactyly, polydactyly, contracted palmar fascia and club foot (when not due to paralysis) do, however, appear sometimes to be connected with mental deficiency; but whether they really are more frequently met with than would be expected from a random distribution of these defects in the general population has not yet been demonstrated.

Stigmata.—Among the signs which are to be found among aments are the so-called "stigmata of degeneration," popularized by Lombroso on the Continent and given great prominence in the writings of alienists during the last few decades. These stigmata are physical signs such as high and narrow palate, short minimal digit and malformed ears. They have been so fully portrayed in current textbooks that their description here would be superfluous. It was formerly considered that the presence of such signs as these were indicative of hereditary mental degeneracy, but the belief, for which no satisfactory evidence has ever been produced, is now losing its popularity. Degenerate ears, palate or hands are so common among persons who wish to be considered normal, that the association of these signs with mental impairment or disorder is, at best, a matter involving statistical differences in the frequencies with which they occur in different groups. There is, in fact, very little difference between the mentally normal and the mentally defective as regards the frequency of the presence or absence of these stigmata, except in so far as certain low grades and special types of defectives are concerned.[1],[2] To the modern human biologist, the interest of the various stigmata becomes identical with the interest of other polymorphic human characteristics, like eye or hair colour. It remains to be discovered which characters represent genetic variants and with what other

[1] Channing, W. and Wissler, C., Comparative Measurements of the Hard Palate in Normal and Feeble-minded. *Amer. Journ. Insan.*, 1905, Vol. LXI, p. 687.

[2] Burke, N. H. M., Stigmata of Degeneration in Relation to Mental Deficiency. *Proc. Roy. Soc. Med.*, Feb., 1931, Vol. XXIV, pp. 413–28.

characters they may be genetically linked. In view of the possibility of discovering such linkage, it may be useful to note the presence or absence of the stigmata of degeneration, together with such things as hair and eye colour, in every patient and in all their available near relatives. Peculiarities in the texture and colour of hair and skin may also serve this purpose. But even if mutual associations are discovered between any of these characters, it is difficult to evaluate the importance of the finding unless we know already how the individual characters are inherited. A survey of the frequency of incidence of such characters can, however, be used in defining different types of mental defectives. This use will be demonstrated when we come to consider mongolism (p. 100).

Many physical characters for which man is polymorphic are extremely difficult to observe. For example, though eye colour can be classified roughly under three headings, blue, brown and intermediate, when viewed minutely it is found that there are scores of different types among these three classes. Eye colour alters with age and health. Moreover, two observers may classify the same eye under quite different headings, according to their ability to match colours and appreciate details of form. In fact, the study of the inheritance of eye colour, which originally appeared to be a very easy problem in human genetics,[1] has since been shown to be extremely difficult and complex.[2] Similar difficulties apply to the study of types of ears and the relative lengths and shapes of fingers.

When we come to purely metrical characters, some, at least, of these difficulties can be avoided. Methods of measurement can be properly standardized and the errors of technique can be estimated with precision. It is to the metrical characters, such as head measurements, height and weight, that our attention here will first be directed. Afterwards we shall consider biochemical and pathological examinations which have similar advantages over less exact clinical criteria. Then we shall have to consider the somewhat less precise methods of mental measurement.

[1] Hurst, C. C., On the Inheritance of Eye-colour in Man. *Proc. Roy. Soc.*, Vol. LXXX, 1908.

[2] Galloway, A. R., Notes on the Pigmentation of the Human Iris. *Biometrika*, Vol. VIII, 1911–12, p. 267.

CHAPTER III

SYSTEMATIC INVESTIGATION : PHYSICAL MEASUREMENTS

Head Circumference—Cephalic Index—Cranial Capacity—Head Size and Intelligence—Diagnostic Value of Head Size—Head Size and Stature—Physique of the Mentally Defective—The "Physical Quotient"—Significance of the Relation of Bodily Development to Intelligence.

Head Circumference.—The simplest method of ascertaining the size of the head is to find its circumference with a tape measure. For this purpose usually the greatest circumference is taken. It is found by adjusting the tape round the frontal part of the head and carrying it back round the region of the occipital process. This circumference, in a normal adult, measures about 22 inches and a reading below 17 inches is regarded as indicating microcephaly. It has never been made clear whether any correction for age is to be made in assessing the diagnostic importance of the reading below 17 inches, and this largely destroys its value since very few adult microcephalics have heads whose measurement is less than this figure. Moreover, there is no satisfactory standardization of the measurement for normal males and females of different ages and, without a clear idea of the normal variability, its diagnostic value is bound to be very small. To those who are anxious to make careful investigations, the measurement of length and breadth appeals more strongly, and a good idea of the maximum circumference can be obtained by averaging these two figures and multiplying by π.

Cephalic Index.—The length of the head is defined as the distance between the point in the mid line between—and on a level with—the orbital ridges and the most prominent point on the occiput.[1] The breadth is defined as the widest diameter

[1] Occasionally this point coincides with the occipital protuberance in dolichocephalic heads, as it does in the gorilla.

at right angles to the sagittal plane above the level of the auditory meatus.[1] From these two measurements we can obtain the breadth-length ratio or cephalic index,[2] which is of considerable interest in the study of mental defect. A microcephalic head, by the definition aforesaid, may have an entirely different shape according to whether it is long and narrow or short and round. The head which is characteristic of microcephaly, as described by most authorities,[3] is long and narrow or dolichocephalic. The small round head with flattened occiput is characteristic of young mongolian imbeciles and also of certain rarer types with peculiar head shapes where there is a definite flattening above the occipital region on palpation.

The cephalic index varies with age, sex, stature and race, as well as with clinical type, and these factors have to be given full consideration when it is being used for diagnosis. The cephalic index of females is higher than that of males as a general rule, partly owing to the greater length of the male head, to which strongly developed brow ridges contribute. This difference may be connected with males' being rather taller than females; for even among members of the sexes taken separately there is a tendency for the index to be negatively correlated with stature.[4] The following table shows the effect of age on cephalic index in defectives of the mongolian type.

Mongolian Type of Defective (Males).

Age in years.	No. of cases.	Average Breadth.	Average Length.	Cephalic Index.
0–4	8	130·7	150·6	0·869
5–9	17	136·6	159·0	0·848
10–14	27	138·4	167·4	0·826
15–19	14	141·2	170·1	0·828
20–24	18	141·2	174·9	0·807
25 and over	13	141·2	174·1	0·810

[1] Keith, A., *Human Embryology and Morphology*, 1921, Arnold, London.

[2] Sometimes known as "first cephalic index."

[3] See, for example, Shuttleworth, G. E., *Mentally Deficient Children*, 1st ed., 1895, H. K. Lewis, London.

[4] Reid, R. W. and Mulligan, J. H., Relation between Stature, Head Length and Head Breadth of 847 Natives of the North-East of Scotland. *Journ. Roy. Anthrop. Inst.*, Vol. LIV, p. 287–299, 1925.

The effect is marked here, but a similar, though slight, effect is noticeable among normals also.[1]

It has been claimed that it is possible to alter cephalic index to a significant degree by laying a baby, shortly after birth, on its side or its back.[2] The results of this procedure must be read in the light of the knowledge that, in early life, cephalic index decreases to some extent with age in any case, and may alter more in certain types of heads than in others. Moreover, it is extremely difficult to measure accurately a young infant's head, and, as the initial measurements are small, the error of their ratio is likely to be considerable. It is essential to measure in units at least as small as one millimetre in order that the resulting ratio may be significant to more than one figure. It is impossible, without specially contrived apparatus, to measure the head with accuracy greater than to the nearest millimetre, and the third figure in the cephalic index has no significance except in averages. The simplest method of measuring these two diameters is to use plain metal calipers, which are afterwards compared with a ruler. The points should be rounded off smoothly so that they do not scratch. The method adopted should be as comfortable as possible for the subject, if only because normal controls are more likely to object to being pinched tightly than defectives and this will tend to make the normal head measurements appear too big. Graduated calipers, such as are in common use by obstetricians for pelvimetry, are far too inaccurate for head measurement. The average of three measurements, taken at one sitting or on different days, is preferable to a single observation.

Cranial Capacity.—A good practical estimate of the cranial capacity can be obtained from the sum of the two measurements of length and breadth. While it is customary, at all events with adults, to make use of Lee's formulae for general use,[3]

[1] Pearson, K. and Tippett, L. H. C., On the Stability of the Cephalic Indices within the Race. *Biometrika*, Vol. XVI, pp. 118–38.

[2] Basler, A., Über den Einfluss der Lagerung von Säuglingen auf die bleibende Schädelform. *Zeits. f. Morph. u. Anthrop.*, Vol. XXVI, pp. 225–46, 1927.

[3] Lee, A., *Proc. Roy. Soc. Trans.*, Vol. CXCVIA, p. 230.
Males : $(l-11).(b-11).(h-11)\times 0{\cdot}000337+406{\cdot}01$.
Females : $(l-11).(b-11).(h-11)\times 0{\cdot}000400+206{\cdot}60$.

to do so involves measuring the auricular height of the head. This procedure is only possible with some form of craniometer, such as Gray's. The instrument is cumbersome, liable to certain inaccuracies, and children often find its application irksome. Moreover, Lee's formulae are meant to apply to adults and have no precise meaning when used for children. The present writer found the correlation between cranial capacity as calculated by Lee's formula and the sum of the length and the breadth of the head in 100 adult male mental defectives of very mixed types to be $0{\cdot}91 \pm 0{\cdot}02$; the correlation for a similar group of females was $0{\cdot}93 \pm 0{\cdot}02$.[1] In Lee's formula the three head diameters are multiplied together, after being corrected for thickness of skull and scalp by subtracting 11 mm. from each. The vertical measurement has more effect than the other two on the final result because it is the smallest. The vertical measurement is also the most difficult to take and most liable to error. In view of the closeness of its proportional agreement generally with the sum of the other two diameters, it may be omitted where head size and cephalic index are the only quantities which it is required to estimate.

Head Size and Intelligence.—It would be interesting and useful if it could be shown that any close relation existed between head size and intelligence. The relatively large size of the brain in man as compared with other animals is mostly due to the superior development of his cerebral cortex : it is probably to this accumulation of grey matter that he owes his superior intelligence. It is natural enough to enquire whether some relation may not hold between size of the head—which is largely determined by the size of the brain—and intelligence in members of the same species. In the case of man, males and females have to be treated separately, otherwise we should

[1] A rough formula for general use, with both male and female adults, connecting the sum of the two measurements with capacity, is as follows :—

$$B = 10A - 2{,}000$$

where A = length + breadth (mm.), and

B = cranial capacity (cc.)

Brain weight (gms.) = (approximately) $9/10 \times$ capacity (cc.).

start with the *a priori* fallacy that males are more intelligent than females. Similar *a priori* reasoning has led some observers to suppose that, if the brains of negroes can be shown to be smaller than those of Europeans, the negroes must necessarily be less intelligent.[1] Without full opportunities for education and comparison on the basis of the application of standardized tests of wide ranges, no valid conclusions can be drawn concerning intelligence in different races.

A serious attempt has been made by Pearson to discover a connection between head size and intelligence within a homogeneous group. In his first study [2] on this subject he found only insignificant or very small correlations which, he pointed out, might be due to selection of older persons (with larger heads) who were the most intelligent in his group. Later he examined schoolchildren as well as university graduates [3] and the result was to demonstrate a weak, but positive and significant, relationship between head size and intelligence. Head size was judged by separate sets of measurements of length and breadth. Intelligence was measured, in the case of schoolchildren, by teachers' estimates and, in the case of graduates, by examination results. The correlations obtained were as follows :—

	Boys.	Girls.	Cambridge graduates (Male).
Intelligence and length of head	+0·139	+0·084	+0·111
Intelligence and breadth of head	+0·109	+0·113	+0·097

The standard deviations of the correlations for the children were of the order of 0·013 to 0·014, and those for the graduates of the order of 0·020 to 0·021. A more recent study by Estabrooks [4] on a group of schoolchildren all aged 6, gives the result of correlating their scores in various intelligence tests

[1] Oliver, R. A. C., The Comparison of Abilities of Races with special reference to East Africa. *East African Medical Journal*, Sept., 1932.

[2] Pearson, K., On the Correlation of Intellectual Ability with Size and Shape of the Head. *Proc. Roy. Soc.*, Vol. LXIX, p. 333, 1902.

[3] Pearson, K., On our Present Knowledge of the Relationship of Mind and Body, *op cit.*

[4] Estabrooks, G. H., Cranial Capacity and Intelligence. *Journal of Applied Psychology*, Vol. XII, 1928, p. 524.

with their cranial capacities, calculated from Lee's formula. Estabrooks separated out children of different racial origins, but, as each group gave similar results, the final figures for the whole group can be taken as representative.

American Schoolchildren (Estabrooks).

	172 Males.	207 Females.
Correlation coefficients of Scores in Intelligence tests (Stanford-Binet, Otis and Dearborn) and Cranial Capacity.	+0·23 to +0·31	+0·08 to +0·16

Most of the correlations were significant, and they were on the whole higher than those found by Pearson. There may be two reasons for this. Firstly, Estabrooks uses a variable for head size which is compounded from length, breadth and height, and, however unsuitable Lee's formula may be for application to children, it is likely to give a closer correlation with actual cranial capacity than any single measurement. Secondly, standardized mental tests were used and the scores in these tests, while closely correlated to teachers' estimates or examination results, give a wider range of variability than Pearson's ratings. None of the children were, as far as could be ascertained at the age of 6, mentally defective, and the addition of idiots and imbeciles, so as to include the whole possible range of intelligence, might have given rise to an even higher correlation value. Taking the variable *length plus breadth*, the present writer correlated it with intelligence quotient,[1] as judged by the Stanford-Binet tests, in a series of 131 males of different ages at the Royal Eastern Counties Institution, and the following values were obtained :—

66 males, aged 11 to 14 years . . . $r = +0{\cdot}25 \pm 0{\cdot}08$
65 males, aged 15 to 18 years . . . $r = +0{\cdot}34 \pm 0{\cdot}07$

Further, head length, breadth and height were measured in a series of 100 males over 19 years of age, drawn from the same source. Lee's formula was applied to the measurements and the resulting estimate of the cranial capacity was correlated with mental age : the magnitude of this correlation was

[1] Following Lewis's recommendations, given in the Wood Report, 14 years is used throughout this book as the maximum chronological age for the denominator in determining I.Q.

TABLE A.

CRANIAL CAPACITY AND MENTAL AGE.

100 ADULT MALES.

c.c.	Mental age in years.														Total.
	0	1	2	3	4	5	6	7	8	9	10	11	12	13	
2,000–49									1						1
1,950–99															
1,900–49															
1,850–99															
1,800–49			1												1
1,750–99															
1,700–49			1												1
1,650–99														1	1
1,600–49						1			1						2
1,550–99							1	1		1		1			4
1,500–49		1						1	3	1		1			7
1,450–99				3			1	1	6	2	2	1			16
1,400–49			1	1	1			2	2	1	3	1	1		13
1,350–99		2	1				3	1	2	2	1	1			13
1,300–49	1	1		1	2	1	3		3	2	3				17
1,250–99	1		1	2	1		1	4	1	1					12
1,200–49		1	2				1	1							5
1,150–99	1		1	1						1					4
1,100–49			1	1	1										3
Total . .	3	5	9	9	5	2	10	11	19	11	9	5	1	1	100

+0·29±0·09 (Table A). There were insignificant correlations between age and mental age (+0·09) and between age and cranial capacity (+0·03). In a similar series of 256 females the correlation value was +0·21=0·06 (Table B), and there were insignificant correlations between age and mental age and between age and capacity (+0·01 and +0·00 respectively). The patients, both male and female, were given the Porteus Maze performance test (Vineland Revision) as well as the Stanford-Binet tests. An estimate of mental age was obtained by weighting the Stanford-Binet score three times as heavily as the Porteus score : a justification for this weighting being that the Stanford scale ascends by two-monthly intervals while the Porteus score advances in intervals of six months. The result was to correct the Stanford mental age by as much as one year in 12 per cent. of the cases.

TABLE B.

CRANIAL CAPACITY AND MENTAL AGE.

256 ADULT FEMALES.

c.c.	Mental age in years.															Total.
	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	
2,200–49							1									1
2,150–99																
2,100–49																
2,050–99																
2,000–49																
1,950–99																
1,900–49																
1,850–99																
1,800–49																
1,750–99																
1,700–49																
1,650–99																
1,600–49				1												1
1,550–99																
1,500–49			1									1				2
1,450–99								1	1	1						3
1,400–49		1		1	3		2	1	1	4				1		14
1,350–99			1		1	4	1	3	3	2	3	1				19
1,300–49		1		2	2	2	4	2	7	5	3	2	1			31
1,250–99		1	2	1	3	2	8	8	13	6	5	2	3		1	55
1,200–49		1	1	2	1	1	3	3	4	2	2	1	2	3		26
1,150–99			5	3	2	2		9	7	4	1		3		1	37
1,100–49			2		3	1	3	6	4		2	1				22
1,050–99	1	3	3	3	3	1	5	2	3	1	1					26
1,000–49		3	2	1	1	1			2	1	1					12
950–99						2		1	1							4
900–49	1						1									2
850–99	1															1
Total .	3	10	17	14	19	16	28	36	46	26	18	8	9	4	2	256

These groups of patients were quite unselected for grade and type and included hydrocephalics, microcephalics, mongols as well as psychotics and paralytics. Only the very deaf or blind were excluded because of inability to perform tests. Persons with mental ages below 2 years had to be divided into two classes according to whether they were capable of simple reactions like feeding and walking, or whether they were what Tredgold terms absolute or profound idiots. Any attempt to exclude special types from such an investigation as this confronts us with difficult problems. For example, it might be

argued that hydrocephalics should be excluded because here head size will probably be negatively associated with intelligence. But, if so, macrocephalics should also be excluded for, in this condition, we are dealing with excess not of true brain tissue but of supporting structures. It is, however, not possible by examining the outside of the head to be sure, in any case, even with normal or microcephalic measurements, how much of the bulk is true brain tissue and how much is not. The proportion of the special mongol type, in the age group above 19 years, is so small that if all cases of this type had been excluded it would not have made any important difference. There must be, in any group of unselected patients, a number of other special types which have never yet been described and therefore could not have been excluded. Thus it seemed best to take all and sundry together, and the result is a demonstrable and regular, but slow, rise of head size with intelligence. The picture can be supplemented by the addition of control

TABLE C.

MALES OVER 19 YEARS.

Number.	Description.	I. Q.	Mean cranial capacity.	Standard error.	Coefficient of variation.
24	Idiots	0–24	1334	34	0·126
19	Imbeciles	25–49	1376	44	0·085
48	Feeble-minded	50–74	1412	19	0·091
9	Very high grade feeble-minded	75–100	1482	30	0·061
20	Normal Controls	—	1423	15	0·046

FEMALES OVER 19 YEARS.

Number.	Description.	I.Q.	Mean cranial capacity.	Standard error.	Coefficient of variation.
30	Idiots	0–21	1149	25	0·121
77	Imbeciles	22–49	1238	20	0·140
126	Feeble-minded	50–77	1243	10	0·088
23	Very high grade feeble-minded	78–100	1266	19	0·070
20	Normal Controls	—	1290	21	0·081

groups of normal persons, that is, persons of similar age and social class to the certified defectives, but not suspected of mental deficiency. Such a group is formed by the staff at an institution. The figures in Table C show the steady rise of cranial capacity with intelligence among the patients.

Diagnostic Value of Head Size.—There is a very noticeable decrease in variability as the normal heads are approached and this is not merely due to the increasing size of the head. It is partly owing to the wide range of variability in the head measurements of low grade cases that the possibility of estimating intelligence quotient from individual measurements is slight. Certain investigators, however, have attempted to justify inferences of this kind by the comparison of head sizes of defectives at different levels of intelligence with a percentile scale of normals, based on the frequency of various head sizes.[1],[2] The same principle has been applied by Porteus [3] to distinguish temperamental groups of feeble-minded and imbeciles, such as the stable and the unstable, the former, in his view, having smaller heads than the latter. The prognostic importance of such data is small. It is not sufficient to know the relative frequencies of head sizes within groups of normals and defectives, the actual frequencies of the classes themselves must be considered. If we actually know an individual is of low intelligence for other reasons, the knowledge that his head is small may be of use in describing his type, but will not help to disclose his I.Q. On the other hand, if we know nothing about the mentality, we must consider the initial probability of a person, taken at random, having a subnormal I.Q. before proceeding to make any inference about his intelligence from the size of his head.

In the following table the frequencies of various cranial capacities, in a sample of normal adult males collected by

[1] Berry, R. J. A. and Büchner, L. W. G., Correlation of Head Size and Intelligence of 355 Melbourne Criminals. *Proc. Roy. Soc. Vict.*, Vol. XXV, N.S., pp. 229–53.

[2] Miller, E. M., *Brain Capacity and Intelligence*, 1926. Monograph Series No. 4, Australian Assoc. of Psych. and Phil.

[3] Porteus, S. D., *Studies in Mental Deviations*, 1922, Vineland, N. J.

the writer, is compared with the frequencies of the various capacities in 100 male aments already referred to.

Cranial Capacity.	100 Adult Male Aments.	100 Normal Adult Males.
2,000–2,099	1	—
1,900–1,999	—	—
1,800–1,899	1	—
1,700–1,799	1	1
1,600–1,699	3	5
1,500–1,599	11	24
1,400–1,499	29	41
1,300–1,399	30	24
1,200–1,299	17	5
1,100–1,199	7	—
Total	100	100

Now there is, in the general population, roughly one ament to every hundred mentally normal persons, hence the chance that any given person, taken at random, is an ament is 1/100. If such an adult male, taken at random, has head size of, say, 1,250 c.c., although this head is small, the chance that he is mentally defective is only slightly increased by this new knowledge, and it is, in fact, $17/5 \times 1/100$ or 1/29·4, judged by the frequencies given in the above table. Only if heads are very big or very small can a likely diagnosis of amentia be made on head size alone. Unless a given head size is at least a hundred times as frequent among defectives as among normals, the chances are still in favour of the individuals being mentally normal as judged by this variable alone. It is remarkable what small heads it is possible for normal and even very clever people to have. Clearly quality is an essential factor as well as quantity in the functioning of the brain. But, whereas it is almost impossible to make useful prognostications about intelligence given head size in a single individual, such inferences may be drawn if a large enough group of individuals is taken. Thus in Table C, given on p. 30, both male and female idiots have, as groups, significantly smaller heads than the corresponding groups of very high grade

patients. It is, however, impossible similarly to distinguish the high grade feeble-minded from the normal.

Head Size and Stature.—There is another important problem in connection with the attempt to make prognostications from the size of the head. Nutrition may enter in as a significant factor in determining the cranial volume. Nutrition may also express itself in other physical measurements, such as stature, and some people regard a stature-capacity index as more reliable in estimating the size of the brain : that is, we are to judge head size in relation to body size. This index is simply the capacity of the cranium [1] divided by stature. When this index was correlated with intelligence in Estabrooks' data, the associations he obtained were diminished to a level of insignificance. It is probable that a similar result would follow if these two variables (stature-capacity index and intelligence) were correlated in aments, because of the strong association between physical growth and mental growth.

Physique of the Mentally Defective.—Some interesting observations have been made on the measurements of physical development of mental defectives. Tarbell appears to have been the first to compare the growth of normal and mentally defective children. He came to the conclusion that the latter were shorter, less heavy and slower in growth than the former. Shuttleworth, soon after, confirmed these observations and, much later, Goddard gathered data from 11,000 defectives,[2] showing that, on the whole, the lower the grade of defect, the earlier and more marked was the decline of physical growth as compared with the normal. When high grade defectives, however, are considered, the results of various observers are somewhat contradictory, and this is still more evident when deviations of intelligence between normal limits are studied. Though it is to be expected that, on the average, the well-endowed brain has a good physique, efficient circulation and metabolism, etc., to keep it going, we are all familiar with

[1] It is more logical to take the cube root of the capacity.

[2] Goddard, H. H., Height and Weight of Feeble-minded Children in Am. Inst. *Journal Nervous and Mental Disease*, April, 1912, Vol. XXXIX, No. 4, p. 217.

exceptional instances. Common experience suggests that, among schoolboys and undergraduates, those who are best endowed with muscle are less efficient in their studies than their myopic, flat-footed and unathletic contemporaries. An analysis of routine physical examinations on 14,176 retarded schoolchildren, published by Dayton (1930),[1] reveals a different picture. Significant associations were found between the number of physical defects observed and the amount of the individual mental retardation, both for males and females. It must be conceded that, as far as the study of mental deficiency is concerned, this result is in accordance with general experience because of the many pathological conditions found among low grade cases. It does not follow that the same result would be given by a study of normal schoolchildren. Special factors come in here which do not affect the retarded group, those who give more time to athletic pursuits having less time for study and *vice versa*.

The "Physical Quotient."—Quite recently Davenport and Minogue[2] have attempted to estimate physical development in children by taking certain landmarks—the condition of the teeth as regards cutting and dehiscence, the state of pubic and axillary hair and stature—and using them as mental tests were used. The physiological or standard age is found in each case for the event's occurring in the subject at the time of examination. For example, if a boy is 15 years old and his stature is 1,407 mm., his age with respect to stature is 11 years —or four years retarded. The arithmetical average of physical ages for several events is found. This average age, divided by chronological age, gives the "physical quotient." The authors compared the "P.Q." with I.Q. in 78 feeble-minded boys under 14 years of age, and obtained correlations varying between 0·29 and 0·49. The desire to express all qualities in terms of "quotients" seems here to have outrun its usefulness. The systematic study of physical and physiological development in

[1] Dayton, N. A., Correlation between Intelligence and Physical Condition in 14,176 Retarded Schoolchildren. *Medical Journal and Record*, Sept. 3rd, 1930.

[2] Davenport, C. B. and Minogue, B. M., *Human Biology*, Dec., 1930, Vol. II, No. 4.

mental defectives which these workers have initiated requires much more elucidation, especially in connection with endocrine disorders where, contrary to what is often supposed, physical development brought about by the exhibition of glandular preparations need not be associated with a like degree of mental improvement.

Significance of the Relation of Bodily Development to Intelligence.—If we consider together the evidence of Shuttleworth, Davenport and others, and add it to our own common sense observations, we are practically forced to concede that some general positive relationship holds between physical and mental development. Moreover, if the ratio of head size to body size tends to be constant, the same order of correlation should hold for body size and intelligence as holds for head size and intelligence. Actually Whiting [1] found correlations of +0·324 and +0·154 respectively for mentality and weight and for mentality and height in criminals: the standard deviations of these quantities were from 0·02 to 0·04. But when we have at last proved to our satisfaction, by such methods, that the size of the individual is correlated with his intelligence within certain wide limits, a new difficulty arises. What meaning has this relationship? There are, as usual in such matters, two schools of thought. The eugenists would have us believe that physical and mental weaknesses are both indications of degenerate stock. Environmentalists, on the other hand, would put smallness of size down either to lack of nutrition, as in rickets, or to pathological causes such as paralysis of trophic nerves or imperfections in the ductless glands. Furthermore, many idiots, by their nature, are unwilling or unable to feed themselves or to masticate their food properly. This state of affairs cannot be favourable to their proper physical development. In so far as muscular development and stature may be increased by physical culture and exercise, not only the very low grade aments, but also imbeciles and even higher grades are handicapped in growth by their natural indolence, sluggishness of reaction, or idleness consequent upon mental or physical incapacity. It has even

[1] *Op cit.*

been suggested that the brain itself may be increased in bulk by the exercise of its functions so that the child actually, by the mere process of " taking thought," may increase the size of its head. The converse is seriously held to be true by pathologists who have observed, for instance, diminution in substance of the occipital cortex in subjects who have been blinded, or become deaf.[1] A " disuse atrophy " is supposed to be the rule in the cortex of the brain as in the muscular system. To what extent any of these suggestions are true remains, at the present time, largely a matter of conjecture. The positive association found between metrical characters and intelligence among mental defectives has to be looked upon, to some extent, as the result of the mental deficiency. Their significance cannot, therefore, be regarded as very great. At best, measurements of height and weight can be used as an aid, though not an infallible aid, to diagnosis. Their value is similar in this respect to head measurements. Only a very low probability can be given to any inference of mental grade made from physical measurements. As Pearson [2] points out, when scrutinizing Norsworthy's data from the comparative examination of 150 defective children, as far as height and weight were concerned, with the exception of one dwarf, the whole of the defectives might have been selected out of a group of normal children of adequate numbers.

[1] Berry, R. J. A. and Gordon, R. G., *The Mental Defective*, 1931, Kegan Paul, Trench and Trubner, London.

[2] Pearson, K. *Questions of the Day and of the Fray*, IX, 1914.

CHAPTER IV

SYSTEMATIC INVESTIGATION : LABORATORY WORK

Blood Examination — The Wassermann Test — Blood-Groups — Cerebrospinal Fluid—Morbid Anatomy—Histology.

Blood Examination.—Although it seems probable that the study of mental deficiency, in common with other branches of biological science, will eventually be influenced by the recent increase in biochemical knowledge, the results of the application of laboratory tests in this study up to the present time, with one or two exceptions, have been anything but illuminating.

The medical investigator naturally expects to find out a good deal from examining the blood in various ways. In what respect, he asks, will the blood of the mentally defective be found to differ from that of the mentally normal ? He may then proceed to count the various types of cells present, to find the percentage of hæmoglobin, and the results give him precisely the same information whether the tests are done on the blood of defective or normal persons. While certain observers have called attention to some general tendencies, nothing specific has been found in the blood of mental defectives. Reduction of the hæmoglobin content and various degrees of anæmia are fairly frequent findings.[1] But these are likely to be simply the results of malnutrition and ill-health. A slight lymphocytosis has been found fairly constantly in mongolism : but this may be due to tuberculous or other infection. Progressive anæmia with a rapid fall in the hæmoglobin index is sometimes found in congenital syphilis and may be the only sign of the disease.

The Wassermann Test.—In mental deficiency, serological reactions of the blood are of considerable interest, though

[1] Kuenzel, M. W., The Hæmoglobin Indexes of Two Hundred Feeble-minded " Children." *The Training School Bulletin*, Vol. XXVIII, March, 1931, No. 1, p. 11.

not always as informative as one would like them to be. The Wassermann test which, when positive, is usually held to be a strong indication of syphilitic infection, sometimes fails, in congenital syphilis, to give any hint of the condition, even though clinical examination points strongly to such a diagnosis. Furthermore, if the blood gives a negative reaction in a case of congenital syphilis, it is very unlikely that the cerebro-spinal fluid will give a positive reaction. There is, of course, some acquired syphilis among the higher grades of patients. Such patients are very likely to be promiscuous and to spread venereal diseases and, on account of mental inertia or ignorance, they do not tend to go to hospitals for treatment.

The results of testing the blood of mental defectives for evidence of syphilis have been extremely inconsistent with one another. The following table gives a summary of a number of such investigations carried out by different observers :

Date.	Investigator.	Type of Case.	Number of cases.	Positive reactions.	Percentage positive.
1909	Raviart	Unselected	246	76	30·9
1909	Kellner	,,	216	8	3·7
1910	Dean	,,	330	51	15·4
1911	Thomsen	,,	2061	31	1·5
1913	Gordon	,,	400	66	16·5
1913	Fraser	Feeble-minded children	99	45	45·5
1922	Key and Pijper	Unselected	217	120	55·2
1924	Weiss and Izgur	,,	1633	41	2·5
1925	Stewart	Idiots and Imbeciles	800	158	19·7
1931	Cobb	Unselected	1275	129	10·1

It might be supposed that much of the disparity in the above figures is due to different types of cases coming under the different surveys. For instance, a large number of cases of acquired syphilis would distort the results and one would perhaps be more inclined to suspect congenital syphilis in low grade paralytic cases than in healthy medium grade patients. It is noticeable in the above examples that the incidence of positive reactions is on the whole higher when the number of cases tested is small than when it is large. It

looks as though investigators usually began testing because they were attracted by a group of cases where positive findings seemed likely to be, or actually were, very frequent. After such groups were used up negative results were the rule. Apart from this source of variation, there are other even more serious causes of error. The Wassermann reaction, until recently, has not been a highly standardized test and, even at the present time, a number of techniques are in use, differing slightly from one another, although Wyler's No. 1 Method, recommended by the Medical Research Council, is most generally accepted.[1] The reaction, moreover, is so delicate that it is quite possible for two observers to make tests on the same specimen, using slightly different techniques, and for them to give *bona fide* results which differ from one another. For example, 500 specimens of venous blood, taken from patients at the Royal Eastern Counties Institution, were tested independently by two separate laboratories. Laboratory A returned 38 positives, or 7.6 per cent., and laboratory B returned 40 positives, or 8·0 per cent. So far, so good; but when it is found that in only 11 cases, or 2·2 per cent., did the two laboratories agree that the *same specimen* gave a positive reaction, the result is less satisfactory. Taking into account the varying strengths of the reactions given, the correlation of the findings of laboratory A with laboratory B as regards the Wassermann reaction on the same specimen of blood was only $+0{\cdot}30\pm0{\cdot}04$.

Weiss and Izgur,[2] although finding among their patients only 2·5 per cent. with positive sera, recorded, in addition to these, 2·7 per cent. giving doubtful reactions. The present writer has arranged for 1,049 unselected cases of mental defect to be tested by the Wassermann reaction and, of these, 32, or 3 per cent., were positive, but an additional 103, or 10 per cent., gave doubtful or inconsistent results. One of the difficulties seems to be that, if the test is very delicate, it is liable to give falsely positive reactions in conditions other

[1] Wyler, E. J., The Wassermann Test. *Medical Research Council, Special Report, No.* 129, 1929, H.M. Stationery Office.

[2] Weiss, M. and Izgur, L., Syphilis as a Factor in the Etiology of Mental Deficiency. *Journ. American Medical Assn.*, 1924, Vol. LXXXII, pp. 12–14.

than syphilis. On this account it is advisable to avoid taking blood for the test during a febrile attack or too soon after a meal. On the other hand, if the test is not delicate enough, many cases of congenital syphilis, especially in adults, will be missed. In view of all these complications, the incidence of positive Wassermann reactions in a group of mental defectives cannot be taken, by itself, to represent the incidence of congenital syphilis, unless a very wide margin of error is allowed. The precipitation reactions, such as the Kahn test, Sigma test and Meinicke reaction, are of no greater value than the Wassermann test in diagnosing congenital syphilis. Not infrequently one of these tests is found to be positive, perhaps strongly positive, in a case of suspected congenital syphilis, where the Wassermann finding is negative, but the converse of this is also quite common.

Biochemical tests of a more definite character than these reactions are sometimes usefully applied to the blood of mental defectives. In the rare condition of renal dwarfism, the evidence supplied by a blood urea test will probably be conclusive. Sugar tolerance tests may sometimes be of value in pituitary dystrophies and a high sugar tolerance has been reported in mongolism. In *dementia præcox* and in some other types of mental disorder, the blood cholesterol has been found to be increased, and this fact could perhaps be used to differentiate between psychosis and mental deficiency. Generally speaking, experience bears out what common sense might lead one to suspect, namely that, as regards tests of this kind, the great majority of mental defectives give normal reactions. There is, however, need for a great deal more research work to be done in this part of the subject.

Blood-Groups.—An interesting branch of serology concerns the identification of the agglutinogens A and B which were discovered, by Landsteiner, to be present in the serum of human beings and monkeys. It was shown by Snyder that no peculiarity existed in the blood-grouping of mental defectives to differentiate them from the rest of the general population. There are, however, uses to which the blood-grouping of mental defectives can be applied.

The first of these is in tracing paternity, the second concerns linkage studies in heredity, and the third use is connected with ethnological considerations. If we are able to test parents and children for blood-group there will be certain cases where the possibility of a given parenthood can be excluded or rendered improbable. The value of this test is rather limited, but, owing to the low frequency of the B allelomorph (5·5 per cent.) in English people, it sometimes gives useful results.

When a genetic condition is being studied, it is always conceivable that the gene determining it may be located on the same chromosome pair as those determining blood-groups. Should this happen, the application of an analytical method, devised by Bernstein, may, in a large series of cases, give an indication of linkage. Unfortunately, the chances are very much against an investigator's being lucky enough to discover anything of this sort in the study of mental deficiency, since so few conditions coming under this heading can be attributed to single gene substitutions.

If, in a given collection of persons, the proportions of the different blood-groups are found to differ considerably from those in another collection, the difference may be due to the two classes having been been derived from different ancestral racial stocks. In a mixed community, like the British people, one would expect to find slight differences between the Welsh and English, for example, and bigger differences between Jews and the rest of the community. The present writer has tested the bloods of mongolian imbeciles, in this connection, and obtained the results given below. No sufficient difference was demonstrated, between the distribution of the blood-groups among these patients and the distribution among other patients, to suggest that racial Mongolian ancestors are any more frequent for mongolian imbeciles than for other defectives.

PERCENTAGE DISTRIBUTION OF BLOOD-GROUPS.

	Type of case.	O	A	B	AB
158	Mongolian imbeciles . .	41	49	8	2
225	Non-mongolian aments .	43	47	7	3

The M and N agglutinogens, which were more recently discovered by Landsteiner, might form the subject of

investigation from the points of view already mentioned. The technique of testing for them is, however, considerably more complicated than the technique of testing for the A and B agglutinogens.

Cerebrospinal Fluid.—When studying mental deficiency, the examination of the cerebrospinal fluid has a certain value, and it has been asserted that the process of drawing off the fluid may be beneficial to epileptics. As in general medicine, Wassermann reactions of the fluid may be useful in the differential diagnosis of paralytic symptoms. An enquiry, affecting specifically the problems of mental deficiency, was made by Riddell and Stewart,[1] who described an abnormality present in over fifty cases of mongolism.[2] The fluids all gave a mild " luetic curve " in the colloidal gold test. The Wassermann reaction was positive in only one of these cases, and it may be legitimately doubted whether the luetic reaction had any relation to congenital syphilis. It seemed, however, to show a clear biochemical distinction between these defectives and normal persons.

Morbid Anatomy.—The well known naked eye appearances encountered in general medical study form a large part of the pathological anatomy which comes under consideration in the special study of mental defect. But there are a number of peculiar conditions, mostly concerned with the brain, not frequently met with in hospital practice, which come under the eye of the pathologist in an institution for the mentally defective. These conditions are described in all thorough-going textbooks on morbid anatomy.[3] They include microgyria, porencephaly, hydrocephaly and various types of brain sclerosis as well as pathological changes due to cerebral inflammation. It may be impossible to assign any known cause to the anatomical findings. Occasionally tumours of various kinds are encountered, and the effects

[1] Riddel, D. O. and Stewart, R. M., Syphilis as an Etiological Factor in Mongolian Idiocy. *Journal of Neurology and Psychopathology*, 1923–4, Vol. IV, p. 221.

[2] This abnormality was first described by Stevens, H. C., Mongolian Idiocy and Syphilis. *Journ. Amer. Med. Assn.*, 1915, Vol. LXIV, pp. 1636–40.

[3] Aschoff, L., *Pathologische Anatomie*. 1923, B. II, p. 327, Fischer.

of cerebral hæmorrhage in congenital syphilis sometimes present a clear picture. On the other hand, it is remarkable how difficult it often is to detect any naked eye deviation from the normal in the brain of a person who has during life exhibited the phenomena of mental deficiency. This is especially so in high grade cases, and a natural inference to be drawn is that some pathological changes are present but only detectable microscopically.

Histology.—Although many recent textbooks concerned with this subject have repeated the statement that mental deficiency has a characteristic cerebral micropathology,[1],[2],[3] the actual evidence for these statements is not of a very satisfactory nature. We are here faced with the same problem which arises in connection with the cerebral micropathology of dementia præcox. A great number of histological studies have been made of the brains of the insane, but in estimating the value of the results, allowance has to be made for secondary changes, which may have occurred in the brains examined, due to old age or intercurrent disease. Mentally defective patients, even more than the insane, tend to live for a long time in the institutions where they are cared for. This applies more to the high grade patients than to idiots, who often die of intercurrent disease. Thus it is only natural that most of the histological studies which have been made on the brains of aments have been carried out on low grade subjects. The results of the classical work of Bolton,[4] who examined the brains of a number of defective patients, cannot be applied to the higher grade defectives who now form such a large proportion of certified cases. If histological examinations were carried out systematically on the brains of mental defectives with I.Q. above 50, it seems highly improbable that any large proportion would show characteristic pathological changes.

[1] Tredgold, A. F., *Mental Deficiency* 5th ed., Baillière, Tindall & Cox, 1929.

[2] Berry, R. J. A. and Gordon, R. G., *The Mental Defective*. Kegan Paul, Trench and Trubner, 1931.

[3] Shrubsall, F. C. and Williams, A. C., *Mental Deficiency Practice*. Univ. of London Press, 1932.

[4] Bolton, J. S., Amentia and Dementia. *Journal of Mental Science*, 1908.

In view of the slightly diminished average head measurements of this group, as compared with the normal, it is possible that some quantitative changes might be observed in the number of cells per unit brain volume or in the thickness of the cerebral cortex. Such quantitative results, however, would only express themselves in the average of a large number of cases. A person who, from the clinical point of view, appears to be perfectly normal, but who is merely stupid, is suffering from a disability of a very subtle kind. The belief that micropathological changes can be easily found to account for it implies a great scientific optimism on the part of those authorities who hold it. Probably the real abnormality lies in the functioning of the individual brain cells and their inability to form adequate connections with other cells. There is no known histological method which differentiates between normally active nerve cells and those slightly less active. Tredgold, Berry, and others have laid great stress on the immature nerve elements which are to be found in the cortex in certain cases ; but it seems that these cells occur only in low grade aments, where there is gross cerebral damage or deformity. It is possible that immature cells of this kind may be found to be characteristic of the brains of people who are merely simpletons. At the present time experimental evidence is insufficient to decide the question one way or the other. Our knowledge of the relationship between the structure of the cerebral cortex, as revealed by the microscope, and the intelligence, as ascertained by mental tests, is extremely limited.

CHAPTER V

SYSTEMATIC INVESTIGATION : PSYCHOLOGICAL EXAMINATION

The Psychology of Mental Deficiency—Psychophysical Tests—Intelligence Tests—Conditioned Responses—Emotional Factors.

The Psychology of Mental Deficiency.—At the present time there are many separate schools of thought in psychology and it is very difficult, while speaking in the language of one school, to be intelligible to adherents of another. I shall, therefore, confine myself to the simplest issues, which can be discussed, for the most part, in ordinary language.

There are three sides to the mental examination which it is necessary to make in order to study the psychology of a mentally defective, or supposedly mentally defective, person. Sensitivity, intellectual ability and emotional development have to be examined. These investigations are intimately bound up with one another and can scarcely be separated either practically or theoretically. The emotional and intellectual aspects of the mind both have their origin in instinctual behaviour which has been modified by a process of conditioning ; and the extent of this modification is dependent on the organism's sensitivity.

The main peculiarity of the behaviour of man as compared with other animals is that his instinctual habits are so extremely complex. He has to acquire many reactions which, in animals, are inborn.[1] For example, he even has to *learn* to walk. In order to take part in civilized life, he has to learn innumerable performances which, later on, become habitual, such as talking, dressing and maintenance of personal cleanliness. He must learn to control his primitive instinctual desires and emotions in a way acceptable to the society in which he lives. He has to learn a great many other things which are usually

[1] Wood Jones, R. and Porteus, S. D., *The Matrix of the Mind*, ch. XXXIV. Edward Arnold & Co., London, 1929.

characterized as education. These educational or scholastic attainments vary greatly in different races and in different classes of society within the same race. Much of this process of learning takes place quite unconsciously by copying the behaviour of other persons. Success in learning the behaviour patterns which are acceptable to society necessitates the integrity of certain mental mechanisms. There must be sufficient sensitivity to perceive what other persons are doing ; there must be ability to remember and to form associations ; and there must be some instinctual desire, which has to be satisfied, to set the learning apparatus in motion.

Now if we leave out of consideration, for the moment, individuals afflicted with gross physical disease or deformity, there is still, in the general population, a large range of variation in mental capacity. A person is judged to be mentally defective or otherwise according to his social behaviour, and a fault involving a quantitative diminution in any part of the learning apparatus may lead to mental deficiency. I have already mentioned that defectives are sometimes found to have diminished sensitivity to pain. This may be a fundamental factor in producing anti-social behaviour, since it prevents the subject from understanding the feelings of other people. There is a traditional category of patients designated amentia by *sense deprivation*, which includes persons deaf or blind from an early age. It is not surprising that many deaf or blind children are mentally below par. To overcome the obstacles of sense deprivation the subject requires more than ordinary ability in other spheres.

It is commonly held that what defectives specifically lack is intelligence as shown in the failure of the standard mental functions, such as memory and association. Clearly, a person with no ability to remember will not learn, however keen his senses may be. There are, however, certain defectives whose disability seems to consist simply in the lack of sufficient instinctual drive—call it will-power, libido, or what you may—while their sensitivity and their intellectual capacity seem to be normal. They are listless, inattentive, and they lack stamina. They invariably take the easiest course, which usually leads them

into trouble, and then they complain of being badly treated. It is quite possible that in such cases we are really dealing with some form of mental disorder.

Psychophysical Tests.—The earliest psychological measurements, made under laboratory conditions, were those of Weber. His work, published in 1846, was concerned with sensation and appreciation of stimuli. These experiments were not aimed at detecting individual differences and the first studies which had this as their object were made by Wundt, who noted, for example, the difference of speed of reaction in different subjects. It was not long, however, before a great number of metrical tests, showing individual differences, began to be applied. Most of these tests were purely psychophysical and unrelated to educational attainment, and it is interesting to note how little the results of these tests were correlated with intellectual development. Quickness of motor reaction, in fact, showed a negative correlation with class standing.[1] Sensitivity to pain, however, showed a positive correlation with intelligence. A great deal of work remains to be done in these fields, and it is a pity that they are at the present time eclipsed by the enthusiasm for intelligence tests.

Intelligence Tests.—On the intellectual side, individual differences in ability to remember began to be studied systematically at the end of last century, and some relationship was demonstrated between teachers' estimates of mental ability and the results of tests.[2] The classical work of Binet, which subsequently attained such enormous popularity, was definitely aimed at ascertaining educational standing. The majority of intelligence tests at present in use are on the same basis, and they are applicable only to groups of persons with similar educational opportunities. The Binet scale, like many others, is composed of a number of different types of problems. The total score of the subject represents an arithmetical sum of the marks obtained on the various constituent tests. This method of scoring is quite arbitrary and has no sound theoretical basis,

[1] Bagley, W. C., Mental and Motor Ability. *Amer. Journ. Psychol.*, Vol. XII, p. 193, 1900–1.

[2] Bolton, T. L., Growth of Memory in Schoolchildren. *American Journal of Psychology*, Vol. IV, pp. 362–80, 1891.

as Spearman has pointed out.[1] Some tests may be more diagnostic of intelligence than others and should therefore score more.

By the introduction of performance tests, with such apparatus as form-boards and blocks, it was hoped to remedy some of these limitations.[2] Sometimes very marked differences are noticed between performance ability and scholastic test achievement—the former being greater than the latter.[3] This appears to be a more frequent finding among mentally defective than among normal people, and it shows that performance tests often discover quite unexpected special aptitudes. They are, however, less suitable than scholastic tests for persons with physical disability. Taking a series of 200 consecutive female patients, tested by both the Stanford-Binet and Porteus Maze tests, I found a mean difference of over six months between the two sets of scores, the Maze test average scores being the higher. On the other hand, I have found a significantly stronger correlation between teachers' estimates of ability in useful employments, such as carpentry, sewing and housework, and Stanford-Binet scores, than between the same estimates and scores for Koh's Block Design test. This result suggests that the Stanford-Binet scale is more diagnostic of general ability in defectives than the performance test used. Burt [4] has expressed his view that the Stanford-Binet tests, or modifications of them, give, in fact, more accurate results with children of low intelligence than with normal children.[5]

There are, however, one or two practical objections to the scholastic tests at present in vogue. In the first place no scale has been standardized for adults. The standard mental age of adults, for most tests, is supposed to be 16 years, but,

[1] Spearman, C., *The Abilities of Man*, Ch. V. Macmillan & Co., London, 1927.

[2] Gaw, Francis, *Performance Tests of Intelligence*. H.M. Stationery Office, 1925.

[3] Turner, F. D., *Mental Welfare*, Vol. VII, No. 4, Oct. 15th, 1926, p. 95.

[4] Burt, C., *Mental and Scholastic Tests*, P. S. King & Co., London, 1922.

[5] A useful summary of the theory, practice and results of intelligence tests has been given by Pintner, J. R., *Intelligence Testing*, 2nd ed., 1930, Holt, N.Y.

although the scale of tests comes to an end, chronological age goes on increasing. Burt has suggested that the mental age of 15 years should be taken for average adult performance on the Stanford-Binet scale and Lewis has used a maximum mental age of 14 years. The modifications and revisions which are used in different places do not solve this general difficulty, and the result is that an I.Q. given to an adult on the basis of his performance on an educational test is usually meaningless. For similar reasons the I.Q. becomes progressively inaccurate as the child approaches the limiting age. Secondly, the scales do not reach down with any accuracy below three years. The Merrill-Palmer scale [1] is convenient for low mental ages, but so far no accurately standardized tests for children below the age of two have been brought out. It is clear that educational tests relevant to these mental ages are scarcely possible. When dealing with mental defect, however, the question is important in relation to early diagnosis.

The ideal test in the study of mental deficiency would be one which investigated the ability to learn. There are several possible ways of devising such tests. According to Spearman, the most important element of intelligence is the ability to educe relationships. Form-boards have been devised which involve processes of this kind in their solution.[2] There is no theoretical reason why tests should not be built up on a principle involving recognition of the simplest relationships, and suitable for very young children. The same principle would be used in a more complicated way to build up tests for older children. The materials can remain similar, but the relationships which have to be educed can be made increasingly complex. The non-verbal tests devised by Fortes [3] and others are examples of attempts to do this. No one has, however, been able yet to overcome all the practical difficulties involved in the presentation of the material.

[1] Stutsman, R., *Mental Measurement of Preschool Children.* New York, 1931. This book contains historical and bibliographical notes.

[2] Kent, G. H. and Shakow, D., Graded Series of Form Boards. *Person J.*, Vol. VII, p. 115, 1928.

[3] Fortes, M., *Trans. Roy. Soc. S. Africa,* Vol. XX, p. 281, 1932.

Conditioned Responses.—It was suggested, in the report of the British Medical Association, 1931,[1] that the development of mental defectives could be regarded as a kind of slow-motion picture of the development of normal children. If this were true, many experiments might be carried out on defective subjects which would not be possible with the normal. An attempt has been made by Aldrich [2] to investigate the formation of conditioned responses in idiots. The experiments were carried out as follows :

> The observer was stationed in an observation room equipped with a one-way vision screen. Two boxes, similar in every respect except size, were placed on a table, across the end of the room opposite the door. A cookie was under the smaller box. The subject was allowed only one choice during each trial, and given five trials per day. Eighteen out of twenty trials was taken as the criterion of successful conditioning. There were no verbal instructions ; the child was merely admitted to the room and remained there until he had raised one box. The first trials took from one to ten minutes, although thereafter five seconds were often sufficient.
>
> Reconditioning to the opposite box, after completion of the first conditioning, revealed facts about the relation between building up a conditioned preference and breaking it down. There was a tendency among the subjects to show a reverse correlation between the two problems. In other words, those who learned most readily took longest to inhibit the response learned. Among those who learned the problems were marked individual differences, which revealed only a negligible correlation with mental age within the limits of the group.

It appears that experiments on conditioned responses, like the old psychophysical tests, must not be expected to be of much use for diagnosis of mental grade. Their scientific value may, however, prove to be great.

Emotional Factors.—When we come to the consideration of the emotional side of behaviour, analysis of the scores obtained in intelligence tests may reveal points of considerable interest. The phenomenon of "scattering," that is, the inability to pass relatively easy tests coupled with unexpected

[1] *Report of The Mental Deficiency Committee*, 1931. British Medical Association House, London.

[2] Aldrich, C. G., Experimental Studies of Idiot Behaviour. *Proc. of the American Association for the Study of the Feeble-minded*, May, 1931.

success in tests of greater difficulty, is regarded as indicative of emotional disorder. Apart from this there is a certain amount of evidence that emotional difficulties cause retardation in scholastic achievement. The crude score of an intelligence test, applied on different days by different persons, may vary enormously in emotionally unstable subjects. The effect of a friendly or antagonistic relationship between examiner and patient in such cases must not be underrated. The actual way in which a question is answered, even if the answer is wrong, may give information that the patient has a specific neurotic symptom. The way in which the description of pictures is performed, in the Binet tests, is sometimes of great interest. It may be worth while for the examiner to spend a considerable time eliciting the phantasies which rise in the mind of a child when presented by these pictures. From the point of view of phantasy stimulation the plates which are in use in the Binet tests could be greatly improved.

Not only in the scoring of tests and in their performance is material obtained for the diagnosis of mental disorder underlying or associated with mental deficiency. The experienced psychiatrist also judges the patient by his methods of reaction in the interview. It is not the purpose of this survey to consider the details of the diagnosis of mental disorder, but some general principles can be postulated as good practical rules. The emotionally disordered child, in some way or other, exhibits anxiety in his reactions and it is ultimately this anxiety which prevents him from performing the tests naturally. The most useful criterion for mental equilibrium is the individual's capacity for establishing and maintaining satisfactory personal relationships. If the physician can perceive to what extent this capacity is developed, whether it is disordered or constrained by anxiety and feelings of guilt, in the interview with the patient, he will be able to pronounce judgment on the emotional development of the subject. As will be seen when we come to discuss different types of persons certified mentally defective, great interest attaches to the emotional development, both from the point of view of diagnosis and prognosis.

Many psychologists have, from time to time, attempted to

provide tests which will measure individual emotional or temperamental differences. Some of the proposed tests, such as those in which a situation is described to the subject and a moral judgment elicited, are really pure intelligence tests. The intelligent subject will discover what answer is expected and may give this rather than express his own feelings in the matter. A more scientific approach is opened up in the study of perseveration and its relation to emotionally disordered states of mind. Perseveration tests have been successfully used to demonstrate ideational inertia in dementia præcox. Jung's word association tests and reaction time experiments have a similar value in skilled hands. Tests designed originally to investigate performance ability are sometimes found to give useful diagnostic hints in unexpected ways. A disproportionately high score in the " dotting test," for example, is said to be a sign of obsessional neurosis. The fact must not be overlooked that all mental tests are to some extent character tests and require the cooperation of the subject with the examiner : hence the importance, already stressed, of the personal relationship between them at the time of testing.

CHAPTER VI

PERSONAL AND FAMILY HISTORY : COLLECTING FACTS

Personal History — Conception — Pregnancy — Birth — Post-natal Environment—Scope of Enquiry into Family History—Pedigree Study—Example—Interpretation.

Personal History.—In connection with the medical examination of every patient who presents himself to the physician, for whatever complaint, an essential piece of information to be obtained is the patient's own personal history. Unfortunately, when we are dealing with persons of low intelligence, their own accounts are not likely to be of much use. We have to rely on the statements of parents, guardians and other persons, who have knowledge of the patient's childhood or more recent events. But the object of the personal history, or the whole story of the reaction of an individual to his environment, from the moment of fertilization onward, is the same whatever class of case is being dealt with. There are two main considerations. Firstly, we find out when the complaint arose and what indications there are, if any, of the patient's having been previously normal or having been better or worse than at the time of examination. Secondly, we examine every event in the patient's life which may have possibly contributed to the onset of the disability or to changes which have taken place in its intensity.

It is, moreover, important not to neglect the personal history in cases where the physician already believes, from the clinical diagnosis, that the complaint could not have been influenced by any environmental agencies and is of a purely hereditary nature. The vast majority of diseases in man require environmental stimuli to make them manifest, even if hereditary disposition is present. It is, therefore, methodologically correct to begin by examining the environment even in mental deficiency.

It is a waste of time to look for every possible cause in every disease. The examination must be guided by previous knowledge and by the inspiration of the moment. There are certain facts which it is well to examine in every case, if only for statistical purposes. Large stereotyped forms for filling up are, in general, to be deprecated because it is impossible for them to be comprehensive enough to cover the important points in the histories of all cases and, at the same time, to retain practical value. The most essential equipment for the person recording an individual history is a sound medical and psychological knowledge, and this applies even to the apparently simplest cases.

Unfortunately, a great deal of the information given on medical or psychological points by parents about their children is wholly unreliable. This is not only due to natural forgetfulness about past events, for people are liable to substitute unwittingly something which they wish had happened for something unpleasant which actually did happen. Parents are often anxious to prove that a child's disease is not congenital; this wish may lead to the elaboration of false stories of injury and disease in infancy which they eventually grow to believe. Parents are also frequently ignorant of what facts are likely to be important and omit to make useful observations at the time when important events occur. The history given by the parents becomes of much greater value when supplemented by the observations of the medical attendant, or notes from a hospital. The investigator should make a point of noting down particulars of any persons likely to have medical knowledge who may have witnessed the events concerning which he wishes to obtain information.

We will now consider some of the special circumstances which have to be enquired into in taking the history of any mentally defective person. It is, of course, essential to note the date or period at which each circumstance arose.

Conception.—In view of the belief held by some authorities (Myerson,[1] Stockard, Tredgold) that the germ plasm is sus-

[1] Myerson, A., *The Inheritance of Mental Diseases*, Williams and Wilkins Company, Baltimore, 1925.

ceptible to the action of poisons and other physical agents, the presence of adverse circumstances, at the time of conception, which might affect, in this way, the mentality of the offspring, should not be entirely overlooked. Though the formerly popular belief in the danger of " Die Zeugung im Rausche " is now discredited, it may be worth while to consider the possibility that some of the various methods of control of conception now in vogue may act adversely on the germ cells just before fertilization. These considerations do not come strictly under the heading of personal history any more than does the possible effect of chronic poisoning (*e.g.* lead) on the parental germ cells, but they arise directly out of the investigation of factors affecting fertilization. In spite of the work of Stockard [1] and others, who produced experimental results in animals indicative of injury to the germ plasm, transmissible from one generation to the next, biological opinion does not lean towards the view that such blastophthoria [2] plays an important part in human inheritance. As regards the blastophthoric effects of alcohol on guinea-pigs, Stockard's work has been carefully repeated but his results have not been confirmed.[3] It seems likely that, although a few oddities here and there may be due to blastophthoric influences, this hypothesis is not to be looked on as having much scope in describing the causes of mental deficiency.

Pregnancy.—Shortly after fertilization, the ovum becomes embedded in the wall of the maternal uterus and its subsequent development is dependent upon its being able to take root there and absorb adequate nourishment. The relation between the maternal and fœtal organisms is so intimate that, though their bloods do not mix, many things besides food substances may pass from one to the other. The standard examples of noxious influences which may pass from mother to fœtus through the placenta are the pathogenic micro-organisms of

[1] Stockard, R. S., *Experimental Production of Degeneracy in its bearing upon Hereditary Abnormalities in the Nervous System.* Hoeber, New York, 1923.

[2] From the Greek *βλαστός*—a bud, sprout, leaf (germ) and *φθόρος*—destruction, ruin.

[3] Durham, F. M. and Woods, H. M., *Alcohol and Inheritance.* Medical Research Council Special Report No. 168, H.M. Stationery Office, 1932.

syphilis and smallpox. It is not improbable that other infective diseases either of protozoal origin (malaria) or caused by filter-passing viruses (mumps) may adversely affect the growing fœtus if the mother suffers from them during pregnancy. Many types of poisons pass through the placenta if their concentration in the maternal blood is sufficiently high. And there is always the possibility that drugs taken in order to secure abortion may injure the developing embryo without dislodging it. In view of these contingencies, great stress is to be laid on obtaining as complete a history as possible about the gestation of the patient. Not only should diet (with special reference to vitamins), illnesses (*e.g.* albuminuria), attempted abortion, etc., be considered, but the whole maternal gynæcological history is relevant. Indications of disorder of endocrine glands (*e.g.* goitre) in the mother should especially be noted. In certain cases idiots have been reported to have been born after the mother has received radium or X-ray treatment. (See p. 126.)

The favourite tale of fright during pregnancy is, in most instances, found to be an exaggerated account of some quite ordinary event. Occasionally, however, one meets with *bona fide* instances of severe mental shock to the mother during the early months of pregnancy. It is possible that mental shock may disturb the maternal endocrine equilibrium and hence may affect the developing embryo or fœtus.

Birth.—There are singular opportunities in the process of birth for the child to be injured. It is, therefore, expedient to gather as much information as possible about the nature of the delivery when taking the history of a patient. It is usually impossible to get accurate information from the mother and corroborative witnesses are desirable when a history of difficult labour is given. Points specially to be noticed are whether the delivery is at full term, how long each stage lasted and what part of the fœtus presented. It is desirable also to have information about the size of the infant and its condition at birth. Birth weight is quite a useful check on the diagnosis of prematurity and also useful in considerations

relating to the health of the young infant. A mere history of instrumental labour is not to be taken as indicative of any special difficulty and the story that the child was asphyxiated at birth usually requires further investigation.

Post-natal Environment.—Unless it is clear that an infant was so grossly deformed at birth that it was practically certain to be an idiot in any case (*e.g.* microcephalic), the post-natal history must be carefully searched for adverse circumstances which, even if they are not supposed to be sufficient in themselves to cause mental deficiency, may be factors contributing towards mental retardation. The diet of the child can be enquired into and compared with the average. It is worth while to note whether, and for how long, the infant was breast fed. This part of the history gives information useful from more than one point of view. A mother's inability to feed her child may be due to some pathological condition affecting her or it may be due to the infant's inability to suck. If the latter is the case it is sometimes the earliest historical piece of evidence elicited bearing on the mental condition of the child. Accounts of fits or convulsions in infancy are frequently obtained from relatives and it is always extremely difficult to know what importance to attach to these stories. Some mothers seem to take a pride in having revived their children from convulsions and are anxious to make out that the infants have been subject to these dangers. In other instances the parents of a child, whose disability is supposed to be due to an infectious disease or injury, may conceal the fact that it was subject to epileptiform attacks long before this particular illness occurred.

It is not usually believed, by those who study mental deficiency, that the retardation observed can be due to psychological causes. In view of the recent work of psychologists who have stressed the exceptional importance of the first few years of life in the formation of character, it is well to reconsider this belief, especially where the patients show neurotic tendencies. Those persons who have been brought up in well-ordered homes with kindly parents find it difficult to realize the possible mental state of a young child in a family

where, say, the mother is feeble-minded and the father a drunkard or epileptic. Serious mental retardation may possibly occur as a consequence of fright in infancy. But unequivocal evidence for this view will not easily be found, because it is probable that the psychological traumata which are most harmful to the children are usually, at the time of their occurrence, quite unrecognized by the parents.

It is hardly necessary to point out that the educational opportunities of different children vary with social class. Lack of such opportunity may, later on, cause failure in mental tests of a scholastic nature.

Scope of Enquiry into Family History.—The investigation of a patient's personal history merges imperceptibly into the study of his family. If the patient's birth is recorded as difficult, then this must be compared with the births of the brothers and sisters. Apart from hereditary likenesses, the child's mentality is, in many ways, copied or modelled on that of the parents. The parents' social status and ability determine the physical and mental nutrition of the children. Moreover, the infant's health is dependent upon the mother's health. We at once find ourselves asking about the health of the mother's relatives and of the father and his relatives. The further the investigation is conducted, the more scanty the information obtained. This is shown clearly when the investigation extends to the grandparents of the patient. How far is the enquiry to be pursued ?

It is common practice to ask specifically whether this or that relative was affected in the same way as the patient. This may be an excellent initial method of procedure, but it gives misleading results unless every member of the family group is made the subject of separate enquiry. The shorter method of enquiring whether *any* member of the family has suffered from this or that disease is to be deprecated strongly in serious investigations. In taking family histories of persons affected by any form of mental disease it has been customary, in the past, to ask this question. If the answer was in the affirmative, the patient was supposed to be likely to be suffering from a complaint hereditarily determined. There is a large initial pro-

bability of mistake or concealment in the answer to this question. In certain classes of the community, for instance, even relatives by marriage are considered to be members of the family. Apart from this, it makes all the difference in the world whether a brother or a great-uncle had the same condition as the patient.

Enquiries into family history should be guided by the consideration of what use is to be made of the knowledge when it is obtained. If we suspect that the disease under investigation is inherited according to some simple dominant mendelian principle, and if the history be taken in the form of a pedigree study, the more generations which are included the more convincingly will the hypothesis be tested. Examples of simple dominant inheritance are, however, very rarely met with in mental deficiency.

If it is supposed that a single mendelian recessive character is the cause of the disease, and if the disease is rare, it is unlikely that the parents or remoter ancestors will be found to be affected, however far back into the family history we may probe. On the other hand, it *is* probable that another member of the patient's fraternity will be affected in the same way as the patient whether the type of inheritance is dominant or recessive. It is sufficient, with modern methods of analysis, to have full knowledge of the condition of the parents and of the patient's brothers and sisters in each case, provided also that the occurrence of consanguinity between the parents is always noted. The larger the series of families studied in this way the more accurate will be the result of the subsequent analysis.

In medical literature it is customary to find only family histories recorded where positive results are obtained or results easily explicable on simple mendelian lines. Thus the subsequent perusal of medical records can give an entirely false impression. Those examples in which no familial incidence occurs are usually not published. A systematic family study of every available case of the disease in question is necessary.

Pedigree Study.—As with the patient's personal history,

so with the history of the family it is a methodological error to assume at the start that the condition which is being studied is due solely to hereditary causes. Such an attitude is opposed also to the medical endeavour to cure or alleviate diseases by altering the environment of the individual in some more or less drastic way. The fact that a genetic predisposition to pneumonia or tuberculosis may be present in patients suffering from these diseases is not one of the considerations which affects the immediate task of the physician in treating them. To have assumed in a lazy fashion that such diseases were due to heredity would have retarded very seriously the progress of medical science had it been applied in practice. Mental deficiency is a group of diseases and symptoms which require investigation and treatment in exactly the same way as other diseases. Until the environmental precipitating factors which are involved are properly sorted out we cannot hope to clear up the genetic basis—except in a small minority of cases.

The first essential in a human pedigree study is to get a complete picture of the sibship of the patient, giving dates of birth. When the ages of the parents are added to this information, a base-line is formed with which further observations can be compared. Accurate ascertainment of miscarriages and stillbirths can be made much more often than is generally supposed, though for this it is essential to make enquiries from the mother personally. Gaps of three or more years between births, when not accounted for by absence or estrangement of the husband, may be due to birth control, but they often suggest the possibility of intervening miscarriages or stillbirths. The fact of such an occurrence, though disputed by the mother, is sometimes confirmed by her medical attendant.

To sum up, the value of the family history depends on the completeness with which the individuals in a given generation are studied. It is far more useful to have accurate knowledge of the sibship and parents of the patient alone than to have any amount of information about cousins and grandparents.

Example.—As an example of some of the peculiarities which may be met with in the study of pedigrees of mental defectives, the following case history may be worthy of notice.

THE PATIENT. (See plate II, facing p. 108.)

A male, characteristic microcephalic imbecile, aged 20 years : head-circumference 18½ inches : cephalic index 0·69 : cranial capacity 1,112 c.c. : Wassermann reaction negative : mental age 3 yrs. 6 mo. (Stanford-Binet).

He has a spastic paralysis of cerebral origin, affecting right arm and leg, without athetosis. He suffers from epileptic fits of the major type. The hair is stiff and black and the scalp shows well marked longitudinal ridges or convolutions. He is, on the whole, quite good-tempered, but is very lazy. His speech is imperfect, but he can carry on a simple conversation. When asked about his home, for instance, he answers " wy wather wer on wayway " (my father works on railway), showing the typical " lalling " speech of imbeciles—the substitution of the same easy sound for a number of difficult ones. Apart from deformities due to long-standing paralysis, his body is well developed. The genital organs are normal and he is much addicted to masturbation. He can feed himself and can dress himself with help, but is not always clean in his habits.

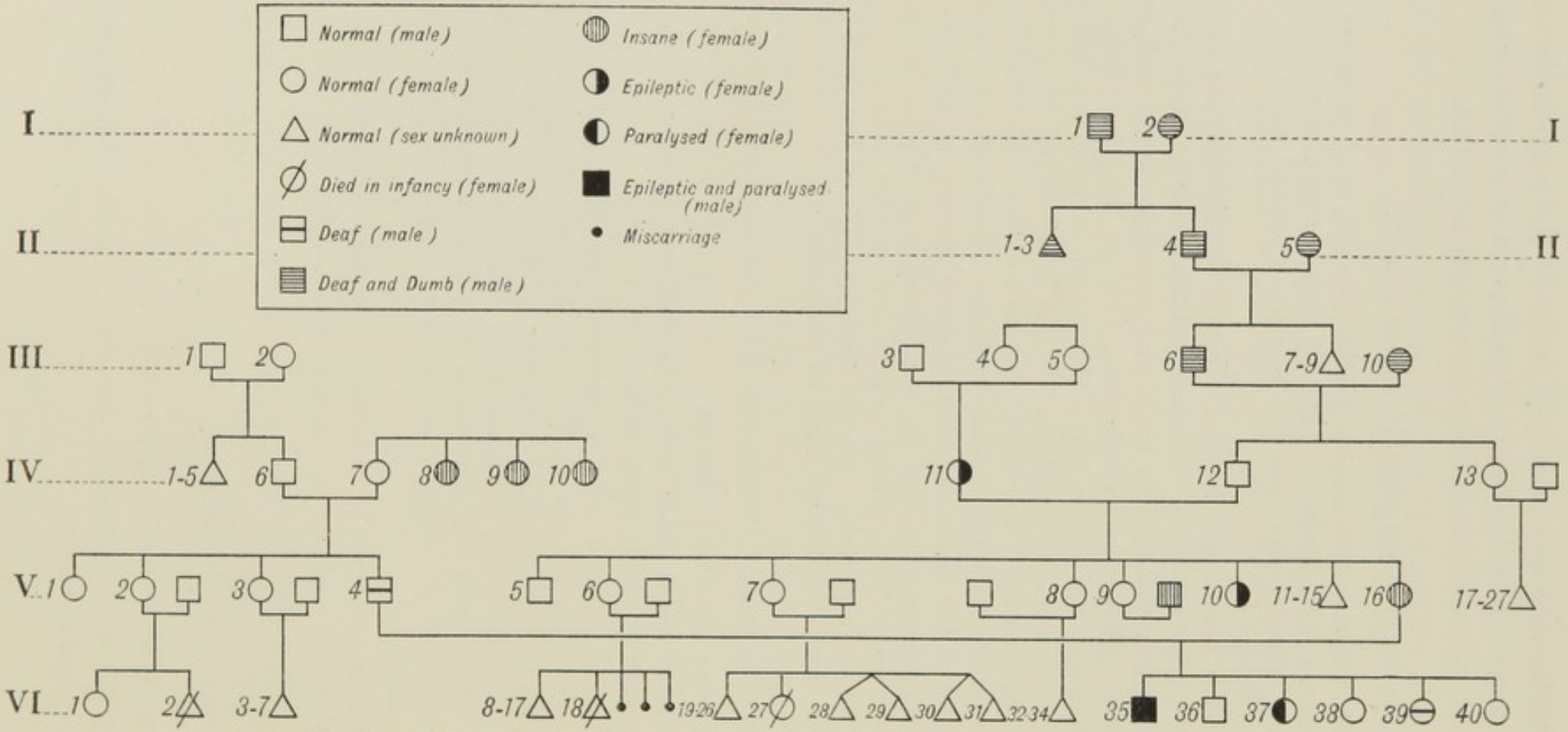
PATIENT'S SIBSHIP (Generation VI : see diagram, page 62).

35. Male : aged 20 : patient.
36. Male : aged 19 : history of difficult labour : works in a factory and is doing well.
37. Female : aged 16 : a severe case of cerebral diplegia, thought, by the mother, to be due to falling out of a pram : at 3 years there was marked motor disability and a slight mental impairment : the mother took great care of her and she appears to have been regarded as mentally normal at school and is now employed in a printer's office.
38. Female : aged 11 : a perfectly normal, intelligent child.
39. Female : aged 9 : seems to be normal physically and mentally, but is slightly deaf.
40. Female : aged 7 : normal.

No history of miscarriages.

FATHER (Generation V. 4).

Aged 48 : warehouseman : earns 70*s*. a week : steady and hard-working : deaf in one ear.



Pedigree Chart showing Numerous Pathological Conditions. (See pp. 61–65.)

FATHER'S RELATIVES.

Generation III.

2. Lived to be 93 in spite of breaking her leg at age of 87.

Generation IV.

1, 2, 3, 4, 5, 6, 7. No evidence of mental abnormality.
6. (Paternal grandfather.) Died aged 80.
7. (Paternal grandmother.) Died aged 75.
8. A patient in a mental hospital for many years.
9. Died in a mental hospital.
10. Reported to have been insane.

Generation V.

1. Died of tuberculosis aged 20.
2. Died in childbirth and the infant died of whooping-cough at 8 months. A healthy girl survives.
3. Normal, with 5 children (VI. 3–7).

MOTHER (Generation V. 16).

Aged 46 : a superior, intelligent woman, large in stature, but with a small head. She reported that many of her family also had small heads. For this reason the small size of the patient's head in infancy did not worry her. She was admitted to a mental hospital at the age of 38 suffering from melancholia 7 months after the birth of her youngest child. She was discharged, recovered, 3 months later.

MOTHER'S RELATIVES.

Generation I.

1. and 2. Reported to have been deaf and dumb.

Generation II.

1, 2, 3, 4. All reported to have been deaf and dumb : number not known exactly.
5. Reported to have been deaf and dumb also.

Generation III.

3, 4, 5. Reported healthy.
6. Deaf and dumb, good financial position, married a deaf and dumb woman (III. 10).
7, 8, 9. Exact number not known : none of them reported abnormal in any way.

Generation IV.

11. (Maternal grandmother.) Said to have been healthy as a girl, but suffered from fits latterly. Died at the age of 54 from heart failure.

12. (Maternal grandfather.) Died age of 54 of Bright's disease, previously healthy.
13. Said to have been healthy and to have had a family of nine or ten children, also healthy. This family could not be traced (V. 17–27).

Generation V.

5. Killed in the War.
6. Healthy : ten children alive and well, one died at birth and there were three miscarriages (VI. 8–18).
7. Healthy : has nine or ten children all living and healthy and has also lost three—has had twins twice and each time one child has lived and one has died. A girl of 10 months died of meningitis (VI. 19–31).
8. Healthy : several healthy children (VI. 32–34).
9. Died aged 50 : married a man who has now been for some years in a mental hospital : no children.
10. Suffered from epilepsy for many years, no mental impairment recorded.
11. Died of diabetes aged 9.
12, 13, 14, 15. All died in infancy, two of whooping-cough and two of measles, order uncertain.

Interpretation.—This family history is exceptional only on account of the number of generations concerning which information has been obtained. The diversity of pathological conditions which have cropped up among various members is not an unusual feature. From the point of view of studying hereditary factors in the causation of mental deficiency in the patient, this pedigree might be used by the advocates of a theory of the inheritance of neuropathic constitution as a magnificent example confirming their view. Insanity on the father's side is clearly neuropathic. Deafness and dumbness can perhaps be brought under this heading with a stretch of the imagination. The epilepsy in the patient's maternal aunt and grandmother would, of course, be evidence of neuropathic constitution and the mother herself has been in a mental hospital ; a sister of the patient suffers from a definite organic nervous disease. Such an interpretation of the data is entirely out of touch with modern genetical concepts. What we must look for is inheritance on mendelian lines, of which this pedigree furnishes very little evidence, if, in fact, any at all. It is necessary, in order

to establish the presence of mendelian factors, at least to begin by recognizing the *same* condition in different members of the family. The insanity in the paternal aunts, the deafness in the maternal grandfather's family and the epilepsy in the mother's family are, perhaps, evidence of diseases recurring within small related groups. But no recurrence of microcephalic imbecility is to be found in any relative. In the patient's sibship, although the cerebral paralysis in two members might be considered to be evidence of a mendelian recessive character, only one of these two affected children is an imbecile, the other having passed as mentally normal. Even if the cerebral paralysis is due to a factor of this kind, it does not explain the occurrence of the mental deficiency in the one and not in the other.

To account satisfactorily for such pedigrees we must assume that numerous genetic factors are involved and also probably environmental agencies. In order to establish the thesis that a recessive gene is present, causing the paralysis in the patient, a great many similar cases must be collected and the results analysed for numerical relationships between affected and normal persons. The only possible way of making use of the rest of the pedigree in the present state of our knowledge of human genetics is to apply statistical methods in a series of pedigrees containing similar conditions. Until this has been done on a large scale no one can say, for example, that a woman with an epileptic sister and mother, who marries a man whose aunts are insane, is more likely to have a microcephalic imbecile child than any other prospective mother taken at random.

CHAPTER VII

PERSONAL AND FAMILY HISTORY : ANALYTICAL METHODS

Ordinal Place in Family—Size of Family—Interpretation of Results —Example of Rigorous Analysis of Place in Family Data—Mendelian Ratios—Interpretation of Observed Ratios—Gene Frequency—Correlations.

Ordinal Place in Family.—We will now consider some of the various ways in which statistical use can be made of facts which the family histories of defectives reveal. First of all we shall discuss the relatively simple question of ascertaining whether, in a given disease, there is any peculiarity in the order of birth or position in family of affected individuals.

If all sibships of affected persons in a given series were of the same size, little difficulty would be experienced in detecting any deviation from random placing.

For example, suppose the following distribution of affected individuals as regards order were to occur in 5 families containing 3 sibs each, B representing a normal and A an affected individual :

Order of Birth.		5 Sibships.					Observed Totals.		Expected Totals.	
							A	B	A	B
1st		B	A	A	A	A	4	1	3	2
2nd		A	A	B	B	A	3	2	3	2
3rd		B	B	B	A	A	2	3	3	2
Totals	A	1	2	1	2	3	9	–	9	–
	B	2	1	2	1	0	–	6	–	6

The expectation is the same for all three ordinal places and is found by supposing the 9 A's and the 6 B's to be evenly distributed among the three possible places in the total.

In practice families of different sizes are always present in the data, and it is necessary to treat separately each group of families of the same size. The expected numbers of affected and normal individuals in each ordinal place are found in the

way just described. The families of different sizes are then pooled and the expectations in each ordinal place added together for comparison with the total numbers observed. Since the sum of the expectations for normal and affected is equal to the total number of children, it is only necessary to work out the expectations for the numbers of affected persons. The results given by this method are not vitiated if some of the families included are unfinished. A test for goodness of fit can be conveniently applied to the resulting comparison between expectation and observation by Pearson's χ^2 method.[1]

Now follows a worked example. The figures are taken from the results of a personal investigation into the family histories of 60 unselected idiots.

IDIOTS.

Ordinal position (r).	Size of Sibship (s)												Observed total.	Expected total.
	1	2	3	4	5	6	7	8	9	10	11	12		
1	3	4	6	5	4	3	1	—	—	—	—	—	26	17·8
2		5	1	3	—	—	—	2	—	—	—	—	11	14·8
3			3	1	1	—	1	—	—	—	—	—	6	10·3
4				3	1	—	—	—	—	—	—	—	4	7·0
5					2	1	1	—	—	—	—	—	4	4·0
6						—	1	2	—	—	—	—	3	2·4
7							1	1	—	1	1	—	4	1·7
8								1	—	—	—	—	1	1·0
9									—	—	—	—	0	0·3
10										—	—	—	0	0·3
11											—	1	1	0·2
12												—	0	0·1
Total (n_s)	3	9	10	12	8	4	5	6	0	1	1	1	60	59·9
(n_s/s)	3	4·5	3·3	3·0	1·6	0·7	0·7	0·7	0·0	0·1	0·1	0·1		

The method of obtaining the expected values can be summarized thus. If n_s be the number of children in sibships of s members and u the size of the largest sibship represented, then the expected number of r^{th} born is

$$\frac{n_r}{r}+\frac{n_{r+1}}{r+1}+\frac{n_{r+2}}{r+2}+\ \ldots\ +\frac{n_u}{u}.$$

In the example given, $u=12$, and the expected number of 5th born children is

$$1{\cdot}6+0{\cdot}7+0{\cdot}7+\ \ldots\ +0{\cdot}1.$$

[1] Fisher, R. A., *Statistical Methods for Research Workers*, 3rd ed., 1930, Oliver and Boyd, London.

The fit between observed and expected values is not good owing to the large excess of first born children and the probability of this excess being entirely due to chance sampling is low (about one in fifty).

Certain types of idiocy are apparently more likely to occur in first born children than in other ordinal family places. An example from general medicine, where primogeniture appears to have ætiological significance is congenital pyloric stenosis —a disease affecting infants in the first few weeks of life.

Further complications in the study of sibships from the point of view of order of birth are introduced by the inclusion of members of sibships whose condition, as regards the specific quality under investigation, is not ascertainable. This is unavoidable in practice whether the family investigation is well or badly done. Attempts to find out more about living members of a family are liable to disclose more potentially affected or unknown members. Remarriage of parents and twin births also introduce difficulties. The precise analysis of such material is more laborious than that described above. In a worked example, which follows shortly, I shall indicate how it can be attempted.

Size of Family.—Much information has been published concerning the size of the family in different pathological conditions, and some of the results are vitiated by a certain fallacy. In a set of sibships selected by the presence of at least one affected member, the crude average size is misleading and, in fact, invariably too large. This is because large families are more likely to contain affected persons than small ones, for a large group of persons is more likely to contain an individual with given characteristics than a small group. Methods of correcting for this have been devised by Greenwood and Yule.[1] The simplest proceeding advocated by them for estimating the true mean size of group of families employs substantially the same arguments as the method just described for finding the expected proportion of affected individuals in each place in family. It implies counting every sibship once for each of its affected members. The true average size of the family is the

[1] Greenwood, M. and Yule, G. Udny, On the Determination of Size of Family, etc. *Journal of Statistical Soc.*, 1914, Vol. LXXVII, p. 179.

reciprocal of the expected proportion of first born. For instance, the crude average size of the sibships in the table on p. 67 is 4·6 persons. But the corrected true mean size is 60/17·8 or 3·4 persons. Failure to apply this correction gives an erroneous impression of the fertility of the parents of defective children.

Interpretation of Results.—Even if a considerable discrepancy is found between observed and expected numbers in a " place in family " analysis it is not certain, or even very likely, that the discrepancy is actually due to causes really related to the ordinal position of affected persons. Genuine causes might be maternal exhaustion due to multiple parity, difficulty of birth of first born offspring or lack of nutrition at the end of a large family due to economic pressure. But the younger or older members of a sibship may be specially noticeable.

Selection of this kind quite possibly accounts for the apparent increase of intelligence with succeeding births observed by Thurstone and Jenkins.[1] These authors were concerned with brothers and sisters brought to a clinic for mental welfare. By a special method of analysis, they demonstrated that younger siblings were found with great regularity to be more intelligent than the older ones. But the family only began to be noticed when a mentally defective child was brought to the clinic. The remainder of the sibship, while no more likely to contain defectives than the previous portion, was probably the only part of the family to be studied carefully at the clinic. The authors also showed that I.Q. was liable to deteriorate as the child grew older, and the chances were in favour of defective children in the latter part of the sibship being noticed earlier in life than the first defective.

The possibilities of selection need very careful consideration before the order of birth, by itself, can be asserted to be a factor in the causation of a given abnormality. The study of a condition, such as insanity, where the incidence depends upon the age of the subject, is a case in point. If a given mental disease develops late in life, affected persons, at the time of their certification, are more likely to be found to have normal younger siblings than normal older siblings still living, for

[1] Thurstone, L. L. and Jenkins, R. L., *Order of Birth, etc.*, Chicago, 1931.

comparison of mentality. It is possible, however, to devise methods for eliminating such effects in calculating the expectations of affected individuals of different birth rank. In mongolism, for example, there is a well known tendency for the affected individuals to occur towards the end of the family. But maternal age is also found to be a factor of significance, elderly mothers being more likely to have offspring affected in this way than young mothers. The worked example which follows is given as a demonstration of how the effect of such a factor closely related to order of birth can be eliminated in finding the expected numbers of affected persons in each ordinal family place. The data are 150 sibships collected by the present writer in the course of his own investigations (see also pp. 103–105).

Example of Rigorous Analysis of Place in Family Data.—It is convenient to divide the calculation into a series of separate operations, the results of which are finally combined. The first sections (1, 2, 3) deal with the problem of determining the expected numbers of affected persons in each birth order when the data include members of sibships whose birth order is known but whose condition is unknown in respect of the quality which is being studied. It so happens that a considerable discrepancy is found on comparing the observed numbers with the expectations. There is, in fact, an observed excess of affected persons in all ordinal positions after the 5th born. The next part of the calculation is concerned with the estimation of the effect of variations in maternal age on the incidence of affected children. In the sections 4, 5 and 6, we find out how much the proportion of affected persons is likely to vary above or below the mean value in consequence of the different maternal ages which are found to be associated with different degrees of parity. The effect of maternal age alone increases the expected number of affected individuals towards the end of the family, from the 4th born onwards, and decreases it in the early ordinal positions. In section 7, the discrepancy previously discovered between the observed and expected numbers is shown to be equal to the variations in expected incidence due to the effect of maternal age, within a small margin of error.

1. Observe number of *known* persons (*s*) in each sibship and note exact position of each in respect to the mother's parity. The number of affected persons is termed *n*.

In one instance the sibship was composed as follows :—

Order of birth (parity).	Offspring.	Maternal age.
1	Stillbirth	
2	Stillbirth	
3	Normal female	(26)
4	Died in infancy at 2 months (female)	
5	Miscarriage	
6	Normal male	(33)
7	Normal female	(35)
8	Normal male	(36)
9	Affected male } twins Normal male }	(40)
10	Affected male	(45)

There are, here, 11 offspring, of whom 4 must be regarded as unknown; this leaves 7, and their order of birth is as follows :

3. Normal.
6. ,,
7. ,,
8. ,,
9. Normal and Affected.
10. Affected.

Thus, $s=7$ and $n=2$, though usually in the data for mongolism $n=1$.[1].

2. If there had been but one affected individual in the above sibship, the random chance of finding the affected person in any given position in family here would be 1/7 in each of the places, 3rd, 6th, 7th, 8th, 10th, and 2/7 in the 9th place because there are twins in this position. Since there are two affected persons in this sibship, the chance of finding an affected individual in any given place is doubled. Thus, for a given sibship, n/s is the random chance of finding an affected person in the ordinal position corresponding to each known individual.

The sum of all such values (n/s) in a series of sibships gives the total expectation for each ordinal place for the whole group. (Column (iv), Table D, p. 74.)

3. We next observe the actual numbers of affected children in each ordinal place in the whole group. (Column (iii), Table D.)

Clearly there is a wide discrepancy between the observed numbers and those expected from a random distribution in each place. Too

[1] If a woman marries twice and has affected children by the second husband and not by the first, the half-sibs of the affected child, whilst ranking as unknowns, affect the parity of later births.

few affected persons are found at the beginning of the sibships and too many at the end.

To what is this discrepancy due ? Is it due to the effect of parity or can it be due to the concurrent ageing of the mother ?

4. We next observe the age of the mother at the birth of each known child (normal and affected) in these families and, from the original data, we find the mean maternal age at the birth of the 1st born, 2nd born and so on. These mean ages are as given in Column (v), Table D.

5. The next step is to find the most probable incidence of affected members in these families at different maternal ages *regardless of birth order*. This is ascertained by the ordinary method of product moment correlation from the table given below, which is compiled from the same original data :—

DISTRIBUTION OF BIRTHS OF NORMAL AND AFFECTED OFFSPRING AT DIFFERENT MATERNAL AGES IN 150 SIBSHIPS.

Maternal age (*q*).	Normal (*b*).	Affected (*a*).
17	1	—
18	2	—
19	5	2
20	8	1
21	13	3
22	11	2
23	22	3
24	31	1
25	25	—
26	31	3
27	31	4
28	22	2
29	30	1
30	36	—
31	32	2
32	32	2
33	35	6
34	27	7
35	35	9
36	21	5
37	21	9
38	22	11
39	21	10
40	16	13
41	10	15
42	9	16
43	6	8
44	5	7
45	8	4
46	4	5
47	1	3
Totals . .	573	154

The regression equation, derived from the above distribution, which gives the most probable proportions of offspring affected (A) at each maternal age (q) is

$$A = 0{\cdot}0219q - 0{\cdot}499.$$

Now, A represents a ratio—the proportion of affected to total offspring in these sibships. To obtain the expected *number* of affected persons in any group we multiply A by the total number of persons in that group.

6. We next obtain the series of most probable values of proportional incidence of affected offspring, in each order of birth, by substituting the average maternal ages (q_1, q_2, etc.) from Column (v) in the above regression equation, *e.g.* $A_1 = 0{\cdot}0219q_1 - 0{\cdot}499$. We then multiply the values so obtained by the total numbers of children in each corresponding ordinal place given in Column (ii). The final results, A_1t_1, A_2t_2, etc., are shown in Column (vi).

It should be remembered that these expected numbers are based, firstly, on the most likely (or average) maternal age for a given ordinal place in family and, secondly, on the most likely proportional incidence associated with this particular maternal age. They are, therefore, quite independent of any observed place-in-family distribution of affected persons. They are also independent of the selection of sibships by at least one affected member. From them can be inferred the expected number of affected persons to be found in each position in family when the effect of maternal age on the incidence is the *only* factor under consideration.

The incidence of affected persons in the whole group ($\bar{a}$), that is, the ratio of affected to total children, is 154/727 or 0·212. If maternal age has no effect, and so long as we are not obliged to make any correction for the special selection of sibships by affected members, the incidence expected from random distribution in each ordinal family place will be the same as the total incidence (0·212). To obtain numerical values for this average incidence we multiply the ratio 0·212 by the totals given in Column (ii). The result is given in Column (vii). The expected *deviations* in the number of affected children due to the effect of maternal age in each ordinal place will be found in the difference between the figures derived from the regression equation (A_1t_1, A_2t_2, A_3t_3, etc.), and expected numbers derived from the mean value 0·212 ($\bar{a}t_1$, $\bar{a}t_2$, etc.). The resulting differences ($A_1t_1 - \bar{a}t_1$, etc.) are given in Column (viii).

7. We are now in a position to obtain a new set of values for the expected number of affected persons in each ordinal family place.

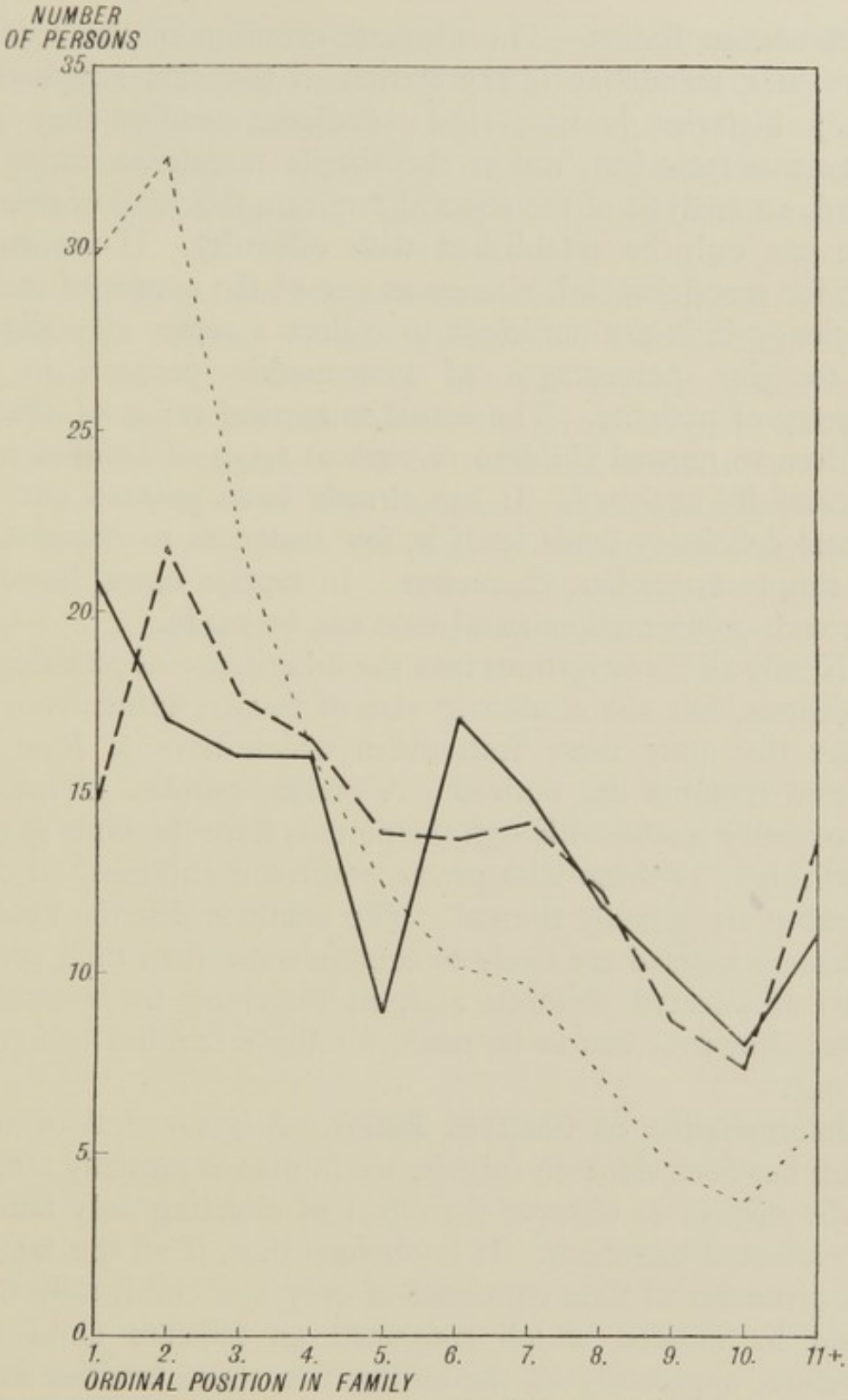
The first set of values, Column (iv), made allowance both for the observed distribution of affected persons in sibships of different sizes and for the gaps in these sibships due to omitting unknowns. We

then obtained, using the regression equation, a series of expected deviations (in the numbers of affected persons) which could be inferred from the observed effect of maternal age on the incidence of the condition in the offspring, Column (viii). Neither of these sets of expected values has assumed anything about the observed position in family of affected persons. These two sets of values, when added together, give the final estimate for the expected numbers of affected persons in each ordinal family place. The result of correcting the expected values, $\Sigma(n/s)$, by adding the expected deviations due to maternal age $[(A-\bar{a})t]$ is given in Column (ix). We now compare these new expected values with the original numbers observed, Column (iii). The factors used in correcting the expectations are just about sufficient to account for the observed distribution. As judged by the χ^2 test, the fit is to be regarded as very good. The supposition that the order of birth is, by itself, of ætiological importance here is therefore very unlikely to be true. (See Graph, p. 75.)

TABLE D.

(i)	(ii)	(iii)	(iv)	(v)	(vi)	(vii)	(viii)	(ix)
1	107	21	29·9	26·0	7·5	22·7	−15·2	14·7
2	118	17	32·5	28·3	14·2	25·0	−10·8	21·7
3	100	16	22·0	30·5	16·8	21·2	− 4·4	17·6
4	77	16	16·1	32·7	16·6	16·3	+ 0·3	16·4
5	73	9	12·4	33·5	17·0	15·5	+ 1·5	13·9
6	62	17	10·1	35·1	16·7	13·1	+ 3·6	13·7
7	55	15	9·7	36·2	16·1	11·7	+ 4·4	14·1
8	43	12	7·2	37·8	14·1	9·1	+ 5·0	12.2
9	29	10	4·6	38·9	10·2	6·1	+ 4·1	8·7
10	22	8	3·7	40·0	8·3	4·6	+ 3·7	7·4
11	12	4	1·3	39·6	4·4	2·5	+ 1·9	3·2
12	10	2	1·6	40·0	3·7	2·1	+ 1·6	3·2
13	9	2	1·6	43·3	4·0	1·9	+ 2·1	3·7
14	5	2	0·7	42·2	2·1	1·1	+ 1·0	1·7
15 and above	5	1	0·4	44·0	2·3	1·1	+ 1·2	1·6
Totals . . .	727	154	153·8	—	154·0	154·0	0·0	153·8

(i) Order of birth.
(ii) Total children (t).
(iii) Observed number affected (n).
(iv) Expected number affected : first estimate $\Sigma(n/s)$.
(v) Average maternal age in years (q).
(vi) $(A.t)$.
(vii) $(\bar{a}.t)$.
(viii) Correction to be applied to first estimate $[(A-\bar{a})t]$.
(ix) Expected number affected : final estimate $[\Sigma(n/s)+(A-\bar{a})t]$.



Observed number of affected children (Column (iii))

Expected number of affected children if position in family is distributed at random (uncorrected for effect of maternal age) . . . . . (Column (iv))

Expected number of affected children (corrected so as to eliminate effect of maternal age) . . . . . (Column (ix))

Mendelian Ratios.—The ultimate criterion of the applicability of a mendelian interpretation of the data supplied by family histories is numerical. Pedigree studies may give suggestive facts but, unless the simple mendelian ratios are found, on analysis of the affected families, this kind of inheritance can only be established with difficulty. If we are to look for mendelian inheritance as one of the causes of mental deficiency it is not sufficient to collect a series of pedigrees or to give percentages of neuropathic persons in the ancestry of patients. The actual numerical ratios of affected children to normal children in various types of families must be carefully analysed. It has already been pointed out that mental deficiency lends itself in few instances to examination for simple mendelian characters. In certain cases, however, thorough-going mathematical tests can be made.

Nearly all investigations into the inheritance of pathological conditions, like the studies in size of family, suffer from the defect that only those fraternities which have at least one affected member are noticed. A certain number of families are missed because, although the parents have the same genetic constitution as those who produce affected children, all their offspring are actually normal. The result is that the families which are noticed are likely to contain more than their proper share of affected children and, in searching for mendelian ratios, allowance has to be made for those families which are left out.

Interpretation of Observed Ratios.—A great deal of work which has been done on inheritance in man is partially vitiated by the neglect to observe the effect of selecting only families with affected members. It is obvious that, if all the families in a given set of data consisted of only one child, only those would be noticed which contained an affected child and, therefore, apparently all the children in those families would be affected. Similarly, the ratio of affected to normal can never be less than the reciprocal of the size of the family in the data. Since the average size of human families, in civilized countries, is of the order of four living children, the crude data collected about any condition would make it appear that the condition

presented itself in at least one out of every four children. Goddard's original data on the inheritance of mental deficiency require revision from this point of view, but his figures have been quoted, even as recently as 1932,[1] to prove that precise mendelian ratios occur in sibships containing feeble-minded children.

In order to analyse sibships selected in this way, the factorial method has least to be said against it. The assumption is made that the distribution of affected persons in different families of the same size follows the binomial principle and the result of the argument can be expressed as follows :

Where p is the probability that an offspring is affected in a given family and n_s is the number of s-membered fraternities each containing at least one affected member, the expected number of affected persons, termed r_s, has the value,

$$p.\sum n_s.\frac{s}{1-(1-p)^s}.$$

By use of expectations calculated from this formula, Sjögren,[2] dealing with juvenile amaurotic idiocy, has been able to show that the proportions of affected to normal in sibships containing affected members are very close to the proportions expected on the supposition that the disease affects a quarter of all children in such families. More recently, Slome has given an analysis of the infantile type of the same condition (amaurotic family idiocy) or Tay-Sachs disease. Since parents themselves are never affected, this analysis, together with certain other considerations, makes the likelihood of the condition's being due to a single recessive gene very high. The expected ratios were more closely approached in large families than in small ones and this can be explained by supposing that there is a certain bias in the selection of data. Families containing several affected persons are more likely to be noticed than those containing only one. In large families the effect of this bias is small.

Owing to the graded character of mental defect, examples of

[1] Berry, R. J. A. and Gordon, R. G., *op cit.*, p. 126.

[2] Sjögren, T., Die juvenile amaurotische Idiotie. *Hereditas*, Vol. XIV, p. 197, 1931.

sharply defined characters are not easy to discover, but there are other difficulties which also have to be considered. Environmental factors may prevent the true mendelian ratios from being realized and they may do so in two different ways.

Firstly, the affected individuals may be unfitted to survive in the average environment and they may, therefore, be very likely to be missed out in the process of collecting data. If the condition studied is sub-lethal the effect on numerical data may be serious, and it is necessary to devise some method of allowing for the way in which the relative mortality of normal and affected may alter the numerical ratios. If we are dealing with diseases such as congenital syphilis or any gross neurological abnormality, it is very important to get what information can be obtained about the members of the family who have died in infancy or perhaps even before birth. Neglecting to take account of these factors produces too low an estimate of the familial incidence of the condition which is being studied. The result may be to make a disease which is really hereditary appear sporadic.

The second way in which environment comes into the question is even more important. As Jennings [1] has pointed out, the final effect of every gene is only seen as the result of an interaction between heredity and environment. Although suggestive results can be obtained from the straightforward analysis of ratios of affected persons to normal in diseases like tuberculosis, what we are really studying is not the inheritance of the disease but the inheritance of susceptibility to the disease. According to Aschoff,[2] the intensity of an infective disease is the result of the interaction of factors which can be classified under the following headings : hereditary disposition ; acquired disposition[3] ; number of infective organisms and virulence of these organisms. There are also differences in local resistance to be considered. Heredity is only one of a

[1] Jennings, H. S., *The Biological Basis of Human Nature.* Faber and Faber, Ltd., London, 1930.

[2] Aschoff, L., *Pathologische Anatomie*, Bd. 1, S. 135, 1923. Fischer, Jena.

[3] For example, dogs are naturally immune from typhoid fever, but if first starved they can be infected.

great number of variables which has to be taken into account and the effect of heredity will only be clearly shown when all the factors in the environment, which are favourable to the production of the abnormality, are constant. In conditions other than bacterial diseases the presence or absence of chemical and physical agents are very important considerations. In dealing with mental disease, whether we are concerned with disorder or defect, but particularly in the study of the various types of insanity, great care must be taken to give full weight to the effects of mental environment. Undue optimism in the search for simple mendelian ratios when dealing with mental characters has led to great confusion, and the importance of environment will have to be properly considered before any exact knowledge can be obtained concerning the inheritance of the various forms of insanity and mental deficiency.

Gene Frequency.—Of recent years some important developments in the mathematical theory of mendelism have been made by extending the *principle of random mating* which was originally stated independently by Pearson and by Hardy.

The principle of random mating as applied to genes which are not borne on the sex chromosomes may be stated thus :

Let the frequency of a given gene in the general population be p. Since every chromosome is represented twice in the individual, three types of individual are possible,

(i) DD, possessing two of these genes, one on each chromosome,
(ii) DR, possessing only one of these genes,
(iii) RR, possessing no such gene.

If the gene is distributed at random in the population, the chance of its occurring in the same person twice will be p^2 ; the chance of its not occurring is $(1-p)^2$; and the chance of its occurring only once is $2(1-p)p$. It follows that, if the gene is rare, the heterozygous type (DR) will be very much more frequent than the homozygous type (DD). If both these genotypes are separately recognizable, the relative frequencies of the three types in a large sample of the population are distributed in the ratio $(1-p)^2 : 2(1-p)p : p^2$, for some value of p, so long as mating takes place entirely at random.

This principle provides us with a useful test for a given mendelian hypothesis.

For example, if mental deficiency were due to a single recessive gene present in homozygous form, and its incidence 8 per thousand, then the incidence of heterozygotes, or carriers, should be 163 per thousand. It might be suggested that dull persons were carriers of mental deficiency. If so, we should expect to find about one-sixth of the population of dull mentality. Lewis's estimate, however, of dull persons is one in ten, which is rather too low but of the right order of quantities.

One of the most important ways in which the theory of gene frequency may be utilized in human genetics concerns the correct interpretation of the incidence of consanguineous unions. It has been shown that if a recessive character is rare it will appear more frequently in the offspring of first cousin marriages than will a character not determined in this way.

The formula given by Lenz [1] is as follows :

If a is the incidence of first cousin marriages in the general population and p^2 the incidence of the recessively determined character, the incidence of first cousin marriages among parents with affected offspring is

$$\frac{a}{a+16p}.$$

In both the juvenile and infantile forms of amaurotic idiocy about 15 per cent. of affected individuals are the offspring of first cousins, whereas the incidence of first cousin marriages generally is less than 1 per cent. When we are dealing with relatively common conditions, such as mental deficiency, no significant excess of first cousin marriages would be expected even if it were determined by a single recessive gene. In groups of mental defectives there has, however, been frequently found a high incidence of consanguinity among the parents. In a series of 416 cases, whose families were investigated by the Research Department at the Royal Eastern Counties Institution, 12 instances of first cousin marriages in the parents were discovered. This ratio of 3 per cent. is definitely high. It is appropriate to the parents of persons whose condition is determined by a recessive gene where the

[1] Lenz, F., *Münch. med. Woch.*, Vol. LXVI, 1919.

frequency of such affected persons in the general community is only 1 per ten thousand instead of 8 per thousand. It may be, of course, that these mental defectives are taken from a class in the community where consanguineous marriages are relatively frequent. Another explanation is possible, namely that the high incidence of consanguinity found here is due to the presence of a few patients suffering from rare conditions determined by single recessive genes. If this were so the number of affected children should agree with the expected number calculated from the formula given on p. 77, even if more than one rare defect were present. The following table shows the analysis of 12 families in which mental defectives occur as the offspring of first cousins. For the purpose of this analysis, all the parents here are regarded as normal. While some of them may have been of poor mentality, they had escaped being regarded as mentally defective and were certainly of much greater intelligence than the affected offspring. The table is given simply as an example in the method of analysis where the sibships are selected by the presence of at least one affected member.

s Size of family (No. of known sibs).	n_s Number of families.	$s.n_s$ No. of sibs in these families.	Observed No. of sibs affected.	$\frac{s.n_s}{4} \cdot \frac{1}{(1-(\frac{3}{4})^s)}$ Expected No. affected.
1	3	3	3	3·00
2	1	2	1	1·14
3	1	3	1	1·55
4	3	12	3	4·39
5	1	5	2	1·64
6	1	6	2	1·82
7	—	—	—	0·00
8	1	8	1	2·22
9	—	—	—	0·00
10	1	10	4	2·65
Total	12	49	17	18·41

Although from the crude figures it seems that in these families 17 out of 49, or more than one-third, are affected, the number actually falls just short of the expected value, when allowance is made for the omission of sibships without affected

members. The correspondence between the observed numbers of affected children and the expectation on the recessive gene hypothesis is quite good.[1]

Correlations.—The product moment correlation coefficient has been widely used by Pearson to investigate the heritability of metrical characters in man. He has also used this method to study characters which had to be rated rather than measured, such as temperamental differences.[2] The results of examining correlations of a great number of characters as they appeared in parents and offspring, and in pairs of sibs, were almost uniform, all the coefficients having values close to +0·50. It has been shown by Fisher [3] that this result is theoretically to be expected if a metrical character like stature is determined by a great number of alternative genes, both dominant and recessive. Let us take an example of the use of correlation from mental deficiency.

In the course of research work at the Royal Eastern Counties Institution, the families of 416 patients were investigated: of these patients 101 were epileptic. A history of epilepsy in some relative or other was discovered in 23 instances or 23 per cent. of the cases. Among the remaining 315 non-epileptic cases, a similar history was obtained in 33 instances or 10 per cent. of the cases. Thus the result of the time-honoured method of noting neuropathy of a particular kind in *any* member of the family shows that the incidence of epilepsy is more than twice as common in families of epileptic patients than in the families of non-epileptic patients. It is possible, however, that a history of epilepsy is more likely to be sought out in a distant relative where the patient is epileptic than where he is not. This source of error is eliminated if we specialize on one particular relationship. By the application of the correlation method, an entirely different picture is

[1] A method of defining the limits of error of the expected values in such tables has been devised by Hogben, L., The Genetic Analysis of Familial Traits. *Journal of Genetics*, Vol. XXV, 1931.

[2] Cf. Pearson, K., On the Inheritance of Mental and Moral Characters in Man. *Biometrika*, Vol. III, p. 131, 1904.

[3] Fisher, R. A., The Correlation between Relatives on the Supposition of Mendelian Inheritance. *Trans. Roy. Soc. Edin.*, Vol. LII, p. 399, 1918.

obtained. Let us take all the 416 pairs of parent-child relationships and group them as shown here :

	Epileptic Parent.	Non-epileptic Parent.	Total.
Epileptic Children. .	4	198	202
Non-epileptic Children	11	619	630
Total	15	817	832

The correlation between the incidence of epilepsy in parent and child is seen at once to be exceedingly small and the result indicates that, in this sample, heredity is quite unimportant.

Unfortunately, the method of correlation is most suitable to the kind of data which are collected from a large number of pairs of individuals from different families in the general population. It is a less suitable method for dealing with data in which families are only selected if they possess some particular characteristic. The absolute value of the correlation coefficient in such cases is too low ; also it depends on the size of the sibships and on the gene frequency. The correlation technique for demonstrating hereditary likenesses is, in general, unsuitable for use in the study of mental deficiency since one is not dealing here with a random group of the population.

There are, however, special ways in which crude correlations arising from the data concerning mental deficiency can be useful. It has been pointed out that when sex-linked genes take part in the determination of a character the correlation coefficients between different types of pairs (*e.g.* father and son and mother and son) vary in magnitude. Even though the absolute magnitude of the coefficients obtained from data is not reliable the relative ordering of these various coefficients may remain unaltered. For example, with autosomal characters, the correlation father-son should be the same as father-daughter, but, when sex-linked characters alone are responsible, the former should vanish and the latter should be increased. This particular test for sex-linked characters is one which can be used in data even in which the paternity of some individuals

is in doubt, for there is no reason to suppose that males are more likely to be illegitimate than females. The correlations between the incidence of characters in different pairs of relatives can be used for detecting the presence of sex-linked genes in a variety of ways. The original papers on these subjects should be consulted by anyone wishing to attempt this type of analysis.[1]

Several writers have discussed the possibility of the presence of sex-linked genetic factors in the causation of mental deficiency.[2] The numerical excess of male defectives over female in certain surveys has led some persons to think their existence probable. At the present time, sufficient evidence is not available to decide the question one way or the other except in so far as a few conditions, known to be determined by sex-linked genes, are sometimes associated with mental deficiency. An example is pseudohypertrophic muscular dystrophy.

[1] Hogben, L., The Correlation of Relatives on the Supposition of Sex-linked Inheritance. *Journal of Genetics*, Dec., 1932.

[2] Cf. Rosanoff, A., A Sex-linked Factor in Mental Deficiency. *Amer. Journ. Psychiat.*, Sept., 1931.

CHAPTER VIII

CLASSIFICATION OF MENTAL DEFECT

Administrative Grouping—Congenital and Acquired Amentia—Primary and Secondary Amentia—Frequency of Primary and Secondary Cases—Subcultural and Pathological Amentia—Limitations and Advantages of Lewis's Classification—Social Environment, a Factor in Every Certified Case—Heredity, a Factor in Every Certified Case.

Administrative Grouping.—Since classification is the first stage in scientific knowledge, a discussion of the classification of mental defect is a necessary part of the task undertaken in this book. In Great Britain the legal definitions of idiots, imbeciles, feeble-minded and moral defectives, already discussed in a previous chapter, are of no scientific value. They are liable to lead to confusion in the minds of those who are not fully acquainted with their meaning. In fact, they are about as much use from the biological standpoint as a classification of aquatic organisms based upon their suitability for consumption as articles of human diet. In America more precise biometrical considerations have been suggested as criteria: persons of I.Q. 0 to 20 to be "idiots"; those from 20 to 50, "imbeciles"; and those above 50 and below 70, "morons." Similar scales were advocated recently in England for Education Act cases, in the Report on the Primary School Child. The very simplicity of metrical definitions may, however, be a snare. We are not at liberty to suppose that inability to pass certain tests necessarily implies failure in social adjustment, although it may make this prognosis probable. Conversely, a few persons of intelligence of normal standard, or even better than average, as judged by tests, have to be certified under the Acts on account of persistent anti-social conduct because they seem to be quite incapable of the exercise of what is usually called "common sense."

Any grouping of defectives based on administrative necessity is open to the objection that it neglects the biological and pathological sides of the question. To remedy this omission many supplementary schemes have been used or suggested. I shall review a number of such schemes, bearing in mind that the objects of a method of classification are also, from the scientific point of view, strictly utilitarian. A satisfactory scheme should help to further our knowledge as well as sum up what we already know, and it should cover every case.

Congenital and Acquired Amentia.—The earliest attempts at scientifically useful classification of mental defect were based on the division of diseases commonly accepted in clinical medicine into the congenital and acquired groups. These terms are usually considered to refer to ailments caused by events which occur respectively before and after the birth of an individual. Injuries occurring during the process of birth have sometimes been put into the one and sometimes the other category. In mental deficiency, the position was at one time complicated by certain French physicians who used the term idiocy to signify congenital and imbecility to signify acquired defect : the current Italian usage is the reverse of this. In 1877 Ireland [1] proposed a grouping based on pathological considerations and gave the name " genetous idiocy " to the cases which he considered to be determined by heredity, among whom he included mongols. The remaining patients were named under a variety of different headings such as " microcephalic idiocy," " epileptic idiocy," and " paralytic idiocy." For the next few decades the separation of cases into different clinical types continued as new diseases were recognized. It thus became the fashion in any given case to say first to what type it belonged, *e.g.* microcephalic, and then to say whether the type belonged to the congenital or acquired group of diseases, as in general medicine. Some authorities, however, prefer to sort out all the types, without going further into the question of ætiology. For instance, Lapage examined 904 candidates for admission to

[1] Ireland, W. W., *Idiocy and Imbecility*. J. and A. Churchill, London, 1877.

Manchester Special Schools and found that only 114 could be classified as belonging to special types. This left 790, or 87 per cent., belonging to no special types. Obviously, simple enumeration of defectives of different clinical types does not lead us very far. To be really useful, a classification must take into account the origins of the various types.

Primary and Secondary Amentia.—The system in common use to-day is due to Tredgold who, in 1908, introduced the terms Primary and Secondary, instead of congenital and acquired, and gave them a very special meaning. Primary amentia is due to defective developmental potentiality in the germ cell. Secondary amentia is due to arrest of cerebral development by external factors acting after fertilization.

Tredgold's system was put forward before the effects of modern genetic research were realized. While rejecting the simple view that, when hereditary, mental deficiency is due to a single recessive gene, he retains the belief that it is a manifestation of the *neuropathic diathesis* or constitution which is transmitted from parent to offspring in the form of some special pathological germinal material. This hypothetical substance is inherited in some direct, but not very regular, manner from parent to offspring, rather like the inheritance of personal property. The pathological material is supposed to come into being by the action of various kinds of poisons, bacterial or pharmacological, on the germ plasm. However, it is not essential, from the point of view of Tredgold's classification, that the precise method of genetic determination should be specified. So long as we are clear that the type of case under consideration is definitely of hereditary origin it can be classified as primary. Its mode of transmission can be made the subject of separate and subsequent enquiry.

The classification of defectives into these primary and secondary groups has been very widely adopted both in this country and abroad and has now become the traditional method. The extreme simplicity of the dichotomy is attractive on account of the apparent ease of its application and its wide scope. It is a very practical method from the medical point of view. The relatives of patients frequently enquire of the

physician what is the cause of the illness. Having pronounced judgment on the case as being either secondary or primary amentia, the physician can either explain to the relatives that the disease is a pure accident and that there is no fear of its appearing again in the family or warn them against having more children and support propaganda for sterilization.

There are many conventions which are useful in medical practice but which are not necessarily of value to science. For example, a diagnosis of influenza or neurasthenia frequently represents a convenient fiction covering ignorance. A systematic study of bacteriology or psychopathology reveals under these labels a multitude of diverse conditions. The semblance of separating out the tangle of mental deficiency to uncomplicated strands, which is a notable feature of the current terminology, is further emphasized by the introduction of the term Simple Primary Amentia to cover a type of case very frequently met with and included under primary amentia. This type is characterized by the absence of any special distinguishing features—beyond the occasional presence of stigmata of degeneration, which admittedly occur also among mentally normal persons. If, therefore, clinical examination of the patient gives no clue whatever to the cause of the condition, the classification is *simple* primary amentia.

Frequency of Primary and Secondary Cases.—From the point of view of both prevention and administration, it is desirable to know the relative number of cases which fall into any particular descriptive category. Tredgold's [1] most recent estimate is that 80 per cent. of patients are primary and 20 per cent. secondary. Shrubsall and Williams found 7,124, out of 10,000 defective schoolchildren, to be cases of primary amentia, excluding those special types, mongols, cretins and microcephalics, which Tredgold regards as primary. If these had been included, the proportion of primary aments would have been raised to 78 per cent.—a figure close to Tredgold's estimate.

In Denmark recently, Larsen [2] classified 1,000 institutional defectives according to whether they belonged to the endogenous

[1] Tredgold, A. F., *Mental Deficiency*, 5th ed., 1929, p. 65.
[2] Larsen, E. J., *Acta Psychiat. et Neurologica*, Vol. VI, 1931.

or exogenous group. These terms closely correspond to primary and secondary, but do not carry any implication of belief in the existence of neuropathic diathesis. Larsen excluded a few definitely psychotic patients, together with eleven cases of chronic epidemic encephalitis and one or two neurological cases, from his thousand defectives. He found that 762 patients had endogenous mental deficiency. Among these were included 63 mongols, 28 microcephalics and 24 endocrine dystrophies. The figure of 76·2 per cent. is only a trifle lower than the English estimates. The bulk of the primary or endogenous defectives (in Larsen's data) were derived from a nondescript group labelled " mental deficiency in which there appears to be an hereditary disposition," but hereditary disposition was only actually found in less than half these cases, and the majority of them must have been selected mainly on the same negative grounds that have been laid down for the recognition of simple primary amentia.

When we know nothing about a defective patient, except that he is mentally subnormal, to assume that his condition is entirely the effect of heredity may prove to be an unsatisfactory start in the work of scientific classification. It would be equally unsatisfactory to prejudice the result by supposing that these cases, about which nothing is known, were due to adverse environment only. After careful individual examination, we may be able to argue by analogy from other cases where the ætiology is fairly well established and give an opinion on the causation, although this opinion may have a low probability of being correct. Even so, the possibility of the application of the antithesis of primary and secondary amentia is extremely limited. In a series of 513 institutional cases which I examined physically and mentally, and whose family histories were enquired into, I was only able to apply the terms primary or secondary to about one third. The result was, nevertheless, of some interest (Table E, page 90).

It will be seen that over 60 per cent. of the cases were unclassifiable either as primary or as secondary aments, because the effects of environment and heredity were so very difficult to extricate from one another.

TABLE E.

I.Q.	Primary aments.	Secondary aments.	Unclassifiable aments.	Total.
75–99	2	3	25	30
50–74	71	11	69	151
25–49	44	11	101	156
0–24	20	22	134	176
Total	137	47	329	513
Per cent.	29	9	62	100

The difficulty of classification by this method is felt more when dealing with very high or very low grades than with the medium grade defectives. In the very high grades nearly all cases are certified on account of behaviour difficulties which are often engendered by environmental influences working on a slow intellect. In the low grades we often find it quite impossible to estimate whether the patient would or would not have been normal but for a physical disease or disability. We also meet with cases where hereditary disposition is coupled with a specific environmental accident, which can be pre-natal, as perhaps occurs in mongolism.

In both low and high grades a slight preponderance of secondary causes may be met with. Among the medium grades probably heredity plays a large but not exclusive part. Taking together only those cases where primary or secondary amentia is diagnosed, we find that, out of 184 cases, 137 are primary, *i.e.* 69 per cent. are hereditarily determined. This figure is close to the estimates already referred to for all defectives. We could argue that the remaining 327 should be divided in similar proportions, if we knew nothing whatever about them. But such an argument would be fallacious, for we do know something about them. We have indeed set aside many of them on account of the probable existence of both environmental and hereditary effects. It is worthy of note that when Tredgold supplied his evidence to the Royal Commission in 1905, although he believed 90 per cent. of defectives to be the result of heredity, he admitted that 55 per cent. included in this group had physical diseases similar to those which, in the

10 per cent. of secondary aments, were considered to be of causal significance. The net result, therefore, of this method of classification is merely to indicate wide limits for the possibility of eradicating defect by altering either environment or heredity. By subsuming the unclassifiables in Table E to the secondary or primary categories in turn, we obtain the limiting values that somewhere between 9 per cent. and 71 per cent. of the cases are preventable by altering environment and on this basis it is logical to say that 91 per cent. of cases are due in some measure to heredity and 71 per cent. are due in some measure to environment.

Subcultural and Pathological Amentia.—Let us now consider further possible methods of classification. In a paper originally read at the British Association in 1931, Lewis suggests a new dichotomy.[1] He uses the analogy of mental ability with physical height. The heights of all persons in the community will form a roughly normal frequency distribution curve and so will the intelligence quotients. Lewis believes, however, that, outside a certain range, it is scarcely possible to describe as normal the individuals having these extreme measurements. We find, in fact, on examination of such cases, that a large number show gross pathological characteristics in the ordinary medical sense of the term. Thus, taking the example of physical height, neither the achondroplasic dwarf nor the rachitic dwarf can be regarded as a variant of the normal. Similarly, in mental deficiency, many idiots and imbeciles will be found, such as microcephalic or paralytic cases, who cannot be looked upon as normal variants, although persons of small physical or mental stature, but otherwise healthy, can be looked on as normal. Lewis, therefore, points out that mental defectives are roughly divisible into two groups: one represents the lower part of the normal frequency distribution curve of intelligence, and its members are termed *subcultural;* the other group is formed by pathological cases. One great value of this attempt at classification is that it lays emphasis on the fact that a very large number of those persons who are labelled mentally defective are

[1] Lewis, E. O., *Journal of Mental Science*, April, 1933.

really just as much part of the normal population as people who are more intelligent than their fellows. In an investigation carried out in Sweden by Jaederholm, a complete survey was made of the intelligence of all children of a given age (including defectives) in a given district. In this particular study, the resulting frequency distribution curve was in close agreement with the Gaussian error curve. According to Burt, however, the presence of mental defectives in the population tends to make the curve slightly skew. A slight, but definite, heaping of the curve near the extreme lower end corresponds to the lower grade defectives. Lewis attributes the asymmetry to the fact that, in many cases, mental deficiency is caused by environmental influences.

Limitations and Advantages of Lewis's Classification.—It is possible to regard the intelligence of *all* defectives as more or less extreme variations from the normal. We must therefore limit the subcultural class in some way, if it is to be of practical value. Lewis's own method is to make a theoretical distinction between variations from the mean due to normal and abnormal causes. Subcultural amentia is then defined as a variant from the mean intelligence due to normal causes. It is a *physiological* as opposed to a pathological condition. It is convenient, in practice, to apply the term *pure* subcultural to those patients who are reasonably healthy and not obviously abnormal physically, and who are not mentally deranged. For example, we should exclude mongols and epileptics.

It frequently happens that a person who would in any case have been a subcultural mental defective is afflicted with a disease such as congenital syphilis, epilepsy, mongolism, encephalitis lethargica, or even some hereditary neurological condition. Mixed cases of these kinds form such a large proportion of defectives that it is important to be able clearly to define the subcultural element which forms part of the diagnosis on which they are to be classified. A patient can be classified as subcultural if his mental grade is lower than would be expected if he were judged only by a medical examination.

It is important also to be clear about the difference between

the description of a certain group of defectives as subcultural and the theory of simple primary amentia. The definition of a person as subcultural does not imply that his disability is hereditary. It may be found by research that the normal variability in intelligence has causes which are chiefly environmental and is due to mental nutrition in a way similar to that in which the normal variability in height could be supposed chiefly to be due to physical nutrition and affected by climate, habits, etc. Although the subcultural group may eventually turn out to be mainly of hereditary origin, the question is still left open. In primary amentia the question is closed as soon as the label is applied.

When we come to use, in practice, the dichotomy advocated by Lewis, we find that one difficulty arises, which is the same as that which arose in the application of other methods. Many cases cannot be put into one or other category definitely but belong really to both. I, personally, therefore prefer to retain the concept of the subcultural type for purely descriptive use, to refer to a certain class of cases, but not to use it as a strict alternative to pathological types, as the two classes overlap. The numerical sizes of the two categories among institutional cases, in my opinion, are about equal; nearly three-quarters of the patients can be classified under each heading, so that in nearly half the cases the categories overlap. Lewis, however, regards the subcultural type as much more numerous than the pathological groups in the general population and he holds that the majority of the feeble-minded are subcultural.

Social Environment, a Factor in Every Certified Case.—While it may be possible to make a rough simple descriptive classification of mental defectives which takes into account the probable causation in each case, obviously it is very unlikely that any precise scientific categories will ever be found to describe exactly those individuals who are certifiably mentally defective within the meaning of the Acts. The Acts are framed for administrative and not for scientific purposes. Strictly speaking, an environmental element enters into the description of every case actually certified, since the necessity for certification depends upon social conditions. In homes where there is no

economic necessity, low grade cases can be cared for and trained without recourse to institutional facilities : quiet medium grade cases live comfortably, and often usefully, with their relatives. Persons who have private means committing minor offences against the law are able to get good legal advice and to pay fines instead of going to prison. They are much less likely to be certified as moral defectives than are their counterparts without private means at their disposal. Subcultural mental deficiency is not confined to any social class. Each class contains all grades of mental ability, although there is doubtless a general tendency for the more intelligent to improve their social status. We see here that there is a considerable difference between subcultural mental deficiency and the class of persons defined as the *social problem* group in the Wood Report. The latter term carries with it implications of low social and economic status. It may be true that most of the cases of the subcultural type requiring certification merit such a description, but it should always be remembered that environmental factors have usually been instrumental in bringing such people to the notice of the authorities. These social factors may be quite arbitrary and, from the point of view of human biology, largely irrelevant.

Heredity, a Factor in Every Certified Case.—Looking at the question from the opposite point of view, wherever we meet with what is apparently an environmental effect in a given individual, we can never exclude the possibility that hereditary disposition or susceptibility to such effects may exist. It is, therefore, quite possible that hereditary factors enter significantly into every case of mental deficiency. We have already seen that environmental determinants are involved in nearly every case also. If this be indeed true the attempt to classify mental defectives as primary or secondary, congenital or acquired is foredoomed to failure. The only remaining possibility is to sort out as far as possible all the pure clinical types which occur (subcultural, mongol, etc.). These types form the basis of classification and it is then necessary to determine whether any given ament is an example of a pure type or a mixture of more than one.

The accurate scientific classification of *all* mental defect, under the same scheme, is a practical impossibility unless such a scheme is indefinitely complicated. But, with the help of the concept of subcultural mentality, a reasonable number of defectives may be conveniently grouped both for scientific and administrative expediency. Methodologically, the only satisfactory way is to group cases together first from the clinical medical point of view, judging each case on its own merits, and to leave open the question of causation to subsequent research. We must bear in mind all the time that every case is to some extent secondary and to some extent primary if these terms are interpreted in their strictest senses.

CHAPTER IX

PATHOLOGICAL TYPES : MONGOLISM

Description—The Problem of Definition—Ætiology—Mechanism of Causation—Illustrative Case.

Description.—One of the most interesting conditions to be met with in the study of mental disease and, indeed, one of the outstanding oddities of general medicine, is the condition which has been named " mongolism." An enormous amount of attention has been devoted to this abnormality in recent years. In a discussion a few years ago at the Royal Society of Medicine, a speaker referred to our ignorance of its causes as a disgrace to the medical profession. Without going quite so far one may point out that it is unlikely that much headway will be made in the discovery of causes of mental deficiency in general until a satisfactory analysis of mongolism is made.

Although the condition was first named by Langdon-Down,[1] it had undoubtedly been observed long before this and the patients were regarded as a special type of cretin, sometimes thought to be due to tuberculosis in the parents. The supposed likeness of their features to the Chinese and other orientals led Langdon-Down to propose an ethnic classification of idiots. The classification made no headway because an insufficient number of convincing types of idiots were discovered which might be supposed to belong to other ethnic groups, *e.g.* Ethiopian. But the designation of the Mongolian or Kalmuck type has remained, and recently the hypothesis of their descent from Mongol ancestors has been much stressed by Crookshank.[2] This writer has, moreover, added that, in his view both the racial Mongolians and the so-called mongolian

[1] Langdon-Down, J., Observations on an Ethnic Classification of Idiots. *Clinical Lectures and Reports, London Hospital*, 1866 (iii), pp. 259–62.

[2] Crookshank, F. G., *The Mongol in our Midst*, 3rd ed., Kegan Paul, London, 1931.

type of idiots are closely allied to the orang-utan. By various analogies he explains that the abnormalities to be found in " mongolian " idiots indicate that they are of the nature of a regression to earlier ancestral types. He explains the condition as due to conflict between parental ancestral strains. He would have them called, for preference, *imbecile mongoloids*. At the other extreme, it has been suggested by Tredgold that no such thing as mongolian or kalmuck idiocy exists. We have in these cases, perhaps, merely a conglomeration of characters which can occur, either separately or in groups, associated with other types of mental defect. Let us now consider what are the characters by which the mongolian imbecile is recognized.[1]

The first and most striking feature of the mongol is his resemblance to others of the same type. So close is the superficial likeness that it is difficult to believe they are not all members of one family, and to see and examine one is sufficient to obtain a very good idea of the characteristics of the type.

The cranium is invariably small,[2] free from bossing or eminences, and usually presents a flattening of the occiput which, however, is more apparent than real ; to a lesser extent this flattening is evident in the face. With few exceptions the mongols are brachycephalic and the cephalic index is high. The long axes of the palpebral fissures are oblique and slope downwards and inwards. In infancy there is usually a marked development of the epicanthic fold, part of the skin of the upper eyelid partially covering the inner cushion or canthus of the eye ; but, according to Crookshank, this is less characteristic than the obliquely-placed fold of loose skin—the Mongolian fold—situated above the upper lid and most marked at its inner angle. Even when the epicanthic fold is not present, the palpebral fissures are usually smaller than normal. The eyelids are thin and frequently become affected by various inflammatory conditions, with consequent shedding of the

[1] The description following is based on that given by Dr. R. M. Stewart, *Proceedings of the Royal Society of Medicine*, Vol. XIX, 1926. Section of Psychiatry, pp. 11–25.

[2] A recent account of the morphology and histology of the brain has been given by Davidoff, L. M., *Arch. of Neur. and Psychiat*, Dec., 1928, p. 1229.

lashes. The eyes are often small, and may be unduly prominent. The iris often shows a curious speckled arrangement of the anterior yellow pigment, to which Langdon-Down first drew attention. Ptosis, nystagmus, and eye-rolling are common, and, according to Brushfield, strabismus is present in some degree in every case. Congenital lamellar cataract sometimes occurs.

The bones of the face have a tendency to flatness, the prominences and hollows being less evident than in the normal child. There is hypoplasia of the orbital and nasal bones.[1] The face is sometimes described as moon-shaped, and the prominent cheek-bones, so characteristic of the racial Mongol, are almost never seen. The nose is generally short, squat, with a depressed bridge which may recall that of the congenital syphilitic child. The nostrils look forward as well as downwards, and are often narrowed or triangular in shape. Nasal catarrh is common. The ears are generally small, rounded, simply convoluted, and the lobule is defective. The maxilla is under-developed, particularly in its anterior portion, and the nasopharyngeal cavity small and shallow. The palate is apparently high and narrow : this almost universal effect being due to the thickened mucosa of the gums. The lower jaw is more rounded and more prominent than in the normal child. The teeth are late in making their appearance, especially the upper central incisors ; they are irregular and prone to early decay.

The tongue often protrudes from the mouth and may be abnormally large, although its protrusion more often depends on the smallness of the oral cavity. It is said to be normal at birth, but by the third or fourth year of life it has acquired numerous transverse fissures which become even more marked in succeeding years. This fissured, or scrotal, tongue, as the French term it, may be regarded as one of the most characteristic signs of mongolism, but it is an error to assume that it is found in no other type of ament. Stewart has, for example, recorded four typical examples in defectives who showed no other signs even remotely suggestive of mongolism.

[1] Grieg, D. M., *Edinburgh Medical Journal*, May, 1927.

Curiously enough, three of these patients were the victims of congenital syphilis. Occasionally fissuring of the lips is also seen, and the lower lip not uncommonly protrudes. The cheeks of those who reach childhood are rosy and plump, the skin of other parts of the body being coarse and covered in certain situations with a growth of downy hair. On the scalp the hair is coarse, wiry and sparse. The mucous membranes are peculiarly liable to chronic catarrh and adenoids are very frequent.

The hands are remarkably soft and flabby, the digits thick and clumsy-looking, the thumb and little finger short; the latter is often abnormally curved towards the medial line, and sometimes one transverse crease is lacking. The palms show various departures from the normal arrangement of lines. A characteristic feature is a marked transverse line running straight across the palm of the hand. The feet are likewise short, broad, clumsy. There is also frequently a large cleft between the second toe and the hallux: webbing of the fingers or toes is not an uncommon finding. In some cases the sole presents a well-defined line running towards the heel from the first interdigital cleft. In stature the mongol is usually stunted, with a straight spine, a relatively narrow long trunk and short limbs. The abdomen resembles that of the cretin in being enlarged so that it protrudes beyond the level of the chest when the mongol is recumbent. Umbilical hernia is common. Owing to the laxity of the ligaments there is an unusual mobility of the joints, especially those of the digits, wrists, ankles and hips. When seated, the young mongolian imbecile usually disposes his lower limbs in the horizontal plane, in other words, he " sits cross-legged like a Turk." The genital organs in the male are sometimes very much smaller than normal.

The mental characteristics of the mongol show a certain parallelism with the physical, and although many degrees of mental defect may be found, the majority tend to remain imbeciles or idiots. Speech is usually guttural and indistinct: in some cases it is never acquired. A mental age of 7 years is to be regarded as the upper limit of their intelligence. As a rule they are lively, imitative, and often have a love of music which is

rather striking. In disposition they are usually, but not invariably, good-tempered and, as Sutherland remarks, the " smiling face of the mongolian imbecile suggests the possession of a secret source of joy." Lastly, they appear to have an almost complete immunity from true epilepsy, though fainting attacks occur owing to defective circulatory system to which reference has already been made (Chapter II, p. 16).

The Problem of Definition.—The characters which have been enumerated show great variability from one patient to another, and, while it may be true that all mongols superficially look alike, in detail multiple differences are to be observed between any two cases. For example, the hair may be curly, the hands long and thin, the palpebral fissures not oblique, the cephalic index low and yet the diagnosis be sustained on grounds of the presence of many characteristic features. It is, possibly, these considerations which have led such an authority as Tredgold to doubt the existence of a real type. Many of the characters are, moreover, inconstant in the same individual, varying with age and health. The epicanthic fold fades with age, as it does also when present in normal individuals ; the head grows longer (see p. 23), the tongue tends to grow more deeply fissured and inflammation of the eyelids may set in as age advances ; baldness and obesity may become marked features not present in early life.

Some of the characters, like the malformed ears and other stigmata of degeneration, are somewhat difficult to identify. But if a group of fairly constant and characteristic phenomena are taken, and the frequency of the association of the phenomena in mongols be compared with the frequency of their association among other defectives, we see at once a striking difference. This fact makes it almost impossible to deny that their association in mongolism is of significance. To demonstrate the point, let us examine a number of patients diagnosed as mongols and a random group of a number of other certified mental defectives for the frequency of certain characters.

The seven characters chosen here are all fairly easy to observe : other characters would have served the same purpose.

A. Intelligence quotient between 15 and 29 inclusive, *i.e.* the most likely range of intelligence for mongols.

B. Cephalic index 0·83 or higher.

C. Epicanthic fold on either eye.

D. Fissured tongue.

E. Conjunctivitis at time of examination.

F. Transverse palmar line on either hand.

G. One crease only on minimal digit of either hand.

The incidence of these characters (given as frequency per 50) in two groups of defectives are as follows :—

	A	B	C	D	E	F	G
50 Mongols	35	22	26	37	15	22	9
350 Unselected cases .	12·9	8·7	1·7	3·6	0·7	1·9	0·1

It will be observed that some of the features are much more diagnostic than others ; the first two, A and B, being less characteristic of mongolism than the rest. If we now note the number of these features present in any given patient, we find that two quite distinct frequency distributions are obtained for the two groups of cases, thus :—

Number of characters present . .	0	1	2	3	4	5	6	7
Number of mongols with a given number of characters . . .	1	2	10	15	14	4	4	0
Number of unselected patients with a given number of characters .	188	124	32	5	1	0	0	0

Almost three quarters of the mongols have three or more of these characters, but only six out of 350 unselected patients have three or more. Any defective with four or more of these characters is almost certainly a mongol.

An interesting feature of the occurrence of these characters is that, amongst the mongols, they are practically uncorrelated with one another. For example, in these 50 cases, the number with both D and G was 6 and the number with both B and F was 10. The expected number of random agreements for these pairs of characters would be 6·7 and 9·7 respectively. But if the whole group of 400 defectives is considered, the number of associations of these relatively rare characters, occurring mostly among the mongols, is grossly in excess of expectation. In fact, in the whole group the characters are significantly correlated with one another. We have here a system which

is similar in certain respects to that studied by Spearman in mental testing. But, in the present instance, the entity we are looking for is mongolism and not "g." It seems to be indicated that, in the group of mongols, each member possesses the entity "mongolism," and that the characters studied may be present or absent in a given individual according to chance circumstances. The specially frequent occurrence of the characters in members of this group is not due to chance but due to the members of this group being in some way radically different from the rest of mental defectives. We must, however, admit that transitional cases do occur where it is not easy or even, in the present state of our knowledge, possible, to decide whether the patient should be classified as a mongol or not.

Ætiology.—The abnormality is always congenital, that is, present from birth. No one has ever observed any tendency for a child born normal to become a mongolian imbecile or even to grow more mongoloid with age, as in deformity due to glandular dystrophies like hyperthyroidism or acromegaly. Whatever circumstances produce the condition must occur before birth. There is some evidence for believing that the foundations are probably laid not later than the eighth week of pregnancy. The resulting picture is one of retarded development both mentally and physically. Many of the characteristic signs can be regarded as remnants of fœtal existence; the epicanthic fold, the round head, the delayed dentition, the short, stunted limbs and the scanty hair. The name "fœtalism" could well be applied to the condition and this would be a more characteristic name than "mongolism." The peculiar temperament of the affected persons, their secret source of joy, may be akin to the sort of happiness which the fœtus might be supposed to experience in its blissful intra-uterine surroundings.

It is sometimes stated that males are more frequently found to be affected than females. This is true as far as institutional cases are concerned, and it seems likely to be due to the males being more troublesome at home than the females. Certainly, when cases are investigated outside institutions, no

significant excess of males over females is noticeable. This was proved by Van der Scheer [1] in a large series of cases in Holland. The same writer was able to give an estimate of the absolute incidence of the condition in the general population. He ascertained definitely some 500 cases in the whole of Holland with a population of 8 million. These figures can be compared with the Eastern Counties of England, where nearly 200 have been ascertained in a population of 2 million. Thus the crude absolute incidence would appear to be of the order of 1/10,000. We have, however, to correct this minimal estimate by making allowance for failure to ascertain, which will be specially marked in young cases, and the relatively high mortality of affected persons. Judging by the mortality of these cases in institutions, their death rate is, at all ages, something like nine times the general average for the population at large and nearly six times that of all other types of defectives taken together. Frequent causes of death are acute tuberculosis—which may be of the miliary type—acute specific fevers, pneumonia and meningitis. The real incidence of the condition in the number of live births affected must be considered to be more of the order of one per thousand, in England, though it varies from district to district. Social class has no effect on the incidence.

By far the most important fact which has been discovered in connection with the ætiology of mongolism is the relation of maternal age to its incidence. This connection expresses itself in three ways. Firstly, the average age of the mother at the birth of these patients is significantly higher than the average age of mothers at births of normal children in the same families or in a large sample of the general population. The latter comparison has been made by Van der Scheer and the following figures exemplify the former comparison.

150 Sibships containing each at least One Mongolian Imbecile.

Average age of mother at birth of	573 normal children	31·2 years.
,, ,, ,,	154 mongols	37·2 ,,

[1] Van der Scheer, W. M., Beiträge zur Kenntnis der Mongoloiden Missbildung. *Abh. a. d. Neurologie, Psychiatrie, Psychologie u. ihren Grenzgebieten*, Heft 41, 1927, Berlin.

The difference is 6·0 years and has a standard error of only 0·6 years so that there can be little doubt as to its statistical significance.

Secondly, the age of the father has a similar relationship to the incidence of the condition, though the effect is not so marked. Thus, in the same series of sibships, the following figures were obtained.

Average age of father at birth of	573 normal children	33·8 years.
,, ,, ,,	154 mongols	39·4 ,,

The difference here is 5·6 years, but with a standard error of 0·8 it is still very significant.

Thirdly, the position in family of mongolian imbeciles is characteristic: over half are last-born. The average ordinal position in family of 154 mongols, in the series of families already described (p. 74), was 5·51. The expected average position in family of 154 children distributed at random, in the same series of sibships, worked out according to methods already described, was $4{\cdot}05 \pm 2{\cdot}96$. This expected value differs from the observed average position in family of the mongols by 1·46 places, with a standard error of 0·24.[1] As it stands this would seem to show that position in family is a factor to be considered in the ætiology of mongolism. It is not, however, likely to be a causal factor, because the effect has been shown earlier (pp. 70–75) to be accounted for entirely by the concurrently increasing mother's age with the lengthening of the family. It is, however, possible that primogeniture may be a causal agent: more than the expected number of first-born mongols are found, but in the series already described the excess is not significant. The interval between the birth of the mongol and the birth immediately preceding has been shown by Van der Scheer to be longer than usual: this finding is explicable on the grounds that the maternal age and not the place in family is the significant ætiological factor. Fantham [2] takes the view that too frequent pregnancies are the cause of mongolism, but his evidence for this hypothesis is not convincing.

[1] *I.e.*, $2{\cdot}96/\sqrt{154} = 0{\cdot}24$.

[2] Fantham, H. B., *South African Journal of Science*, Vol. XXII, 1925.

Returning to the second point, the effect of paternal age can be studied quite simply by taking groups of children born at or near the same maternal age and observing the corresponding paternal age at the births of mongols and normals. The accompanying table shows how this works out. There is no evidence that paternal age by itself has a significant effect.

THE EFFECT OF PATERNAL AGE IN MONGOLISM.

Maternal age in years.	No. of normal children.	No. of mongols.	Av. paternal age for normals (Pn).	Av. paternal age for mongols (Pm).	Difference between av. pat. ages (Pn − Pm).
Below 25	93	12	26·1	24·6	+1·5
25 to 29	139	10	29·5	28·2	+1·3
30 to 34	162	17	34·0	35·3	−1·3
35 to 39	120	44	39·2	40·0	−0·8
40 to 44	46	59	43·7	42·9	+0·8
45 and over	13	12	48·4	49·7	−1·3
All ages	573	154	33·8	39·4	−5·6

The average paternal ages for mongols and normals are very close for constant maternal age groups, although judged by total number of mongols and normals born at *all* maternal ages the difference between these average paternal ages is large.

Mechanism of Causation.—Having established that the mother's age at the patient's birth is a significant ætiological factor, the next step is to consider in what way this factor may operate. A natural assumption to make is that the effect is primarily a nutritional one. The ageing of the maternal tissues, particularly the walls of the uterus, tends to deprive the embryo of its necessary nourishment in the early stages of development. This failure might be manifested in the way Van der Scheer suggests by producing an inelastic amnionic sac and preventing the embryo from obtaining its full extension. It is, however, difficult to imagine that, if the ageing of the tissues were the sole cause, normal children would be born subsequent to the birth of a mongol, and this is a common finding. I know of one instance where seven normal children followed the birth of a mongolian imbecile. There seems, therefore, to be a likelihood that the environmental factor, whatever it is, may be operative in one pregnancy but not in

the next, but that it is more likely to occur when the mother is elderly. The chief effect of age in most tissues is evinced by the gradual deterioration of the blood supply owing to the hardening of arteries. Hence one would look for an explanation of the disease to a cause of vascular origin.

The corpus luteum in the ovary is considered to be essential for the continuance of pregnancy, but, towards the end of this period, it undergoes degeneration by the occurrence of multiple hæmorrhages. If the corpus luteum degenerates early in pregnancy, abortion is supposed to follow. It is possible that slight hæmorrhages may occur very early, insufficient to destroy the whole structure but serious enough to diminish the potency of its secretions which, in the early stages, determine the embedding of the embryo in the uterine wall. Such hæmorrhages might be deemed more likely to occur in more elderly women. As further evidence for this view it may be mentioned that premature labour is common with mongolian imbeciles and a history of bleeding during pregnancy or threatened abortion is not infrequently obtained. Miscarriages and stillbirths are also common in sibships where mongolism occurs. There are, of course, innumerable other possibilities which might be considered, and the problem cannot be solved until the hormonic and other influences, which determine the condition of the uterus during pregnancy, are fully understood.

Strong arguments have been brought together by Stewart suggesting that a cause of mongolism is to be looked for in the poisons produced by the spirochæte of syphilis. Although the Wassermann reaction is usually negative in these cases, luetic curves are found by means of the colloidal gold test in the cerebrospinal fluid. The clinical picture of congenital syphilis is, in a number of ways, not unlike that of mongolism, and in some cases mistakes are made in diagnosis on this account. In the only recorded example of four cases occurring in the same fraternity, the father had signs of syphilis.[1] The fact that twin births may occur, with one child affected and the other normal, is no objection to the syphilitic theory, since congenital

[1] Babonneix, L. and Villette, J., *Archives de Médicine des Enfants*, Vol. XIX, 1916, pp. 478–81.

syphilis has been recorded in one of twins on a number of occasions. There are, however, a great number of cases of mongolism in which this explanation seems unsatisfactory because syphilis in the parents can be excluded with a fairly high degree of probability. It seems more likely that syphilis in the mother may produce the same effect as age. It is possible that other toxic factors, and even the emotional state of the mother during the early weeks of pregnancy, can precipitate an occurrence accidentally which, owing to the ageing of the maternal tissues alone, may happen in a like manner in their absence.

There is evidence, however, that mongolism is in some degree determined genetically. Apart from the fairly frequent occurrence of twins, one of whom is affected and the other normal, there are two kinds of evidence for this view to be considered. Firstly, the occurrence of more than one member of a fraternity affected with mongolism is not so very uncommon and is seen about once in every fifty families studied, provided these are carefully examined. Giving due weight to the tendency of these cases to occur towards the end of the family, this incidence must be regarded as greater than the random expectation. The disease occurs with a like frequency (one in fifty cases) in children who are first cousins. Pedigrees have been recorded where two or more generations have exhibited the disease. Attempts, however, to describe the results on a straightforward mendelian hypothesis have so far been either hopelessly vague or manifestly incorrect.

The other type of evidence for heredity in mongolism is that the parents or other relatives of these cases sometimes exhibit characters which are considered to be part of the clinical picture of the disease: for example, shortness of stature, straightness of hair, high cephalic index and transverse line on the palm of the hand. There is a field here for a great deal of study, and it is, of course, first of all necessary to be sure that these characters are more frequent in the relatives of mongols than in the general population.

Supposing that there is some genetic element in the ætiology of mongolism, how are we to understand the effect of maternal age? Fortunately, biology has provided us with a

prototype to study. Wright [1] has shown that certain characters in the guinea-pig (polydactyly and coat colour) are affected by the age of the dam. To study this problem he first of all produced an inbred stock, whose members could all be considered to have a very similar genetic constitution. He then studied, among these animals, the relative effects of parental age and place in family on the incidence of these characters in the offspring. His conclusions showed that, at any rate with regard to polydactyly, the order of birth and the age of the sire were not significant factors. The parallel with mongolism is so close that the disease can no longer be considered a biological mystery.[2]

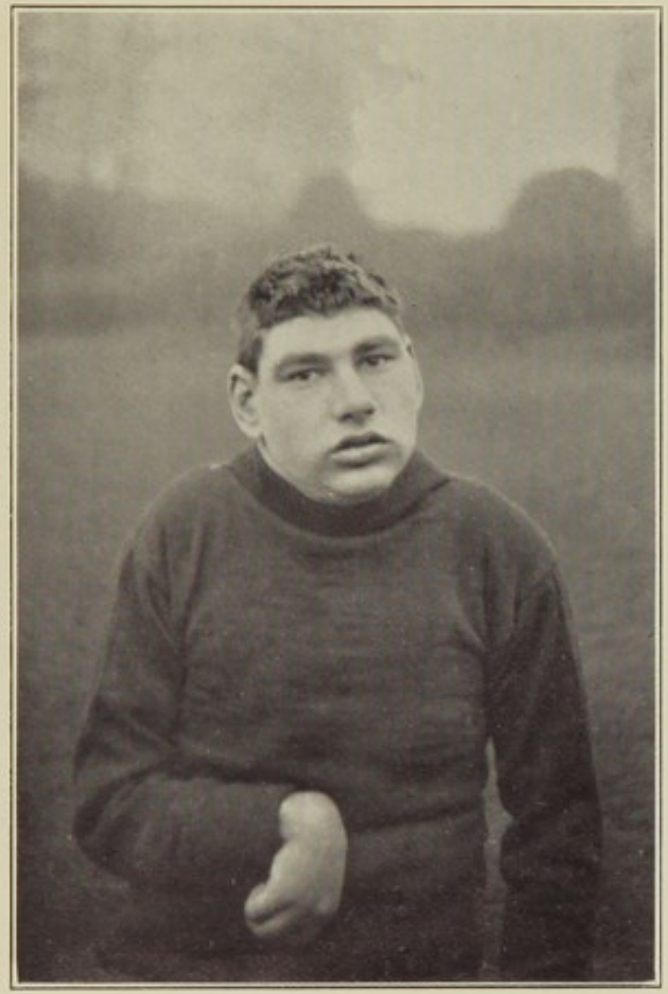
Illustrative Case.—The following case history exemplifies a few of the points we have been considering in the chapter.

The photograph opposite is of a girl aged 11 years who is last born in a sibship of 13 members. The mother was 47 at her birth and the father 55. The family are of the labouring class. One brother (the third born) is feeble-minded : his mental age is 8 years. His general appearance is somewhat reminiscent of mongolism, though no one would suggest the diagnosis of mongolian imbecility. Two siblings died in infancy, there was also one stillbirth and one miscarriage.

The patient herself is very small for her age, but not ill-proportioned ; the hands are long and thin. Her hair is straight and sparse, epicanthus and strabismus are marked. The tongue and lips are fissured ; the teeth are irregular and primitive ; the palate appears very narrow. The bridge of the nose is flattened. Head circumference 17½ inches : cephalic index 0·80 : cranial capacity 800–900 c.c. : mental age 3 years : I.Q. 27 : Blood Wassermann reaction strongly positive in repeated tests. Her character is friendly and cheerful and her talent for mimicry is a source of great amusement both to patients and staff. One of her favourite games is to nurse her foot like a doll. Her general health is poor and she is very liable to attacks of bronchitis.

There is no serological evidence of congenital syphilis in the feeble-minded brother, but at his age, 32, the test is very unreliable. The girl is the only case out of 50 mongolian imbeciles in the Royal Eastern Counties Institution in whom definite evidence of congenital syphilis has been obtained. Although atypical from several points of view the case is undoubtedly correctly diagnosed as belonging to the mongolian type and illustrates well the difficulties of definition.

[1] Wright, S., *American Naturalist*, Vol. LX, 1926, pp. 552–9.
[2] Cf. Penrose, L. S., *Journ. Genetics*, Vol. XXV, 1932, pp. 407–22.



Imbecile : Microcephaly. (See p. 61.)

Imbecile : Mongolism. (See p. 108.)

[*To face p.* 108

CHAPTER X

PATHOLOGICAL TYPES : BIRTH INJURY

Traumatic Amentia in General—Birth Trauma—Congenital Athetosis—Ætiology—Mentality of the Birth-injured.

Traumatic Amentia in General.—If the fundamental cause of amentia is lack of a properly functioning cerebral apparatus, it follows that any injury which is sufficient to cause gross destruction or impairment of the brain at an early age will cause mental deficiency. It has not often been seriously doubted that head injuries in early childhood, followed by signs of intra-cranial hæmorrhage, may in rare cases be responsible for mental deficiency—usually of a severe grade. Tredgold and Goddard, for example, are both quite clearly in agreement on this point, and cases of amentia are to be found where there is a perfectly straightforward history supporting the diagnosis. The frequency of these straightforward cases is about one per two hundred or three hundred institutional mental defectives. A child, for instance, appears to be developing normally until the age of 2 or 3. It learns to walk and talk at the right time and in the usual way : the parents, being sensible people, having other children, would notice if anything were wrong. Then suddenly an accident occurs : the child falls, striking its head against a stone floor. It seems dazed and doctors are called in. They diagnose concussion, but the child recovers slowly and limbs are found to be paralysed. It soon becomes evident that intra-cranial hæmorrhage, perhaps meningeal, perhaps subarachnoid, has occurred. After this the child's mind does not develop properly. It may even regress, and epileptic fits, mild or severe, sometimes continue at intervals throughout life. To obtain such a history as this is a rare occurrence in mental deficiency work, but when it is obtained the circumstances are so clear

and characteristic that there is no reason to doubt the genuineness of the observed sequence of cause and effect.

There are a great many cases where the history of an injury is given and where it cannot be recorded as the main or even as a contributory factor in the causation of the amentia. Slight inconsistencies of dates and in the description of the accident by different witnesses, abnormalities recorded in the previous history of the child or the obvious desire of the parent to attribute the deficiency to some cause other than heredity, may lead the physician to suspect the trustworthiness of the supposed cause. He should seek for evidence of petit mal or convulsions occurring before the accident. Cerebral hæmorrhage is uncommon in infants, but may occur without head injury in consequence of vascular degeneration due to congenital syphilis, and this possibility especially should be borne in mind. In no field of mental deficiency work are accurate personal histories so important as in the study of traumatic amentia.

Birth Trauma.—In the largest group of patients whose disability is apparently due to trauma, the injury is supposed to have occurred at birth. Arguments in favour of this view are based on analogy with those patients whose disability seems to be due to an injury in childhood. Unfortunately there are weaknesses in the arguments when they are applied to birth injury. We can have little evidence of the individual's having been normal previous to the injury. A mongol may suffer from birth injury, but this is, nevertheless, not the cause of mongolism.

Certain evidence may be useful though not conclusive, such as a knowledge of the normal onset of quickening, giving indications of a normal pregnancy and a normal fœtus. The act of birth is a complicated series of events, and what is termed *difficult labour* is considered from the point of view of the mother and the medical attendant or midwife rather than from the point of view of the child. Ehrenfest [1] has laid stress on the danger to the child of prematurity. The blood vessels in the fœtal brain are fragile in premature deliveries and the mere

[1] Ehrenfest, H., *Birth Injuries of the Child*, D. Appleton, New York, 1931.

congestion produced by alterations of pressure during birth may be sufficient to cause severe hæmorrhages. The indiscriminate use of obstetrical forceps, to ease labour for the mother by accelerating it, may also sometimes be injurious to the child's head. In these cases, from the maternal point of view, the labour may not be really difficult.

It seems that careful examination reveals that almost every child is intra-cranially birth-injured to some degree—even if this degree is very slight. To observe the symptoms of such injury, the condition of the child must be investigated immediately after birth, and related to its size, maturity and the difficulty of its birth. Nystagmus, retinal hæmorrhage, pallor, a weak cry, inability to make sucking movements of the lips—these are some of the signs to be looked for. A quite large percentage of children after apparently normal births show one or more of these signs, from which they afterwards make a complete and rapid recovery. It is not surprising that statistics so far available of the mentality of children, classified as having easy or difficult labour, should give little definite information as to the effect of injury at birth on mentality. Statistics of this kind are almost valueless unless a careful examination of each child is made immediately after its birth and the observations related to the history of pregnancy and delivery. Dayton's large series of 20,473 cases [1] showed no correlations between the mental grade and abnormal labour, but his standards of abnormal labour were rough.

Congenital Athetosis.—On the question of actual physical appearances of disease in the child or adult which can have been caused by birth injury, medical science needs enlightenment. There are, in particular, two neurological diseases which have both been, in the past, attributed to birth injury and are both often classified under the name Little's disease. Patients of one group have a spastic cerebral paralysis with all the straightforward clinical signs of a lesion in the pyramidal tracts. Typically, the legs are worse affected than the arms. A

[1] Dayton, N. A., Abnormal Labor as an Etiological Factor in Mental Deficiency and other Associated Conditions. *Proc. of the Fifty-fourth Annual Session of the American Assn. for the Study of the Feebleminded*, 1930.

"scissor-leg" posture tends to develop and is associated with deformities of the feet (talipes equinovarus). The lesion is bilateral, but often not quite symmetrical. The other group of patients suffer from a condition sometimes known as congenital athetosis. The clinical picture is very characteristic. The outstanding feature is the squirming (athetoid) movements of the limbs and face accompanying voluntary movement. These contortions are due to associated stimulation of antagonistic or otherwise related muscles when a voluntary movement is made: the neural impulses tend to spread and the mechanism for inhibiting the spread of these impulses is impaired. The lesion causing athetosis may, according to Wilson, be either seated at the cortical level of the cerebrum or in the mid-brain. Many patients who suffer from congenital athetosis do not show the typical signs of a lesion of the pyramidal tracts. Although deep reflexes are brisk, plantar responses are usually flexor. The rapidity of the deep reflexes seems to be due to an increased muscle tonus, for superficial reflexes, such as the abdominals, are usually very brisk also. It seems likely that these athetoid patients are often suffering primarily from an extra-pyramidal mid-brain lesion.

The mentality of these patients with congenital athetosis is, even in severe cases, superior to that of the former group of patients with equally severe pyramidal lesions. Their physical disability is great, but often their methods of thought are clear and they are capable of performing manual operations with unexpected skill. They are by no means always mentally defective. The severity of the disease and the opportunities for education will vary from individual to individual, but so will the quality of the brain matter still functional. The disease may be present even in people well above the average intelligence.

It is possible for these two types of cerebral diplegia to be mixed in the same individual, and sometimes a subject of congenital athetosis may have one or more limbs showing the typical signs of paralysis of cortical origin. In these cases sometimes large portions of the cortex are found to be undeveloped or atrophied (microgyria, porencephaly). Care

must be taken to exclude syphilis and other inflammatory conditions as causative factors in these cases. No fundamental distinction between paralysis with or without athetosis is made in a recent study by Doll, Phelps and Melcher.[1] Their definition of birth injury includes traumata which may have affected the developing fœtus towards the end of pregnancy, and thus they leave open the question of what is the precise origin of these conditions.

Ætiology.—In the current medical textbooks it is usually stated that there is considerable doubt whether what is known as Little's disease can be due to birth injury since so many of the patients affected have no history of difficult birth. When we are dealing with those persons who, in addition, have mental impairment, the problem is complicated by our desire to know the cause of this mental disability. As with other diseases which may or may not be associated with mental deficiency, there are really two distinct questions to be answered. In the present instance the questions are as follows. (*a*) Is birth injury the cause of the lesion in the nervous system ? (*b*) Is the lesion in the nervous system the cause of the mental deficiency ? In order to attempt to answer these questions in so far as they concern birth injury it is first advisable to distinguish as clearly as possible the two neurological conditions which have been described above and consider their ætiology separately.

Among 500 institutional mental defectives, I succeeded in discovering 8 cases of spastic diplegia of the first type with a history of difficult labour sufficiently suggestive to make the diagnosis of birth injury probable. The blood of none of these 8 patients gave, at any time, a positive result in a test for syphilis and there was no history of any cerebral injury during childhood. The paralysis dated in all cases from very early life, but was not consistently noticed at, or shortly after, birth. Five of these 8 patients were first born children. There were also among the 500 institutional cases, 6 typical examples of congenital athetosis and in none of them was a

[1] Doll, E. A., Phelps, W. M., and Melcher, R. T., *Mental Deficiency due to Birth Injuries*. The Macmillan Company, New York, 1932.

history of abnormally difficult labour obtained. In no instance was there any indication of an inflammatory cerebral condition. Only one of the 6 was first in the family and there seemed to be a slight tendency for them to be born at late maternal ages. The importance of primogeniture in difficult labour is well known. In Dayton's statistical study, referred to on p. 111, a marked association was found between primogeniture and abnormal labour. The frequency of primogeniture in the former group of patients is a further aid to their diagnosis and tends to separate them from the latter group. I think it possible that the typical athetoid patients form a distinct group, the causation of whose infirmity is intra-cranial hæmorrhage occurring *in utero*. This hypothetical disaster may be due partly to some initial developmental abnormality in the formation of the blood vessels. If so, the causation may be ultimately determined by genetic factors. There is no evidence of familial incidence of this condition, though the familial incidence of some other types of cerebral diplegia has been authentically recorded. In the present state of our knowledge, this disease must be regarded as of unknown origin.

Mentality of the Birth-Injured.—In order to determine how far birth injury is responsible for mental impairment, one can but judge by a comparison with the examples of traumatic amentia where the child has been ascertained to have been normal before the accident. In the athetoid type of case the condition resembles, in some respects, amentia by deprivation : here the motor disability is the most marked feature and is often quite sufficient to deprive the patient of ordinary facilities for education. One can sometimes conjecture that the intelligence would have been quite normal, had the motor mechanisms been intact, by observing the general mode of behaviour. Doll maintains that, in mental defectives who are birth-injured, the clinical psychological picture is characteristic, irrespective of their exact neurological type. These patients, he says, improve much more in performance ability with training than do other defectives. The evidence for this view is perhaps not yet strong enough to make it

certain whether these are the only cases in which this phenomenon may be observed. It may be common to other paralytic diseases with different causation. Doll expresses the opinion that birth injury may result in mental retardation or disturbances of personality without conspicuous motor symptoms. The cases he actually describes tend, however, to show that the personalities of birth-injured defectives are, as a rule, well integrated and that mental disorders (as opposed to deficiencies) are rare in such cases. It is interesting to observe in this connection the beliefs of Rank,[1] who considers that the psychological trauma experienced by the child at birth is responsible for many neuroses in later life. Few psychologists agree with Rank on this point, but some support for his view may be considered to have been rendered by Dayton's statistical findings. In the large series of children already referred to, a significant positive association was found between emotional instability and abnormal labour.

[1] Rank, O., *The Trauma of Birth*. Kegan Paul, London, 1929.

CHAPTER XI

PATHOLOGICAL TYPES : CEREBRAL INFLAMMATION

General Considerations — Encephalitis Lethargica — Congenital Syphilis—Juvenile General Paralysis.

General Considerations.—Mental deficiency is not infrequently associated with brain destruction due to the action of toxins of pathogenic microorganisms and the attendant inflammatory conditions. The two most important diseases to be so described are amentia due to encephalitis lethargica and syphilitic amentia. There are a great number of other types of cerebral inflammation which may affect children afterwards showing signs of amentia, but, on the whole, they are less frequent or less easy to diagnose than these two types. By the time the mental retardation is investigated, the causative agent can often only be identified by guesswork unless a very full history is obtainable. Clear examples of the after effects of such diseases as polioencephalitis and cerebrospinal meningitis can sometimes be found. As compared with other diseases, poliomyelitis is not an important cause of mental retardation : this is demonstrated by the following percentages.

CHILDREN IN CRIPPLE SCHOOLS (490 CASES). [1]

Clinical Diagnosis.	Intelligence.			
	Above average.	Average.	Below average.	Nearly defective.
Infantile paralysis (Anterior poliomyelitis) .	18·1	48·7	25·6	7·5
Infantile Hemiplegia . . .	8·8	32·4	33·9	27·9
Cerebral Diplegia	0·0	15·1	24·2	60·6

[1] Williams, A. C., Combined Defect. *Mental Welfare*, Vol. VII, No. 4, 1926.

Encephalitis lethargica (epidemic encephalitis) merits special attention on account of its fairly clear symptomatology and its relatively late onset. It is a condition which provides an opportunity for studying the effect of brain disease on intelligence with accuracy. The problems it presents are similar to those encountered in traumatic amentia. If a disease occurs late we can have good evidence of the child's mental condition before its onset. In congenital syphilis and other inflammatory diseases beginning very early in life, or even *in utero*, the question of the origin of the amentia has to be settled by analogy with similar diseases whose onset is late.

Encephalitis Lethargica.—About two per cent. of institutional defectives have suffered from encephalitis lethargica. The incidence is much higher than would be expected from the consideration of the random occurrence of the disease in the general population. The frequency of these cases in institutions is largely due to behaviour which makes them unmanageable at home or in hospitals.[1]

The clinical condition following on encephalitis lethargica in children is somewhat different from the effect in the adult and it has received full description from numerous medical writers. The Parkinsonian syndrome is not so common in juvenile cases as in adults, but mental changes are commoner. Frequent and characteristic signs of the disease in mental defectives are disturbances of sleep, optic palsies, oculogyric crises and pituitary dystrophy. Almost all post-encephalitic patients tend to put on weight. Their tendon reflexes are brisk but their movements are slow.

The mental condition of these patients is of great interest. Their intelligence, on the whole, is of higher grade than the average institutional cases. It has been clearly shown, however, that not only is their intelligence, as judged by mental tests, diminished by the disease, but that a progressive deterioration frequently takes place. The following table

[1] Mental sequelae are noticed in 70 per cent. of encephalitis lethargica cases by Buzzard, E. F., Riddock, G., and others, *B. M. J.*, 1923 (2), p. 1083. Duncan, A. G., *Brain*, 1924, Vol. XLVII, p. 76, found mental changes in 72 per cent.

summarizing some of the findings of Dawson and Conn [1] is of general interest in this respect. These writers also studied in detail the unaffected siblings of encephalitic children and found that the level of their intelligence was normal.[2]

COMPARATIVE MEAN I.Q. OF PATIENTS, IN THE DIFFERENT DISEASE GROUPS, ATTENDING THE ROYAL HOSPITAL FOR SICK CHILDREN, GLASGOW.

Clinical Diagnosis.	Number of Cases.	Mean I.Q.	P.E. of mean.
Chorea	137	90·8	±0·66
Encephalitis lethargica . . .	53	83·9	±1·20
Epilepsy	49	80·6	±1·43

The average I.Q. of 1,020 unselected patients was 90·27±0·31.

Emotionally, in these patients, there is an increase of anti-social tendencies, which probably were present in not abnormal proportions before the onset of the disease. An increase of personal vanity and a childish desire always to be in the limelight may become evident. The patients may be very easily upset if a momentary whim is thwarted. Sometimes they steal and frequently become unconventional in their sexual behaviour when previously they have shown no unusual tendencies in these directions. Most characteristic of all, when it occurs, is the loss of control over fleeting impulses. These impulses may express themselves in the form of biting, scratching, slapping or throwing objects at people. The persons attacked are often those with whom the patient is on the best of terms. In the middle of a friendly conversation an attack may be launched with amazing suddenness. The patients themselves are quite unable to say what makes them do these things and they are usually sorry for having done them. Some patients even take steps to prevent themselves from getting into situations where they know these reactions

[1] Dawson, S., and Conn, J. C. M., Intelligence and Disease. *Medical Research Council, Special Report Series No.* 162, H.M. Stationery Office, 1931.

[2] Dawson, S., and Conn, J. C. M., Effect of Encephalitis Lethargica on the Intelligence of Children. *Arch. Diseases of Children*, Vol. I, 1926, p. 357.

are liable to occur. For example, a girl patient used to keep her hands behind her apron because she knew that if they were in front of it, they would scratch the attendant's face. Sometimes it seems that such patients are hardly conscious afterwards that anything has happened. The disease appears to destroy certain connections between the emotional centres in the brain and the conscious, controlling apparatus.

The personal history of every encephalitic ament is of great significance and the first step to be taken, in investigation, is to discover what evidence there is of the child's having, at any time, reached a normal standard. Sometimes mental tests have been carried out on such children previous to their illness, but, even failing this, an early school report may give quite clear indications that deterioration has occurred. Not all the defectives who have encephalitis are found to have previously reached normal standards. If they have been dull or troublesome before their illness, it may sometimes be difficult to be sure whether they would not have been mentally defective in any case. At all events the dullness and the encephalitis are likely to produce a lower grade of mental deficiency than either would have produced separately.

In encephalitis lethargica, the final clinical picture is usually distinct from other encephalitic conditions. The actual history of the illness, however, is sometimes very puzzling and it is often difficult to make out exactly when the infection first began. Occasionally acute specific fevers, such as measles or scarlet fever, are the prelude to a condition which is diagnosed as encephalitis. Sometimes encephalitis is a manifestation of the acute specific fever. It is found, however, that encephalitis, caused by whooping-cough, mumps, etc., is hardly ever followed by mental deficiency. Possibly, when mental retardation is a sequel, the fever has lowered the natural resistance to the virus of encephalitis. This pathogenic agent is probably very widely distributed and may remain dormant in the mouth or nasopharynx of a healthy person.

It is exceedingly rare to find more than one person in the same family affected by encephalitis lethargica, but we cannot, on this account alone, rule out the possibility of an inborn

susceptibility in some individuals. This possibility is one which has to be borne in mind in the study of all infective diseases. There seems, however, no justification for supposing that such an inherited susceptibility is necessarily associated with any kind of mental weakness or disorder.

Congenital Syphilis.—Congenital syphilis is a disease which infects the child *in utero*. The spirochætes may attack the brain causing a meningo-encephalitis, the result being mental deficiency. It was formerly believed that syphilis in the father might be transmitted to the offspring without affecting the mother, but more recent researches have shown this to be highly improbable, the infection coming direct from the mother in all cases. In a stillborn child suffering from congenital syphilis, spirochætes can be found in almost every organ of the body. There is, therefore, no doubt that it is a genuine infective process and not a condition of diminished vitality due to the presence of poisons in the maternal blood or in the germ plasm.

The frequency of congenital syphilis among institutional mental defectives, as judged by the Wassermann reaction, on the average is about 10 per cent. If we were to judge the incidence by the clinical signs of congenital syphilis in the patients, the figure would be much lower. It is somewhat unfortunate that medical textbooks emphasize signs which are rare and it is held by some authorities that these signs are less commonly met with nowadays than when they were first described. Discussing this subject, Stewart [1] writes :

> There is, I think, no doubt that congenital syphilis has undergone considerable modification in type since the beginning of this century, possibly because the infection is of a milder type. The more usual clinical signs such as rhagades, Hutchinsonian teeth and eye changes, form a clinical picture which is only seldom met with, and statistics based on the presence or absence of the classical signs will lead to a very erroneous idea of the incidence of the syphilitic taint. In a series of 173 cases showing a positive Wassermann, I was only successful in finding two who presented the classical triad of bilateral eighth-nerve deafness, keratitis and notched incisors. Stigmata of congenital syphilis may indeed be entirely absent. On the other hand, in late

[1] Stewart, R. M., The Secondary Forms of Mental Deficiency. *Post-Graduate Medical Journal*, December, 1928.

congenital syphilis the Wassermann reaction may be negative, although the patient presents obtrusive signs of the disease. In looking for evidence of syphilitic infection, much information of diagnostic value can often be obtained from an examination of the larger joints. Thickening and broadening of the bony structures in their neighbourhood is very suggestive of congenital syphilis, particularly in the case of the elbow-joint.

Paralysis is very frequently associated with congenital syphilis and may affect one or more limbs.[1] Epilepsy is even more common. The mental enfeeblement which accompanies the disease may be of any degree, though most cases exhibiting paralysis are idiots or imbeciles. These people are often, however, like the traumatic cases, liable to be more sensible than one would suppose them to be, judging merely by their test ability. Sometimes congenital syphilis is associated with blindness due to ophthalmia neonatorum. Some degree of sense deprivation exists through deafness or partial blindness in a great many cases of congenital syphilis. In such persons the element of sense deprivation adds to the mental impairment.

Owing to the multiplicity and diffuseness of the lesions caused by congenital syphilis, the neurological and mental signs assume many different forms. Developmental anomalies, such as anencephaly, porencephaly and microcephaly, have been attributed to this cause and cases have also been reported in which there is no paralysis but merely a numerical deficiency of cortical nerve cells. Frequently, however, when syphilis affects the nervous system of the fœtus, it does so in an unmistakable manner, the pathological changes being of a gross character easily discernible to the naked eye. The lesion most often found is of the meningo-vascular type, and though the brain may sometimes show marked damage in one particular area, multiple diffuse affections are more usual.

The personal history of a patient suffering from congenital syphilis can be very characteristic and may take the following form. The child appears normal at birth but somewhat under weight. It remains rather small and appears anæmic.

[1] For a classification of these palsies, see Harris, H., *Mental Welfare*, 1930, Vol. XI, pp. 93–96.

Nothing else is noticed until perhaps the age of one year, when it is suddenly found that a limb is paralysed or that one eye deviates outwards. After this the paralysis may grow steadily worse and may be associated with epileptic fits, mental retardation becoming fairly obvious. Such cases are distinguishable from traumatic amentia by the lack of history of an injury and from encephalitis by the lack of history of infectious disease at the time of onset. They are distinguishable from both kinds of amentia by the history of debility from birth. If the blood Wassermann reaction is taken in early childhood, in such cases, it will almost certainly be positive : in adults the history becomes an important consideration in the diagnosis because the Wassermann test is usually negative, or inconclusive as in the results already described (pp. 37–40).

The family history may give important clues to the diagnosis. A knowledge that the mother or father has been treated for syphilis is useful and it is sometimes possible to get Wassermann reactions done on the blood of the parents. The maternal history may be characteristic. Miscarriages are common and stillbirths more frequent than in families where the mother has never been infected. It is usual to find stillbirths and miscarriages at the beginning of the family, then one or more syphilitic aments and the remainder of the children apparently normal and healthy. The sequence can be interpreted by supposing that the maternal infection gradually wears off with age and it has been suggested that a syphilitic fœtus may be actually beneficial to the mother, tending to confer on her an immunity against the disease.

In deciding whether or not congenital syphilis is the cause of mental deficiency in a given subject, one has to take into account the severity of the syphilitic process and the expected initial possibilities of mental development in the individual. In a family where subcultural mental defect is prevalent, it may be unlikely that congenital syphilis, if found in a given case, is a cause of the amentia. On the other hand, in a fraternity where there are a number of very intelligent children, the syphilitic lesions are to be considered important.

Congenital syphilis is notoriously capricious in its appearance in different members of the same fraternity. Twins may be born and only one of the pair be affected.[1] An example has even been recorded where the twins were apparently monovular and yet only one had syphilis; unfortunately, the mental condition of these children was not accurately recorded.[2] It is possible for children to give serologically positive reactions for syphilis without evincing any physical stigmata or mental retardation. The question of immunity of the fœtus, and especially of the fœtal brain, to the maternal infection is one which requires a great deal more investigation. It is possible that certain individuals are predisposed by heredity to infection and that some are more liable to suffer from mental symptoms in consequence of the disease than others. There is some evidence of the familial incidence of general paralysis of the insane in adults and in some respects the problem of causation of this disease is similar to that of syphilitic amentia.

Juvenile General Paralysis.—A special form of general paralysis affecting children has been distinguished clinically from the other types of syphilitic amentia. It may not develop until puberty, or even later, though the invasion of the nervous system by the organism of syphilis probably takes place at an early age. The disease may, in fact, be regarded as a rather uncommon form of secondary amentia. From the adult type it differs in that the duration is usually longer and remissions are practically unknown.

The usual history is of a gradual mental and physical deterioration, occurring at school age and in some cases initiated by convulsions. But occasionally the deficiency is observed in early life. The child becomes increasingly stupid, indifferent to its surroundings, dirty in its habits, and eventually bed-ridden with contracted limbs, marked emaciation, and, in fact, all the classical features which distinguish the third stage of the adult type. Neurological signs characteristic of general paralysis are invariably present, and persistent grinding of the

[1] Haslund, O., *Annales de Dermatologie et de Syphiligraphie*, t. V, 1924, pp. 321–30.

[2] Dennie, C. C., *The Medical Clinics of N. America*, Vol. VII, 1924, pp. 1219–1225.

teeth is held to be a symptom of some diagnostic value. As in other forms of syphilitic amentia, stigmata of congenital syphilis may be entirely absent. With regard to the microscopic appearances, spirochætes are usually abundant in the cerebral cortex; otherwise the morbid histology resembles that of the adult form.

CHAPTER XII

DIVERSE PATHOLOGICAL TYPES

Microcephaly—Miscellaneous Cranial Deformities—Hydrocephaly—Cretinism—Pituitary Dystrophy—Epiloia—Neurofibromatosis—Neuromuscular Degeneration.

Microcephaly.—It has already been pointed out that the concept of microcephaly has not attained any unity of definition among different writers. While some authorities merely employ it to signify small-headedness, the traditional concept, to which Tredgold adheres, describes a well-marked pathological type. Patients belonging to it not only have very small heads but certain other characteristics. The cephalic index is low (on the average about 0·75), that is to say, the head is long and narrow. The length of the head is much nearer to the normal measurement than either the height or the breadth. This cranial form distinguishes true microcephaly from the small-headedness of the mongol and from other peculiar cranial abnormalities, such as acrocephaly. In acrocephaly, the vertical measurement of the head is normal, or greater than normal, while the antero-posterior is small.

The typical traditional microcephalic ament has a face approximately normal in size, which is rather narrow with a receding chin. The appearance is quite characteristic and reminds the observer, as Lombroso noticed in 1873, of some kind of small animal or bird. Physically, the body is often quite well developed, though it usually remains smaller than normal. There may be neurological symptoms such as nystagmus, optic atrophy and paralysis of cerebral origin. In character, these patients are often troublesome and bad-tempered, but they usually improve with training and often have much more intelligence than the small size of their heads would lead one to imagine was possible. Their reactions are usually quick and their movements remind one of the

movements of animals. The brain in microcephaly is very small and the cortical convolutions are very simple in pattern. Occasionally the brain weighs no more than that of one of the larger anthropoid apes (800–900 gms.).

Darwin took a special interest in this type of case, during his later years, expecting, perhaps, to find that these human creatures with so little brain might show simian characteristics in their behaviour. It cannot, however, be said that, beyond a superficial resemblance in certain cases, there is anything specifically simian about them that is not to be seen in other low grade aments. The simian aspect of idiots is no more than a rather vague analogy. On comparing the behaviour of, say, a microcephalic imbecile with that of an ape, one is chiefly struck by the far greater intelligence of the ape, as evinced by manipulative skill and general sagacity.

The frequency of typical microcephalics among institutional aments is not very high—scarcely 1 per cent.—though it has been already mentioned that a great number of idiots and imbeciles have small heads. Although examples of the familial incidence of microcephaly are to be found in the literature, it is not always clear that the traditional type is being referred to. In the writer's experience, the familial incidence of true microcephaly is low; where it has been observed, a recessive type of heredity is usually believed to be the cause. At the present time, however, this is unproved.

Of great interest, from the ætiological point of view, are the records compiled by Murphy [1] of the conditions of children born to mothers who had been subjected, during pregnancy, to therapeutic pelvic irradiation. His original figures are summarized here.

Period of treatment.	Total number of cases.	Normal children.	Microcephalic children.	Children otherwise abnormal.	Abortions
(i) During pregnancy .	53	17	14	13	9
(ii) Before pregnancy .	256	188	1	9	67

[1] Murphy, D. P., *Surg. Gynæ. and Obst.*, 1928, Vol. XLVII, p. 201.

Among the otherwise abnormal children, born to mothers subjected to irradiation during pregnancy, were two mongols (one with hydrocephalus), one case of spina bifida and one case of deformed skull (open sagittal suture). One hydrocephalic was also found among the 9 abnormal children in the second group. Murphy concluded that pre-pregnancy irradiation had no appreciable effect on the offspring. Treatment during pregnancy, however, seemed to be liable to cause microcephaly in the offspring. If these results are confirmed we can point here to a discovery of the first importance in the study of amentia, showing the possibilities of injury to the child in the intra-uterine environment. Further analysis will perhaps show some relation between the period and duration of X-ray treatment and the type of deformity produced. It is clear from the examination of the figures that these results are not due to germ plasm injury. They may either be due to direct damage done to the fœtus or else to the diseased condition of the maternal organs for which treatment was given. Furthermore, the maternal organs may themselves have been damaged by the X-rays and rendered unequal to the task of providing proper nourishment for the growing fœtus. Cases have been recorded where very large doses of irradiation have been given in the second month of pregnancy with the object of producing abortion.[1] Here a direct effect was produced on the fœtus, invariably resulting in miscarriage. The examination of these embryos showed the presence of neuroepitheliomata of the retina—a condition allied to coloboma—and, in some cases, definite colobomatous clefts were found.

In the development of the nervous system the infolding of the outside layer of the embryo takes place, forming a tube. Failure of the edges of this tube to unite anywhere gives rise to one of a series of deformities (" Spaltbildungen ") in the nervous system, differing according to the position and magnitude of the original fault. In this way, spina bifida (or cleft spine), meningocœle, open sagittal suture and coloboma of the retina can all be regarded as belonging to the same class

[1] Goldstein, I. and Wexlar, D., *Archives of Ophthalmology*, Vol. V, p. 591.

of abnormality.[1] Microcephaly may be another condition belonging to the same group, related closely to the most severe deformity of all, anencephaly. Many rare cranial anomalies have been described which can be associated with amentia and some of these also may be the result of embryonic maldevelopment.

Miscellaneous Cranial Deformities.—A great deal of attention has been paid by anatomists and students of mental deficiency to the variations in skull formation which may be found among aments. Acrocephaly, or " steeple-head," occurs in mentally normal persons and is sometimes associated with syndactyly. There may be more than one condition classed under this name and the term oxycephaly, or " tower skull," may sometimes describe acrocephalic cases. In oxycephaly, the eyes usually appear too close together and considerable facial deformity may be present. Hypertelorism is associated with deformity of the bones at the base of the skull : the eyes are widely separated and the bridge of the nose flattened : cretinism may be a complication. Here, again, are diseases of unknown ætiology, but there is no particular reason to suppose that heredity is the deciding factor. Leptocephaly and scaphocephaly are other terms used to describe peculiar head shapes, but it may be doubted whether they really refer to clinical entities. Some of these conditions are interesting from the point of view of human biological study and anatomy, but they are mostly rare and do not affect the main problems of mental deficiency to any great degree. Their over-emphasis in medical writings has led to an erroneous view of their importance.

Hydrocephaly.—Two abnormalities have to be distinguished, both associated with large heads outside the normal variations of head size.

One of these conditions, *hydrocephaly*, is caused by the blocking of the outlets of the cerebrospinal fluid from the ventricles of the brain or the failure of its absorption.[2] The

[1] Hare-lip and cleft palate are similar deformities, but not specially associated with the failure of development in the central nervous system.

[2] Neurologists distinguish internal and external hydrocephalus according to whether the fluid collects within or outside the brain. The chronic hydrocephalus connected with mental deficiency is essentially internal.

choroid plexus continues to secrete fluid and the intra-cranial pressure increases. When this happens in the growing skull, the head gradually swells, the ventricles enlarge and the brain is stretched and flattened. The result is not always mental deficiency even though the brain substance may be considerably distorted. When mental deficiency is present, it is often less severe than might be expected from the appearance of the patient. Hydrocephaly is often associated with paralysis of the limbs. The cranium is swollen evenly in all directions and both the forehead and the parietal regions bulge outwards. The cranial capacity is often well over 2,000 c.c.

The other condition causing large heads, connected with mental deficiency, is termed *macrocephaly* and here the face is enlarged as well as the skull. The corresponding brain enlargement is due to the overgrowth of the supporting structures and not to the formation of extra nerve elements. Such cases sometimes show peculiar abilities for remembering figures and dates, although their general intelligence may be of low grade.

The cause of hydrocephaly is sometimes congenital syphilis, but infective basal meningitis of different origin may block up the outlets of the cerebrospinal fluid and cause hydrocephaly to appear in infancy. Internal hæmorrhage due to birth injury is believed to have the same result occasionally. It is quite probable that there are also developmental abnormalities which may lead to the same result. Familial incidence of such cases, however, is extremely rare and nothing can, at present, be said about an hereditary mechanism. Macrocephaly is of quite unknown origin.

Cretinism.—Disorders of the ductless gland system have an important place in the study of mental deficiency. Some authorities hold that mongolism should be classified under this heading. Many mongols show signs of endocrine disorder but, so far, no one has been able to describe any endocrine peculiarity which is specific.

The most frequent and most important type of endocrine disorder found among aments is sub-thyroidism ; patients presenting a fairly marked degree of this are frequent and form perhaps from 3 per cent. to 5 per cent. of all institutional

cases. The condition, whether mild or severe, is much more frequently seen in females than in males.

One would gather, from reading the medical textbooks, that it is difficult to distinguish between cretinism and mongolism. The two conditions may be combined in the same individual but, even then, one can distinguish some of the characteristic features of each disease separately. As previously pointed out, a high cephalic index is characteristic of mongolism, but in cretinism the cephalic index is normal or low. This is an important diagnostic point. Moreover, the size of the head in cretinism approaches normal dimensions and may even be larger than usual. In cretinism the tongue is usually quite smooth and the mucous membranes of the palate are rarely thickened : epicanthus is not very frequent. The points of resemblance, which make confusion between the two conditions possible, are the stunted growth, short and podgy limbs and signs of retarded physical and mental development.

The majority of cretins found nowadays in institutions for mental defectives have been subjected, for periods of their lives, to treatment by the administration of thyroid extract and, therefore, one does not often see the gross deformities characteristic of the untreated condition. Furthermore, the majority of institutional cretins show little or no mental improvement on the administration of thyroid extract. This is partly perhaps because the treatment has not been started early enough but, in many cases, it has been successful in removing nearly all the physical stigmata of the disease without affecting the mental condition. It is very difficult to understand why certain cases react well to the thyroid treatment and others fail to do so ; possibly hereditary influences are the determining factor. Some authorities hold that, when the onset of cretinism is late, there is a reasonable prospect of cure, but that treatment is of little avail where the defect is noticeable at birth.

In the medical textbooks, cretinism is usually divided into two classes, sporadic and endemic. It is extremely difficult to apply this classification to any given case. It has sometimes been stated that the sporadic cretins are due to heredity and

the endemic cretins due to environmental influences. This is because endemic cretinism is found in certain geographical districts and not in others. It is usually described as being associated with goitre in other members of the family and occurs in South Germany, Switzerland, and the Tyrol, and also in England, in Derbyshire, the West Riding of Yorkshire and Somersetshire. Some of these districts are mountainous and it has been supposed that the distance of these places from the sea leads to deficiency in iodine and that this is the cause of the endemic thyroid deficiency. An argument against this is that, to persons living in other places where cretinism is endemic, iodine must be available—certainly in England where the distance is only some seventy miles at most from the sea. The water supply has been incriminated by some investigators and this seems a reasonable hypothesis. It is, in fact, supposed that organisms may live in the mountain streams and in rain water supplies which have a pathogenic effect on the people who drink from them habitually. In England, cretins are also frequent in the low-lying fen district of the Isle of Ely where rain water is, or has been until recently, the main drinking supply.

Sporadic cretinism is supposed to occur in families without reference to geographical situation and, as in the endemic type, one child, or more, may be affected. Sometimes congenital syphilis may produce an inflammation of the thyroid gland, causing its function to be impaired, and occasionally other infections may do the same. Possibly the thyroid apparatus of some individuals is more subject (on account of hereditary constitution) to the ill effects of infective processes or lack of iodine.

A remarkably close zoological parallelism with endemic and sporadic cretinism is to be found in the behaviour of certain newts. The triton alpestris fails to metamorphose in certain upland lakes of Switzerland though it never fails to do so in other places. Apparently the anomaly is due to environmental lack of the iodine necessary to the proper functioning of the thyroid. On the other hand the Mexican axolotl (amblystoma tigrinum) which, normally, never metamorphoses, even if provided with excess of iodine, can be made to do so

if fed on thyroid tissue. Here there is hereditary deficiency of the thyroid to utilize iodine. Thus we see how closely related are the effects of environment and hereditary constitution in connection with the functioning of the thyroid gland.

Pituitary Dystrophy.—Of the endocrine disorders to be found among mental defectives, next in importance to cretinism, the various forms of pituitary dystrophy come under consideration.

The typical " fat boy," suffering from dystrophia-adiposo-genitalis (Fröhlich's syndrome), is not infrequently met with and is easy to diagnose on account of the feminine distribution of fat and the infantile genital organs. The cephalic index in such cases is often higher than normal and this is perhaps associated with a small pituitary fossa and short base to the skull. Sometimes the disorder is a subsequent effect of encephalitis, and pituitary dystrophy of the Fröhlich type may follow encephalitis lethargica, the character of the syndrome varying with the age at onset of the infection. Mild degrees of pituitary adiposity are also seen, and they can be associated with mental deficiency of any grade. Various other types of pituitary dystrophy, such as gigantism, or even gigantism with adiposity, are also to be found here and there. It is always difficult to assess how far the endocrine disorder is responsible for the mental deficiency, but there seems no special reason to doubt that it may be fully responsible in certain cases. Mental, as well as physical, improvements have been reported after treatment with various forms of pituitary extract.

A very peculiar and exceedingly interesting form of pituitary disorder, associated with mental deficiency, has been described by Laurence, Moon, Biedl and others. In these cases there are present varying degrees of retinitis pigmentosa, polydactyly and other less constant abnormalities. Night blindness is associated with the progressive degeneration of the retina. The hands or feet have extra digits, usually of a rudimentary character. Males and females are affected with about equal frequency. This disease is usually found in more than one member of a fraternity but, like many other conditions affecting the human species, its character varies from one

affected person to another. For instance, one member of the family may be affected with polydactyly while another has pituitary dystrophy and retinitis pigmentosa. The family recorded by Griffiths [1] is fairly typical and I quote it here.

> The history on the father's side is negative. The mother is one of four children; her elder brother had two thumbs on one hand. She is plump, a tendency which runs in the family. She had six pregnancies: (1) a girl, age 10, is normal mentally, but has webbed toes on each foot; (2) a girl, also with webbed toes; (3) a boy of 6, quite normal; (4) the patient, a girl, age 5, imbecile, obese, six toes on each foot, night blindness, but no typical retinitis pigmentosa; (5) a boy, who died when 10 days old of bleeding from the nose, mouth, and rectum, and who had six fingers on each hand and six toes on each foot; (6) a boy, stillborn, with a deformed head.

The condition is rare and, at present, not sufficient accurate pedigree studies have been made to warrant a belief in any particular form of heredity being responsible. A few instances have been recorded in which there was consanguinity between the parents [2] and, with such a rare condition, this is suggestive of a single recessive gene substitution. There are, however, difficulties in the way of this view. In some cases, although the parents were not affected, atypical instances of the disease were found in the parental sibships.[3] Possibly the heterozygous genotypes are sometimes mildly or atypically affected. The fundamental cause of the condition may be a single gene substitution, the overtones being supplied by both environmental factors and individual differences due to the remaining genetic constitution.

Epiloia.—Another curiosity of medicine, associated with mental deficiency, is a disease which was named by Sherlock.[4] The clinical entity was recognized long ago by Bourneville in 1880 [5] and is a symptom-complex which includes tuberose sclerosis of the brain, sebaceous adenoma on the face (forming a "butterfly rash") with other skin peculiarities, unusual

[1] Griffiths, B. M., *Journal of Neurology and Psychopathology*, Vol. XII, No. 45, July, 1931.

[2] Beck, H. G., *Endocrinology*, Vol. XIII, 1929, p. 375.

[3] Deusch, G., *Deutsche Ztschr. f. Nervenheilk.* Bd. LXXXVII, 1925, S. 117.

[4] Sherlock, E. B., *The Feeble-Minded*, Macmillan & Co., London, 1911.

[5] Bourneville, D. M., *Archives de Neurologie*, 1880–81, p. 81.

visceral growths (rhabdomyomata of the heart, mixed kidney tumours) and finally epileptic psychosis. The disease is progressive, the symptoms appearing slowly and increasing in intensity with age. There is considerable variability in the incidence of the distinctive pathological signs in different cases and numerous clinical types have been described. Mental deficiency is always associated with severe cases and almost all such subjects are psychotic. They may show, in addition to epileptic symptoms, a mental state resembling dementia præcox. The pathological anatomy of the disease has been very thoroughly investigated and there seems little doubt that it is a developmental abnormality.

Genetic study reveals a high incidence of psychosis in families where epiloia occurs, but the familial incidence of the actual disease is low. It is rare to find more than one member of a family affected. The disease itself being very rare, however, the recorded familial incidence is greater than would be expected from mere chance occurrence. Cases have been described where the disease appeared in parent and child. Owing to the variety of clinical types, it is not always easy to determine whether a given member of the family was affected or not, on the evidence of a history, and the familial incidence may appear lower than it really is. Moreover, many cases are probably missed owing to the erroneous diagnosis of the facial eruption as " acne rosacea." At the present time nothing definite can be inferred about the causation of the condition, but the familial incidence points to some genetic mechanism. The effect might be due to the coincidence of two or more dominant genes. The variety in the clinical aspects may be due to hereditary differences in the different subjects attacked, but it also may be due to environmental influences. Brushfield and Wyatt [1] reported that, out of 12 cases, 7 were firstborn, and this suggests that primogeniture is a causative factor. Other observers have not substantiated this finding.[2] Tredgold has pointed out that almost every tissue of the body is subject

[1] Brushfield, T., and Wyatt, W., *British Journal of Children Diseases*, 1926, Vol. XXIII, pp. 178 and 254.

[2] Critchley, M., and Earl, C. B., *Brain*, Vol. LV, Pt. 3, 1932, p. 311.

to tumour formation in epiloia and, as this seems to be the only disease where this occurs, it is possible that some unusual mechanism such as chromosome doubling may be the cause.

Neurofibromatosis.—Multiple nerve tumours of the type described first by von Recklinghausen are sometimes found associated with mental deficiency. The tumours are mostly subcutaneous and vary in size. They are progressive and sometimes occur in enormous numbers, covering the whole body : patches of pigmented skin are usually present. Hashimoto (1890) counted 4,503 tumours on the skin of a middle-aged Japanese man suffering from the disease. It has been found that nearly 10 per cent. of the cases are definitely mentally defective.[1] Since mental deficiency affects only about 1 per cent. of the general population, there is reason to believe, therefore, that there is a common causal element, where these two conditions coexist.

Numerous family histories of patients have been collected, and they are, on the whole, consistent with the hypothesis that a double dominant gene determination is responsible for multiple neurofibromatosis. This cannot be regarded as having been proved. The general appearance of the findings in pedigree studies is not unlike that shown by epiloia. In both diseases latent forms may occur, which are not recognized in family investigation, and, therefore, it is possible for a single dominant gene to be the determining factor. Some writers (Orzechowski, Nowicki, Van der Hoeve) held recently that the two diseases are the same, or at any rate closely related to one another, but their views are not generally accepted.

Neurofibromatosis is sometimes associated with a mild degree of cretinism and affected persons are usually short of stature. There seems to be no special relationship between the severity of the disease and the degree of mental impairment, though the mental and physical condition of an insufficient number of cases has been recorded to make the matter clear. It is, therefore, very difficult to guess why the disease is sometimes associated with mental deficiency and sometimes not.

[1] Preiser, S. A., and Davenport, C. B., *American Journal of the Medical Sciences*, 1918, Vol. CLVI, pp. 507–40.

Neuromuscular Degeneration. — Another group of rare diseases, met with in the study of mental defect, is marked by the occurrence of progressive degenerative changes in the nervous system. It is not yet to be regarded as certain whether all of them are of the degenerative type, some may be inflammatory in origin.

The most definite example is amaurotic family idiocy. Two clinical forms have been described: the infantile and juvenile types. They differ essentially only in the age of onset. The infantile type begins in the first year of life, and is recognized by degeneration of the macular region of the retina, leading to progressive blindness. Concurrently a flaccid paralysis develops, with wasting of the limbs and a progressive mental degeneration. The end result is a profound type of idiocy, although the child may have appeared normal in early life. Affected children usually die at the age of two. In the juvenile type the onset is later and the course slower. The disease is of special interest in the study of mental deficiency because it is the only condition, so far described, which is invariably associated with amentia and which is also almost certainly determined by a single recessive gene. The extreme rarity of the condition, however, makes the importance of this discovery almost negligible from the point of view of the study of the causes of mental deficiency in general, although it is of great value in the study of human biology.

Other nerve degenerations which may be associated with mental deficiency and which are of hereditary origin are pseudohypertrophic muscular dystrophy and Friedreich's ataxia. The former appears to have a sex-linked type of inheritance and the latter seems to act as an autosomal recessive. These diseases rank with Huntington's chorea, which is due to a dominant gene producing progressive insanity. They are all examples of rather rare conditions wherein the genetic analysis, although not yet fully worked out, is comparatively easy to understand. Schilder's disease (encephalitis periaxalis diffusa) and Wilson's disease (progressive lenticular degeneration) may be hereditary degenerative processes in the nervous system; they are also sometimes associated with mental deficiency.

CHAPTER XIII

MENTAL DISORDER

The Neuropathic Theory—Epilepsy—Dementia Præcox—Perversion—Neurosis—Criminal Cases.

The Neuropathic Theory.—The writer has already explained that, in his view, mental disorder and mental deficiency should be studied separately. The relationship between the two kinds of mental abnormality is obscure and it is doubtful whether it can be elucidated unless they are separately considered. Tredgold has advanced the hypothesis that all mental disease is essentially the same and due to neuropathic constitution. In his view, senile dementia and idiocy are both ultimately expressions of the same fundamental cerebral weakness. This is a type of generalization too wide to be of much service in scientific investigation, for it tends to make more difficult the subsequent analysis of similarities and differences. Two important arguments have been used to support the view held by Tredgold. They are derived from observations, firstly, of the frequency with which both defect and insanity are met with in the same pedigrees and, secondly, the frequency of occurrence of both conditions in the same individual. Neither argument implies that deficiency and disorder are due to the same thing, but both tend to prove the existence of some relationship between them.

Now it was observed by Pearson that families of persons affected with severe physical malformation, like "split hand," contained an unusual number of mentally abnormal persons. He attributed this to the intermarriage of the mentally peculiar with the physically peculiar and to the absence of any eugenic conscience in persons of low mentality. The same explanation can be applied to the frequency with which mentally disordered and defective persons are found in the same family group. There is evidence that the mentally defective tend

to mate more readily with one another than with normal persons, and probably the same applies to matings of the mentally disordered with the defective. Whether mental abnormality is inherited or determined by environment, this process of assortative mating will tend to collect together many kinds of mental peculiarities into the same family group. The task of the investigator is to sort out the different kinds of abnormality and discover the causes of each. Any kind of mental abnormality renders the subject less suitable for ordinary life in the community and brings about liability to certification under the Lunacy or Mental Deficiency Acts. The investigator must not be misled into expecting all these abnormalities to have common causes because they have this common effect.

Epilepsy.—By far the most frequent kind of mental disorder affecting aments expresses itself in some form or other of epileptic attacks. About 1/5 of all institutional cases suffer from epilepsy at some time during their lives, and the majority of these have fits at intervals during their whole lifetime. Epilepsy is essentially a symptom and not a disease. The characteristic feature is sudden loss of consciousness for a short period of time without obvious cause, such as asphyxia, cardiac failure, or concussion. It may be produced in a great variety of ways. In a number of epileptic patients some definite abnormality or disease of brain tissue is present, causing the instability which leads to the symptom. Syphilis is frequently responsible, but developmental abnormalities, as in epiloia, also give rise to severe fits. In a large group of cases, however, there is no obvious cause for the attacks, and the term *idiopathic* epilepsy has been used in medicine to signify that the cause is unknown. The brains of so-called idiopathic cases do not necessarily show any deviation from the normal, except in the later stages of chronic epilepsy. Experimental work which has been carried out on animals has shown that the susceptibility to convulsant drugs can be altered if the cerebral cortex is injured.[1] It

[1] Pike, F. H., and Elsberg, C. A., *American Journal of Physiology*, 1925, Vol. LXXII, p. 337.

is, therefore, probable on purely empirical grounds that cerebral damage is one of the determinants of the symptom, but not the only one.

There are several kinds of attacks which are often excluded from the category of epilepsy. Two important examples are hysterical fits and vasovagal attacks. Imbeciles of the mongolian type are supposed to be immune from true epilepsy, but occasionally cases are subject to fits. It is usually assumed that these are some form of fainting or vasovagal attacks and are associated with the poor circulatory system of these individuals. It is, however, difficult for the physician to distinguish one kind of epilepsy from another because he rarely sees the patient actually in the fit. Hysterical attacks are particularly difficult to dissociate from true epilepsy in mental defectives, partly because institutional patients have the opportunity of seeing others in genuine fits and are able to copy their behaviour with great exactness.

Stoddart, Schilder and others regard idiopathic epilepsy as a sign of psychosis, and, in fact, a symptom of a special type of mental disease. The remaining part of the clinical picture is to be found in the patient's reactions at periods between the fits. The epileptic's character is described as emotionally unstable, shallow, sentimental and self-centred. The speech is said to be monotonous and the reactions are stereotyped. Mentally defective epileptics sometimes show this type of character, particularly in the instability and violence of their emotional reactions.

Other types of fits (epileptic variants) such as narcolepsy and ocular crises are to be looked for among certified aments and often give valuable diagnostic information in neurological cases.

Since epilepsy is only a symptom and not, in itself, a disease, statements which have been made about the inheritance of epilepsy must be received with great caution. The incidence of epilepsy in the families of epileptics is very low, even if the investigation is confined to what appear to be true idiopathic epileptics. Moreover, the children of epileptic parents rarely exhibit a tendency to this type of reaction ; Myerson,

Thom and Pilcz all agree about this.[1] It is possible that the symptom is sometimes a manifestation of anaphylactic phenomena, and that it is related to migraine, asthma and to various types of hypersensitiveness. If this is so, whatever hereditary factors may be involved will require elucidation by subtle biochemical methods. It might, however, also be possible to detect the epileptic character in persons belonging to families where epilepsy occurs in other individuals. In the present stage of our knowledge the causation of epilepsy is to be looked for in the individual and not in the family. Indeed, Wilson has remarked that it is time protest should be unitedly voiced by neurologists against the portentous ascription of a sinister prognosis to every case of epilepsy because of a supposed inheritance and consequent incurability.[2]

Dementia Præcox.—Two classical forms of dementia præcox as described by Kraepelin are fairly frequently seen in persons certified mentally defective. Whether the patient would have been mentally defective apart from the psychosis it is usually impossible to judge.

Dementia simplex was originally described as a condition in which the thought content is very meagre but in which reactions are similar to those found in other forms of dementia præcox. When patients, diagnosed as simple dementia, are subjected to intelligence tests, they invariably fail to reach normal standards.[3] Thus, if the onset of dementia is before the age of 18, the patients can be certified under the Mental Deficiency Acts. The condition is often so difficult to diagnose that it is almost a matter of luck whether these early cases drift into mental hospitals or certified institutions.

The other form of dementia præcox seen among mental defectives is a very early type, which may come on in infancy—*dementia præcocissima*. The result, again, is certainly mental deficiency, but the patients are usually distinguishable by their

[1] Myerson, A., *The Inheritance of Mental Diseases*, Williams and Wilkins, Baltimore, 1925, p. 71.

[2] Wilson, S. A. K., *British Medical Journal*, October 26th, 1929, p. 745.

[3] This applies fairly generally to almost all types of dementia præcox. Wentworth, M., Two Hundred Cases of Dementia Præcox. *Journal of Abnormal Psychology*, 1923–4, Vol. XVIII, pp. 378–384.

peculiar mannerisms and their extreme negativism. They may appear to have hallucinations, but usually the mentality is of the idiot grade, or deteriorates into this in a short time, and it is impossible to discover the thought content. This type of psychosis is a disease which is not seen in mental hospitals.

Any type of dementia præcox can occur in persons of initially poor intelligence. The result is merely to make the patients considerably less interesting to the psychiatrist than when a similar disease occurs in clever people.

Other psychotic symptoms are also of quite frequent occurrence in persons already on the level of mental deficiency. Persecutory ideas and ideas of grandeur may occur. More frequent still are such symptoms as echolalia, echopraxia and ideational inertia. Catatonia has been reported in mongolism. A state of chronic mania is not uncommonly found associated with feeble-mindedness. It is, however, extremely difficult to judge whether any of these psychotic symptoms are really more frequent in the mentally dull than in the mentally bright. We may argue that the presence of psychotic symptoms in dull people makes them much more likely to be certified defective. Lange-Eichbaum [1] has used a similar argument to prove that the popular idea of a strong association existing between genius and madness is illusory. Clever people who are unstable attract more notice than the equally clever people who are of stable character.

Perversion.—Exactly the same difficulty applies to the consideration of the frequency of the various forms of sexual perversion among the mentally defective. A careful examination of high grade male and female institutional patients reveals that a great number of them have strong homosexual tendencies such as are to be found also among normally intelligent persons. It is quite likely that latent tendencies in this direction may have been fostered by institutional segregation of the sexes. At the same time some cases are admitted largely on account of homosexual behaviour which

[1] Lange-Eichbaum, W., *The Problem of Genius*, Kegan Paul, London, 1931.

they have not been intelligent enough to conceal from the eyes of the general public and the law. Social class has an important effect here also. It is probable that homosexuality is to be discovered in about an equal degree in all social conditions, but it is better tolerated in the more educated groups. Homosexuals of low social standing are therefore more in danger of coming into contact with the law than those of higher social class.

Mental defectives are not infrequently certified on account of their immoral sexual behaviour towards children and even, occasionally, towards animals. Indecent exposure of the genitals also sometimes leads to certification in a person who has otherwise been regarded as a harmless, sociable, though stupid, creature. Here, again, one cannot help feeling that these offences may occur because of injudicious or foolish behaviour on the part of the subject rather than because there is any particular difference in the sexual orientation of mental defectives and normals.

It has often been suggested that high grade mental defectives have particularly strong sexual impulses. There is very little real evidence for this. It is true that they are often uncontrolled in their behaviour, but really passionate attachments to persons of the same or of the opposite sex are only found among those persons whose mentality is fairly close to normal standards. This is not to be taken to mean that imbeciles are not often very devoted to the people who look after them. It means that, the lower the I.Q., the less vigorous are the sexual emotions, otherwise it would seem probable that the hardship of lifelong segregation would be quite intolerable to the majority of patients, and this is not the case.

A considerable number of cases of incest between father and daughter or brother and sister are found in families where the members are of subcultural mentality. This seems to show not that the subcultural people are more perverse than the rest of the population but to indicate a stagnation in the development of the individual with reference to the society in which he is living. The human race has evolved a system of exogamy which is coupled with the rest of its intellectual

civilized life, and a failure to live up to the latter seems to imply also an involution of the former system.

Neurosis.—The two chief types of the milder mental disorders (neuroses) which are seen in persons of normal intelligence, *hysteria* and *obsessional neurosis*, probably are not found more frequently in defectives than in the general population. Certain special forms of neurosis may be extremely anti-social and, when coupled with backwardness in educational attainments, often lead to actual certification.

A common disorder under this heading is the compulsive desire to commit petty thefts. Here again, social class is extremely important in the determination of who is certified and who is not. A person belonging to a well-to-do family is not in serious danger of imprisonment for committing petty thefts. Similar behaviour on the part of a domestic servant may lead either to imprisonment or certification under the Mental Deficiency Acts, by a magistrate's order. By the time that such cases come to the notice of a medical authority, they are usually adult and little can be done to help them by psychotherapy. In children, however, when such symptoms occur, there is always a chance of cure by careful individual treatment or by alteration of environment. Some children become anti-social because the canons of behaviour according to which they are brought up are anti-social. A new home environment may alter this : and, since the duller they are, the more susceptible they are to bad influence, they are correspondingly amenable to good influence. In the opinion of many modern authorities the treatment of crime should be preventive. It is quite possible that, in many cases, anti-social symptoms leading to delinquency are evidence of deep-seated mental disorder and may simply be the forerunners of some form of psychosis. In such circumstances preventive treatment is much more difficult.

Criminal Cases.—Authorities do not agree at the present time as to how far crime can be regarded as evidence of mental disease or deficiency. There is no implication of this idea in the legal definition of crime.[1] Statistics, however, show that

[1] " An unlawful act or default which is an offence against the public and which renders the perpetrator of such an act or default liable to legal punishment."

there exists a strong association between crime and mental deficiency. More than one tenth of admissions to the Royal Eastern Counties Institution are certified under the Mental Deficiency or Children's Acts after appearance in the Police Courts, and are kept in the institution as an alternative to being sent to a prison or an industrial school. This high proportion of criminal cases in institutions is partly due to the shortage of beds and the necessity of finding immediate accommodation for such patients. Opinions differ, however, about what proportion of all criminals are mentally defective. Estimates varying from 3 per cent. (East) to 15 per cent. (Pailthorpe [1]) have been given for England. Much higher figures, 29 per cent. (Erickson) and 55 per cent. (Goddard), have been given for America. The percentage depends on the definition of mental deficiency adopted. For example, Burt [2] found that 7·6 per cent. of juvenile delinquents were defective, while 25 per cent. were naturally dull (I.Q. 70 to 85), and even more were educationally backward. Numerous adult prisoners are also found to be of dull mentality, and many are mentally deranged. It is of the utmost importance for administrative purposes that the association between crime and feeble-mindedness should be fully recognized. Even if imprisonment is beneficial to the prisoner and to society in the cases of criminals of normal intelligence, it is useless with the feeble-minded, who sometimes have only a vague idea of what offence they are convicted.

[1] Pailthorpe, G. W., *Studies in the Psychology of Delinquency*. H.M. Stationery Office, 1932.

[2] Burt, C., *The Young Delinquent*. University of London Press, 1925.

CHAPTER XIV

SUBCULTURAL AMENTIA

Description—Intelligence and Social Class—The Study of Twins—Family Studies—Environmental Factors—Illustrative Cases.

Description.—The use of the term *subcultural* to denote a certain class of mental defectives has already been discussed. It is convenient to speak of the *pure* subcultural type when reference is being made to the patients who can be included in none of the previously described categories of specific conditions. These people are mentally defective for no obvious reason : as individuals they are usually indistinguishable from the normally intelligent on physical grounds. They can be differentiated from the dull by no exact dividing line, any more than the dull can be clearly differentiated from the normal. Pearson [1] states that the distribution of intelligence in both normal and mentally defective is absolutely continuous, and in purely psychological tests of intelligence there are no rigid and limited categories of normal intelligence and feeble-mindedness.

The subcultural group can be regarded as a part of the normal population just as much as those who form the group of persons of superior intelligence. There is no theoretical upper or lower limit to a continuous distribution of this kind, and there is no objection even to the diagnosis of an idiot as purely subcultural, provided that physical disease is excluded in the ætiology. This idea introduces a great simplification into the methods of investigation, for any causes which are found to be effective in producing variation in intelligence among normals will be relevant in the study of this sub-group of the normal.

[1] Pearson, K., and Jaederholm, G. A., *Questions of the Day and of the Fray*, VIII, 1914.

It is well to distinguish between the subcultural mental defectives and the group of individuals known as the social problem group.[1] Many of the subcultural simpletons [2] who find their way into institutions have been certified on account of social misdemeanour. This does not mean that all simpletons behave anti-socially. Probably the great majority of individuals, whose intelligence is subnormal, are capable of living usefully in the community, though in times of economic stress like the present, they are liable to find this difficult. Even so, it is less serious for a simpleton to be idle, provided he is well behaved, than for a skilled worker to be out of employment. According to the view expressed in the Wood Report,[3] however, the "social problem" or "subnormal" group comprises the lowest 10 per cent. of the social scale and includes "as everyone who has extensive practical experience of social service would readily admit," a much larger proportion of insane persons, epileptics, paupers, criminals (especially recidivists), unemployables, habitual slum dwellers, prostitutes, inebriates and other social inefficients than would a group of families not containing mental defectives. This terrible indictment is coupled with the totally unproved assertion that the mental defectives concerned are of the primary (hereditary) type. That mental deficiency may be to some extent due to criminal parents' dwelling "habitually" in slums seems to have been overlooked. And there is plenty of common sense evidence for this neglected view. Furthermore, there is distinct correlation between the intelligence of school children and their environment, whether measured by the economic position of the parents, by the care taken of the home, or by the clothing of the children. The partial correlations for constant age are uniform in sign, of order 0·3 to 0·4 and five to six times their probable errors.[4]

[1] Lidbetter, E. J., *Eugenics Review*, Vol. XXIV, No. 1, April, 1932, pp. 7–12.

[2] I use the word *simpleton* as a synonym for feeble-minded.

[3] *Report of the Mental Deficiency Committee*, Part III, p. 80, H.M. Stationery Office, 1929.

[4] Isserlis, L., The Relation between Home Conditions and the Intelligence of School Children. *Medical Research Council, Special Report Series*, No. 74, H.M. Stationery Office, 1923.

Intelligence and Social Class.—Many intricate problems are raised in connection with the relation of intelligence to social status. We will take the simpler issues first. Burt [1] regards the class of unskilled labourers to be, on the whole, of definitely subnormal intelligence (I.Q. 70–85) and he designates casual labourers feeble-minded (I.Q. 50–70). Now, almost all the subcultural defectives who drift into institutions come from the poorer social classes, whereas the same is not true for other defectives. On examination, the families of simpletons are found to contain other individuals, of the same mental grade as the patients, carrying on the ordinary life of citizens or villagers. One would not be able to detect a well behaved high grade simpleton by passing him in the street, for instance, or even, in many cases, by employing him in some straightforward occupation. I know of a man who is the father of three daughters (one certified feeble-minded), who has been employed on the same farm as labourer for 11 years and has given full satisfaction in his work. His mentality, as judged by tests, is, however, a good deal lower than that of his certified daughter. He is now over fifty and, had he been born more recently, he might have been sent to a Special School. In rural districts, even at the present time, individuals like this man are not looked upon as abnormal, but merely rather dull.

In the Wood Report, Lewis drew attention to the greater frequency of mental deficiency in the country than in the boroughs. He attributed this to the draining off of the more intelligent elements of the population into the towns where better prospects and higher wages could be obtained for their services than in rural districts. This process has possibly been an important cause of the decay of rural life in England. Thus, we are not surprised to find that a good many subcultural defectives are just members of the ordinary rural population.

In the urban districts, the position is a little different owing to the prevailing higher standards of social and industrial requirements. In towns, there is less opportunity for simpletons

[1] Burt, C., A Study in Vocational Guidance. *Indust. Fatigue Research Board*, H.M. Stationery Office, 1926.

to find suitable work and the family whose members are of poor mental ability sinks rapidly in the economic scale. Thus, in the towns, we are liable to find a more marked association between intelligence and social class than in the country.

A number of examinations have been made of the relation between social status and intelligence by testing schoolchildren. The general result of such examinations has been to show that children drawn from the higher social strata perform better at intelligence tests than do those drawn from the lower strata. Binet and Simon [1] themselves were the first workers in this field. Since then, Burt, Yerkes and Anderson, and Terman have made investigations. Duff and Thomson [2] analysed the test scores of some 13,000 children in Northumberland schools between the ages of 11 and 13. The average intelligence quotients of children of professionals was 112 and that of children of fathers in lower grade occupations, 96. These observations are supported by studies showing the negative relationship between intelligence of children (in the general population) and the size of the family from which they come (Bradford, Terman). Professional men, for instance, have smaller families than labourers, but their mental capacity is higher. Sutherland and Thomson [3] concluded that the correlation between I.Q. and size of family was about —0·2 in over 1,000 elementary schoolchildren studied in the Isle of Wight. As demonstrated by Russell,[4] there is in every class of the community a wide range of mental ability in the offspring. It may indeed be true that purely subcultural mental defectives are commoner in the lower social strata, but subcultural children are also born to parents of the highest social standing. Simpletons of the richer social class, however, are not likely to be found in certified institutions.

The investigation of intelligence in children of different social classes is subject to numerous errors and difficulties.

[1] Binet, A., and Simon, Th., *L'Année Psychologique*, 1911.

[2] Duff, J. F., and Thomson, G. H., *British Journal of Psychology*, Vol. XIV, pp. 192–8, 1924.

[3] Sutherland, H. E. G., and Thomson, G. H., *British Journal of Psychology*, Vol. XVII, pp. 81–92, 1926.

[4] Russell, J. B., *British Journal of Psychology*, Vol. XX, pp. 274–95, 1930.

High Grade Feeble-Minded Girl. (See p. 154.)

[*To face p.* 148

Variations of the I.Q. with age and differences in the educational methods of different schools have to be allowed for. The main causes of variation in test scores from individual to individual are usually taken to be differences in hereditary equipment and differences in home environment. Much work has been done recently which sets out to disentangle the effects of these factors. The data investigated are, on the one hand, the performances of children born in different social strata but brought up in the same environment and, on the other hand, the performances of children born in the same social class educated in different environments. For this purpose, children brought up in orphanages or adopted by foster-parents provide convenient material, though differences in length of time of boarding out introduce complications when small numbers of children are used.

In a study of some 800 children in orphanages, Jones and Carr-Saunders [1] reported that there appeared to be a differentiation of intelligence, according to social class, which tended to diminish as the length of time of residence of the children increased. A more cogent demonstration of similar results was given by Freeman, Holzinger and Mitchell [2] who made a very thorough examination of the mentality of 159 adopted children. They came to the conclusion that two unrelated children, reared in the same home, resemble one another in intelligence to a slightly greater degree than two siblings separated at an early age and educated in different homes. On the other hand, Lawrence [3] has recently published data showing that significant variations could be detected by use of the correlation ratio in the intelligence of illegitimate orphan children whose fathers were drawn from a variety of social classes.

The Study of Twins.—A closer approach to the problem of the relative effects of heredity and environment, in determining

[1] Jones, D. C., and Carr-Saunders, A. M., *British Journal of Psychology*, Vol. XVII, pp. 343–64, 1927.

[2] Freeman, F. N., Holzinger, K. J., and Mitchell, B. D., *Twenty-seventh Yearbook*, Pt. I, pp. 103–217, 1928.

[3] Lawrence, E. M., *British Journal of Psychology*, Monograph Supplements, No. 16, 1932.

intelligence, can be made by the study of twins in the way suggested originally by Galton. The advantage of this method is that it eliminates social class as a factor and does not necessarily assume that persons belonging to the same social group are biologically similar. The theory on which such a study is based is that identical twins (*i.e.* identical in hereditary equipment) reared in different environments should be compared with fraternal twins (*i.e.* of different hereditary constitution) brought up in the same environments. The subject is complicated. In the first place it is extremely difficult to judge how far the environments of twins are the same before birth. There is considerable evidence that the conditions during that period may exert variable influences on one or other of the pair, even of identical twins. Moreover, an environment in early life which may appear, to the casual observer, to be the same for two children can, in reality, be very different. While the likenesses between identical twins are often very striking, such differences as are to be found, both physically and mentally, appear thereby even more remarkable. Newman [1] found that differences in environment and training may cause as great divergence in mental capacity between identical twins as naturally exist between fraternal twins reared in the same environment. In other words, he found environment and heredity, as causes of variation in the intelligence of normal persons, to be of about equal importance. The subject has been very fully investigated by Herrmann and Hogben [2] who amplify, in many ways, the work of Stocks. They show that hereditary differences account for about half the average differences of individuals of same birth rank within the family. This agrees closely with Holzinger and with Tallman. The contribution of heredity to differences between different social levels is probably much less. The studies carried out on the incidence of mental deficiency, crime and insanity in twins so far have not demonstrated any such rule as this applying to mental abnormalities. There is here a

[1] Newman, H. H., *Eugenics Review*, Vol. XXII, No. 1, April, 1930, pp. 29–34.

[2] Herrmann, L., and Hogben, L., *Proceedings of the Royal Society of Edinburgh*, Vol. LIII, 1933.

fertile field for future research.[1] When we are dealing with the subcultural defective we may, however, as previously pointed out, apply directly the knowledge obtained from studies of mentally normal individuals. Hence the studies, referred to above, on normal twins are of direct relevance to the understanding of the causes of mental deficiency.

Family Studies.—In certain ways, investigation of the subcultural group may be useful in ascertaining facts about the inheritance of mental qualities in the general population. Provided the cases are first carefully sorted out so as to exclude special types and diseases, the survey of the families of mental defectives gives interesting results from this point of view. The average mentality of brothers and sisters is similar to that of the parents, but there is a large variation above and below the average. One does not expect to find a genius to be the child of parents both of whom have an I.Q. of 50; on the other hand, every child of such parents will not necessarily be mentally defective. The present writer [2] made a study of the families of a hundred aments of the pure subcultural type and found that the relation of the intelligence of the children to that of the parents could be explained on an assumption, such as was made by Fisher in his analysis of the incidence of different physical statures in familial data collected by Pearson. Fisher's analysis showed that physical stature might be supposed to be mainly the result of a large number of independent genetic factors whose effect was additive. Hurst has recently attempted to show that intelligence (or mental stature) is inherited by means of ten dominant genes. His method of analysis is similar to that of Frets,[3] who attributed variations in cephalic index to nineteen factors of four types. These arbitrary numerical estimates cannot be seriously considered. The principle, however, that both physical and mental characters probably imply a great number of genetic determinants seems to be fairly well established. This principle must be regarded as applying to both the normal and

[1] Lewis, A. J., *Eugenics Review*, Vol. XXIII, No. 2, July, 1931, pp. 119–25.

[2] Penrose, L. S., *British Journal of Psychology*, Vol. XXIV, July, 1933, p. 1.

[3] Frets, G. P., *Heredity of the Cephalic Index*. 1924.

subcultural parts of the population, for intelligence; nevertheless it does not conflict with the possibility of finding, here and there, clearly defined characters, mental or physical, whose mode of inheritance is very much simpler. In the systematic investigation of an isolated Norwegian peasant population, Sjögren [1] found a large number of cases of amentia of a special type which, however, could not be easily distinguished from what is here termed pure subcultural amentia. The majority were imbeciles, but no neuropathological signs were present, and histological examination of the brain in five cases demonstrated nothing abnormal. The familial incidence was not very high, but on grounds of frequent parental consanguinity Sjögren considered a single recessive gene to be the determining factor; albeit the cooperation of a sex-linked gene was regarded as probable. In an earlier chapter here (p. 81), the present writer quoted data, concerning the children of consanguineous parents, which suggested that, occasionally, recessive hereditary factors may be decisive in producing amentia of no specific type. To speak of subcultural mental deficiency in general as due to a single recessive gene, however, would be nonsense. According to Pearson, there is a continuous gradation of intelligence, and it is perfectly idle to speak of mendelian units in respect of such a character.

Environmental Factors.—Since environment is found to have an influence on intelligence in normal children, it is reasonable to look carefully for its effect when dealing with subcultural mental defectives. The order of birth in the family has to be considered as a possible origin of variation in mental qualities. Shrubsall's table in the L.C.C. Report (1912) showed that defectives, brought up for examination in the London area, were most frequent towards the end of the family. A similar effect is noticeable in the large families in Dayton's statistical survey.[2] The work of Thurstone and Jenkins, who take the view, also held by Steckel, that the mean intelligence quotient increases with birth order, has already been referred

[1] Sjögren, T., *Acta Psychiatrica et Neurologica*, Supplementum II, 1932, Levin and Munksgaard, Copenhagen.

[2] Dayton, N. A., *American Journal of Psychiatry*, 1929, Vol. VIII, p. 979.

to and criticized (p. 69). The question of selection, however, may also enter into the results which tend to the opposite conclusion. The present writer found that pure subcultural aments were somewhat less frequently born to young mothers than to those more elderly, but the result may possibly be vitiated by incomplete ascertainment of the mentality of all the children in the families.

The following table shows the proportions of children of various degrees of intelligence born at different maternal ages. The data are 94 sibships each containing at least one subcultural ament :

Mentality of Children.	Maternal Age at Birth of Offspring.			
	Below 25 yrs.	25 yrs. to 29 yrs.	30 yrs. to 34 yrs.	35 yrs. and above.
	%	%	%	%
Normal	42	33	29	31
Dull	8	8	10	12
Mentally defective . .	19	37	35	29
Unknown	31	32	26	28
Total number of children	(119)	(153)	(136)	(164)

The correlation between subcultural amentia and low social standing may also be, to a great extent, environmental in origin. Children who grow up in families where the parents are ignorant and dull suffer both in physical and in mental nutrition. In the present state of our knowledge it cannot be assumed that, if a defective child comes from a low social class, the defect is specially likely to be determined by heredity. On the contrary, it is really more likely, if the home environment is very bad, that, in such a case, part of the effect is due to this bad environment. When subcultural amentia occurs in the upper classes, the probability that heredity is the cause is correspondingly greater.

Illustrative Cases.—The photographs which accompany this chapter are of three patients whom I would classify as subcultural.

Of the two lads, the one on the right is aged 28 and has a Stanford-Binet mental age of 10 years 7 months. He has quite a good physique and plays a good game at football. His cranial capacity is 1,595 c.c. His ability at performance tests (Porteus Maze, Healy Nos 1 and 2)

is normal. This patient came under the notice of the authorities for stealing eggs at the age of 6, and at 8½ years old he was sent to an industrial school. At the age of 15 he was charged in Court with housebreaking and the Bench made an order for his admission to a certified institution. He has been tried in a great number of employment situations but has failed in every case to give satisfaction. He has an elder sister, who is certified mentally defective, whose mental age is scarcely 7 years and is, therefore, almost of imbecile grade. Her physique is poor. She has never shown any anti-social tendencies. There are 12 more brothers and sisters, all of whom passed as normal at school, though they were not thought to be bright, and 10 of these who survive are all living in the general community and most of them are married. One brother was a sergeant in the army. Two siblings died in childhood. One of the children of the eldest sister died of juvenile general paralysis in a certified institution. The father has earned his living as a farm labourer and as a bricklayer. The mother is a garrulous and excitable woman, not very intelligent. If the members of this family group were tested by standardized mental tests, few would be found to have a mental age of 14 years. The lad, whose history is given, however, was unfortunate enough to possess anti-social character traits, and the result has been certification under the Mental Deficiency Acts.

The boy standing on the left is aged 17 and has a mental age of 10 to 11 years. He also is better at performance tests than scholastic achievement. Physically normal, he is very well behaved under supervision, but easily led away if he gets into bad companionship. He was sent to a residential Special School when aged 12 after being before the Justices on several charges of petty theft. The family history is not dissimilar to that in the last case, but the social and intellectual standing is a little higher. In a time of industrial depression it is extremely hard to find suitable employment situations for patients where they will be unaffected by influences of an anti-social nature : but an opportunity for this boy has now been found.

The girl (see photograph facing p. 148) is aged 28 and has a mental age of 11 years. At the age of 13 she was rescued from a very unsatisfactory home environment and placed in a Salvation Army Home. Her parents were unable to care properly for her owing to their very poor financial circumstances. She had been frequently in the company of what she termed " bad girls." One brother is probably feeble-minded, but another is normal.

At the Home she was unmanageable on account of fits of temper and she was later transferred to a residential Special School and, on attaining the age of 16, certified feeble-minded. She has been since boarded out on licence in several situations, in one of which she

High Grade Feeble-Minded Boys. (See p. 153).

[*To face p.* 154

was grossly overworked. In similar cases the threat of sending the patient back to an institution is sometimes used to exploit her. Now she is employed as a domestic servant and is giving satisfaction.

Cases such as the three described above are not at all uncommon, and serve to illustrate the complex relationship between social class, character and intelligence, which determine whether or not persons of subcultural mentality are certified under the Mental Deficiency Acts. These complexities are particularly marked in cases like those described, who are all high grade. Lower grade subcultural cases are often quite incapable of carrying on in the outside world whether their character is steady or anti-social.

CHAPTER XV

TREATMENT

General Considerations — Radical Treatment — Socialization — Symptomatic Treatment—Training—Preventive Treatment—Prevention of Offspring—Prohibition of Marriage—Contraception—Segregation—Sterilization—Conclusion.

General Considerations.—In so far as mental deficiency can be regarded as a group of diseases, the condition of the patients demands the attention of medical science. When medical science sets out to free the patient from a disease entirely, a *radical* method of treatment is adopted. Since many of these diseases are of the congenital type and exist before birth, the majority of attempts at radical treatment which have been made have ended in complete failure. Thus, the physician often has to be content with *symptomatic* treatment, that is to say, he confines his energies to alleviating or removing the effects of a disease which he cannot eradicate. Of late, however, the importance of *preventive* medicine has come into prominence in medical thought, especially in the consideration of incurable diseases.

It is doubtful whether it is permissible to regard pure subcultural mental deficiency as a diseased condition, particularly when the subject is feeble-minded. If he represents part of the normal variation, it is useless to apply the standards of existing medical science to his condition except in so far as it will be the physician's duty to see that his body is healthy and his mind is at ease. It may, however, be argued that subcultural mentality is in itself one of the causes of mental and physical disease in the actual subjects possessing it, in their offspring and among the members of the society in which they live. They can neither look after their own nor their children's health. They act as a kind of dead weight which the social group has to drag along in its progressive efforts in hygiene. It is, thus, arguable that this group of

persons, though themselves technically normal, may constitute a danger to the community and that, therefore, any preventive system which will ensure that their numbers do not increase but decrease is justifiable on ordinary medical grounds.

Radical Treatment.—Although, in general, the radical treatment of mental deficiency is a depressing subject, in certain cases cures have been effected or there are indications that further research may discover curative treatment.

A good example of an attempt at radical treatment which proved entirely useless was the trephining of the skulls of microcephalics. This was a popular operation about forty years ago and the idea of the treatment was based on the false assumption that the brain was small because the skull failed to expand : the converse assumption is probably correct.

The standard example of beneficial results derived from medical treatment in aments, is the improvement noticed in cretins when they are fed upon thyroid gland extract. Unfortunately many cretins, though their physical symptoms may improve, do not progress far mentally and remain imbeciles after treatment. But there are examples, which have been carefully checked by mental testing, where a cretin imbecile, treated with thyroid from an early age, has developed mental capacity close to the average.

Successes have been recorded in other fields of endocrine disorder. For example, it is recorded that subjects with pituitary gland dystrophy may show improvement, physically and mentally, by suitable administration of pituitary extracts. The results, however, are not so dramatic as in the case of uncomplicated cretinism.

Jaensch reported a number of cases with supposed pituitary disorder, associated with mental deficiency, which he considered he had cured by the exhibition of large quantities of organically combined iodine. In these cases the patients all showed, at first, marked deformity of the end capillaries at the base of the finger nails and, after treatment, the deformed capillaries became much more normal. If this work can be repeated and properly controlled, the treatment may prove of the greatest value.

The treatment of juvenile general paralysis by malaria therapy has not been, up to the present time, encouraging. The physical state is sometimes a little improved, but no increase in the mental powers has been observed in these cases. It is not, however, impossible that a curative treatment may be found for juvenile general paralysis of the insane, since the similar condition in adults is quite often curable. In other types of congenital syphilis, the ordinary anti-syphilitic measures have the effect of stopping the advance of the disease and stopping mental deterioration, though it is doubtful whether any mental improvement is produced by the treatment.

In certain cases where a child is deprived of the benefits of ordinary educational methods for any reason, such as blindness, deafness or lack of opportunity, the resulting defect in education will quite possibly amount to certifiable mental deficiency. It can be remedied by the provision of adequate special methods of teaching. Interesting examples involving problems of educational backwardness are sometimes provided by children who have been neglected by their parents, or native children who have been brought up without education and who suddenly find themselves in an educated community. In such instances it may be exceedingly difficult to tell beforehand whether a given child will profit by special education or not. When the deprivation is sensorial, the remedy is very arduous and requires great patience on the part of both pupil and teacher. Blind or deaf children who are also initially dull are almost certain to fail to reach normal standards of intelligence, however well they are taught.

A special type of problem is presented by persons who are able to perform mental tests fairly satisfactorily, or perhaps even very well, but who, by reason of difficulties in their personal temperament and character, are either unable to learn or unable to respect the usual canons of behaviour current in the society in which they live. Such persons, if they become adult before they fall under the observation of medical authorities, may become confirmed criminals—perhaps being convicted over and over again for petty thefts—and they end

up either in gaol or in an institution for mental defectives. If recognized at the school age, they may be very accessible to psycho-analysis. Child guidance clinics sometimes succeed in curing such individuals of their neuroses and enabling them to adjust their temperaments first of all to learning in the school environment and later on to filling some useful place in the general community. The fundamental disorder in these persons, who, if they have no private means, are sometimes certified as moral defectives, is an inability to maintain satisfactory personal relationships. The disorder may be so severe that it ranks as a psychosis and then, in the present state of knowledge, a cure is very unlikely to be brought about. A great number of them are, however, merely psychoneurotics ; and the present view of the majority of psychiatrists is that neurosis is curable. A practical problem is to find people who are willing to spend sufficient time and trouble in treating children, of the poorer classes, suffering from curable neuroses which make them anti-social.

I have mentioned that subcultural mental deficiency hardly ranks as a disease. We, therefore, cannot expect to cure it. Nevertheless, the subcultural mental defectives who find their way into institutions are often there because they exhibit signs of mental disorder. In such cases, although the mental deficiency itself cannot be cured, the neurotic disorder which makes certification a necessity may be tackled by the physician. Even when not suffering from obvious neurosis, dull or defective children, who have been sent to Special Schools, may afterwards drift into a life of crime through ignorance and neglect. On this point the Departmental Committee on Persistent Offenders, 1932, reported as follows :

> We call attention to this matter as, even without the provision of special education, much could be done to prevent the formation of criminal habits in these defectives if they were under constant friendly supervision whilst in school and after leaving school.

Socialization.—The theory of socialization of defectives both in and outside institutions, which must be regarded as a form of radical treatment, has obtained popularity both in England

and in America[1] and is worthy of very careful consideration. It has been found that mentally defective persons, who are difficult to control at home, perhaps violent and obstructive, may become quiet and amiable when subjected to institutional care. On the whole they tend to become more and more sociable as time goes on and, in this way, they become relatively more useful to the community, if only by learning to look after one another. Very few mental defectives are, in actual fact, fully cured by socialization and returned to the community; but there is reason to believe that many more of them could be returned if the original circumstances which stimulated them to react violently or obstructively could be removed. In the present period of economic depression, unemployment and lack of commercial enterprise, it is not to be expected that persons who have been ejected from the community for inefficiency will be favourably received back again even if they have been taught wisdom in the meantime. The patients are first tried out *on licence*, working under supervision, sometimes with friends or foster-parents. A good practical rule is to stipulate that they must retain the same post for three years before they can be discharged. The following figures give an idea of the frequency of licence cases in a large institution.

REPORT OF CASES ON LICENCE UNDER THE CARE OF THE ROYAL EASTERN COUNTIES INSTITUTION, 1932.

	Males.	Females.	Total.
Wage earning	28	22	50
Working, not earning	13	34	47
Not able to work	4	7	11
In Public Assistance Institution	3	18	21
Total	48	81	129
Total Population of Institution .	842	585	1,427

[1] Davies, S. P., *Social Control of the Mentally Deficient*. Constable, London, 1930.

The socializing process is nothing more than an extended application of the original philanthropic work of the middle of last century, but the scope has been widened, particularly for high grade patients. Defectives are not only taught trades and handicrafts, but they are taught how to amuse themselves. Games of all kinds are greatly encouraged, and one of the most popular amusements is physical drill. Perhaps nothing has contributed so much to the pleasure of mental defectives as the boy scout and girl guide movements. Patients are also encouraged to get up entertainments for one another—acting plays, performing at concerts and so on. If all this is backed up by a sympathetic understanding on the part of the medical and nursing staff, the patients can be made very much happier in an institution than they ever would have been in their original surroundings. An important factor is that the element of competition with people who are cleverer is removed and they are able to feel that, at any rate, they can be good at something.

Religious teaching is considered a valuable adjunct to social training in many institutions. As may be gathered from the following paragraph, written in 1854, the idea is not new.

> It is the constant experience of all teachers of the idiot—and I have often heard Dr. Guggenbühl express the same of the Cretins—that religion, and the simple facts and precepts of the sacred Scriptures, make the deepest impression ; and that there is granted to them the beneficial compensation of a remarkable facility for understanding them.[1]

Symptomatic Treatment.—So far, we have discussed forms of treatment which aim at cure or at least at restoring the mentally defective to the community as useful units. These radical methods are, however, usually not entirely successful, on the one hand owing to the small number of cases susceptible to curative medical treatment and, on the other hand, to economic and social difficulties inherent in society itself. When the physician abandons his aim at radical cure he adopts a new aim, namely that of removing or alleviating symptoms. There is often not very much practical difference between the

[1] Sidney, Rev. E., *Teaching the Idiot*, published on behalf of the Asylum for Idiots, London, 1857.

two methods of treatment, but the physician, in the latter instance, guards himself against disappointment.

The great bulk of the medical treatment of the mentally deficient is of the palliative type. Epilepsy, for example, which is such a frequent symptom, can be satisfactorily controlled in most instances either by bromide or barbitonum derivatives, such as luminal. In the days before the latter preparations were on the market, *status epilepticus* was a frequent occurrence and caused a great many deaths in institutions. At the present time, considering the number of epileptic patients in institutions, it has become quite a rarity. The stultifying effect of continuous treatment with sedative drugs is sometimes noticeable in mentally defective patients, but one cannot feel that here the tragedy is so great as in the case of epileptics of normal intelligence. The proper control of epilepsy tends very much to lighten the burden not only of the patients themselves but also of the staff.

Encephalitis lethargica has been the subject of a good deal of human experimental pharmacology. So far, no really satisfactory drug has been discovered which will permanently relieve the mental symptoms in these cases—at least, there is nothing better than stramonium, and this is also advocated as the best drug in cases of post-encephalitic Parkinsonism. There seems to be no reason, though, for supposing that this field of research is exhausted. If a drug can be discovered which would, in some way, control the disordered impulses of the emotional centres without adversely affecting cortical activity, a great change might be seen in the treatment of post-encephalitic mental defect.

Certain rather obvious kinds of medical attention often have favourable effects on the mentality of patients, such as the proper correction of vision by spectacles. In mongolism, for example, there may be very marked myopia and, where the subject shows ability to take advantage of the most elementary education in handwork or in reading, the provision of suitable glasses may make a difference to their ability.

The orthopædic side of mental deficiency deserves further study. In America, systematic training in the use of paralysed

limbs has been carried out in the experimental programme of the Vineland Training School. It is not uncommon to find that paralytic patients have already been operated on by an orthopædic surgeon before entering an institution and it is surprising how rarely such operations succeed in effecting any permanent benefit. The favourite operation is usually tenotomy in the case of spastic diplegia and the usual result is to produce merely a new deformity in place of the old one. The failure of these operations cannot be attributed entirely to the surgical methods employed, but must be considered in the light of the inability of the subject to make a proper use of the new movements afforded to the body. A child who has not sufficient brains to learn to walk will not do so in response to the efforts of orthopædic treatment. Even the somewhat higher grade patient may not react soon enough to take full advantage of the treatment. Probably complete fixation in a useful position is the only surgical orthopædic method suitable for a joint when the child is known to be mentally defective. This gives an opportunity for the child to learn to use the limb later on in life. In rickets, straightening of limbs has been successful in defective children.

Massage and hot baths have their place in the treatment of incipient paralytic contractures ; but this form of treatment requires such tremendous perseverance that it is seldom employed.

Training.—We now come to the consideration of special training of the mentally defective which, although not attempting to produce normal individuals, aims at making the maximum out of the material available. The training of low grade cases follows lines substantially the same as those laid down by Montessori, Séguin and others many years ago, and children are classed together who have similar mental ages whatever may be their physical ages. In the first place they have to be taught coordination of the larger movements which come into play, for instance, in walking, climbing a ladder or catching a ball. Simple occupations such as cleaning spoons, building with blocks and unpicking threads come next : later on, matching colours, modelling, making mats and drawing.

Education of higher grade children, the feeble-minded and the dull, is carried out in the Special Schools, though opinions differ considerably as to the desirability of this or that form of training. Kennedy-Fraser [1] has laid down very clearly the most profitable lines on which the training of backward children can be undertaken. Formerly, it was frequently necessary to teach *adult* defectives handwork and simple educational attainments but, with the present working of the Mental Deficiency Acts, this necessity is becoming increasingly rarer. The training of adult defectives is in no way essentially different from the training of backward children but, as is to be expected, good results are not so easily obtained.

A striking feature of defectives of imbecile and lower grades is their apparent incapacity for being bored with an occupation ; and, provided some simple manipulation can be taught, the defective is perfectly happy in continuing the same manipulation for days and years without any change. This fact makes possible methods of dealing with patients who might otherwise be difficult to occupy. In a regular, even if very monotonous, employment they learn to be useful and worthy people. With high grade patients this monotony is not appreciated. The work must be sufficiently skilled and have enough purpose to keep them interested. Many of them will do very good work under supervision with frequent changes of occupation, but otherwise will do nothing.

Preventive Treatment.—The first consideration in the prevention of mental deficiency is to consider how environmental influences which are held responsible can be modified. In the course of the description of pathological types, I have pointed out that the early environment of the individual is, apparently, sometimes a decisive factor.

First of all, the health of the mother has to be considered from the time of conception onwards. Abnormalities which might adversely affect the intra-uterine environment must be, as far as possible, corrected. Deficiencies in diet and signs of endocrine disorder should be attended to. For example,

[1] Kennedy-Fraser, D., *Education of the Backward Child,* University of London Press, 1932.

a woman, otherwise healthy, who tends to have frequent miscarriages, may benefit by corpus luteum therapy. Pelvic irradiation, either by X-rays or radium, should be avoided in the early months. If the mother is suspected of syphilis, blood tests should be carried out. Anti-syphilitic treatment during pregnancy is believed by many authorities to prevent the child from suffering from the congenital form of the disease. As pregnancy advances, ante-natal supervision takes into consideration the size of the pelvic outlet and the size and the presentation of the fœtal head. Frequent ante-natal examinations, together with skilful midwifery, may do much to avoid birth injuries.

Care of the infant, avoidance of trauma and acute infection, and proper diet, rich in the vitamins A, B, C, and D, can all be regarded as measures indirectly helping mental as well as physical development.

At the present time, nearly all children have the same opportunity of reaching a certain average standard of education, though, in homes where a child is kept away from school on account of its own ill health or the illnesses of its relatives, the opportunities for education may be considerably lessened. It is to be expected that a general all round improvement in standards of living would have some effect, if only a slight one, on the incidence of mental deficiency. It has been shown that patients certified feeble-minded suffer frequently from neurotic behaviour traits which may be largely determined by or intensified by environment. In the sense that improved home environment might cause improvement in behaviour in the children, we might expect that improved social conditions would diminish the amount of mental deficiency due to neurosis or character difficulties. Similarly, since social class is itself a factor determining liability to certification, the financial improvement of any social group would tend to diminish the number of defectives in that group. Social and economic alterations could change the problem of mental deficiency as seen in the eyes of the law. Biologically, the actual intelligence of individuals is a more interesting and less artificial characteristic than social status. Alterations in the social

order are not likely to have much effect on innate mental capacity. Although not really prophylactic treatment of mental deficiency in the medical sense, they might solve the problem socially.

Prevention of Offspring.—The most obvious form of prophylactic treatment of any condition which occurs with high frequency in members of the same family, whether it is due to heredity or not, is some way of preventing the birth of offspring. This prevention may operate at various stages in the life cycle.

First of all, one may consider infanticide, or some kind of judicial destruction of individuals who are obviously seriously defective. Quite apart from the moral questions which are raised by a suggestion of this kind in civilized communities, it must be at once pointed out that the great majority of mental defectives appear normal at birth, at least 90 per cent. Moreover a great number of children who turn out to be normal mentally appear abnormal at birth, *e.g.* those suffering from hare-lip. It would be much easier to destroy at birth or at an early age individuals suffering from physical defects than those suffering from mental defects. We are not considering here the eugenic problem as a whole but only its relation to mental deficiency. We see that the part of the problem with which we are concerned is particularly difficult.

Next comes the question of the encouragement of abortion where the birth of a mentally defective child is supposed to be imminent. To those who are interested in the subject, the suggestion that abortion might be allowed to mentally defective females is worthy of consideration. Very few of the mentally defective women who have illegitimate children, for example, are anxious to produce offspring and they tend to take less than normal interest in them when they are born. Public opinion, however, seems to be more prepared to consider other possible legislative methods for controlling the births of children of feeble-minded parents.

We now come to the question of prophylaxis of mental deficiency by prevention of conception. Four practical

possibilities, and other possibilities not so practical,[1] have been put forward, but only two of them have received much serious consideration. The possibilities are discussed in the next four sections.

Prohibition of Marriage.—Prohibition of marriage of mentally defective persons has been recently suggested by the Board of Control as a means of coping with the problem. This idea has received little general support, mainly because it is thought that an ordinance of this kind would merely tend to increase the number of the illegitimate, as opposed to the legitimate, offspring of mentally defective mothers. The suggestion was made partly from the point of view of simplifying administration and partly because of the existence of a popular belief that marriage cures mental deficiency. It is much more difficult to certify a married woman than a single one since it may be difficult to prove that she is not adequately cared for by her husband.

Contraception.—Provision might be made for the education of mentally defective persons in methods of birth control. One cannot, however, expect persons who are of subnormal mentality to be able to carry out methods of birth control unless they are very simple and foolproof in character, and, at present, no such methods have been discovered. At the present time it would probably be impossible to introduce legislation to enforce on mentally defective persons, or other persons supposed to be likely to have mentally defective children, the use of contraceptive methods, but it is not impossible that biological discovery will alter this state of affairs.

Segregation.—The method of controlling mental deficiency, which is now most widely adopted, is based on the assumption that the majority of mentally defective persons require lifelong

[1] Among the remedies, suggested at the meeting of the Research Committees of the Eugenics Section of the American Breeders' Association at Palmer, Mass., in May, 1911, as possibly efficacious " for purging from the blood of the race the innately defective strains," were the following :— systems of matings purporting to remove defective traits, polygamy, *laissez-faire*. Laughlin, H. H., Report of the Committee to Study and to Report on the Best Practical Means of Cutting off the Defective Germ-Plasm in the American Population. *Eugenics Record Office Bulletin*, No. 10–A, Cold Spring Harbor, 1914.

supervision and control in an institution. The curative process of socialization, which has been referred to, is successful in relatively few cases—though one must not underrate the possibilities of this method by noticing only the results in times of industrial depression like the present. Some persons are of the opinion that segregation is inhuman and cruel. A great deal of such opinion is quite mistaken and is held by those people who have no experience of the behaviour of mental defectives under institutional conditions. The great majority of them are happier in an institution than they would be in the outside world. Of course, the hardships or otherwise which the patients have to undergo by reason of their confinement in any given colony depend on both the attitude of the staff and the temperaments of the patients themselves. Under the best conditions, however, it is not difficult to believe that they are extremely comfortable. Permanent institutional care, however, for high grade cases is sometimes irksome both from the point of view of the patient and of the staff. It has been advocated, on humanitarian grounds, that permanent sterilization should be allowed to such persons. This would, it is thought, enable them, after institutional training, to be discharged into the community where they could enjoy the pleasures of a normal existence and could embark on matrimony, avoiding the extra responsibility of having children. It is urged, for example, that a female defective may be capable of looking after a house, keeping it clean for herself and her husband, but be totally incapable of bringing up a child. In this way we are led to the consideration of the fourth possible method of prevention of the birth of mentally defective children.

Sterilization.—A popular view at the present time can roughly be expressed as follows. There are in England and Wales some three hundred thousand mentally defective persons : a small proportion of these are cared for in institutions at the average cost of £1 per week. Something like half of this cost comes from the Government. The remaining defectives who are not in institutions are at large in the community and are breeding at a prodigious rate, while the rest

of the population is diminishing. Thus, in a few years' time, institutional provision will have to be made, at enormous expense, for a greatly increased number of mental defectives. Therefore, it is urged, let us sterilize the two hundred thousand or more uncertified defectives in the general community and then, when those in institutions have died, there will be no more. Unfortunately this simple view is subject to a great number of fallacies which invalidate it.

The first point is statistical. It is found that something like 5 per cent. of all defectives have one or other parent mentally defective. It does not matter exactly what the figure is, whether it is 3 per cent. or 7 per cent., the argument is the same. Only 5 per cent. of the next generation of mentally defective persons would therefore be prevented if *all* existing mentally defective parents were compulsorily sterilized. Furthermore, a large proportion of existing defectives never could have children because their mentality is of such low grade that they would never, in the ordinary way, find a mate. The only defectives, therefore, whom it would be worth while sterilizing are those of mentality high enough and physique complete enough to enable them to procreate offspring. Practically, this means persons with I.Q. over 40. Now, at any rate in the present economic conditions, the vast majority of mental defectives whose I.Q. is under about 60, as well as a great many above this grade, are unable to earn their own living. Sooner or later, they drift into institutions. They would do so whether they were sterilized or not. With the present system of ascertainment and with the cooperation of the education authorities, an increasing number of juvenile cases come under institutional care, supervision or guardianship, before they have a chance to have children. We have therefore only to consider the group of mentally defective individuals with I.Q. above 60, that is to say, with a mental age of nine years or more. Most people are agreed that these individuals make relatively undesirable parents. But here we are up against a serious difficulty. When an adult has a mental age of nine or ten years, it becomes a matter of expert opinion whether he is, or is not, mentally defective. Many authorities (Shrubsall

and Williams, Kennedy-Fraser) consider the mental ages between nine and ten to constitute the border line. Those persons who are actually certified mentally defective, whose mental age is nine or over, are, for the most part, selected on account of some kind of behaviour difficulty which brings them to the notice of the authorities. They have, in fact, to be put under institutional care on account of their behaviour difficulty. The dilemma is now obvious. As soon as border line cases are recognized as undoubtedly mentally defective, they " require care, supervision and control for their own protection or the protection of others." What would be the use of bringing in legislation to sterilize the particular persons who are already adequately catered for and segregated by the present law ? It is obvious that only some form of compulsory sterilization of all persons with mental age or I.Q. below or between certain limits would suffice. To bring about such a state of affairs would necessitate a much more radical change in the outlook of society than would be needed in order to legalize abortion.

At present the only question before the public is that of voluntary sterilization. Discussing this, Turner, who has had considerable experience in dealing with the high grade feeble-minded, writes as follows :

> The advocates of sterilization at present suggest that it should be voluntary, though all those I know agree that they look on this voluntary sterilization as merely the thin end of the wedge. I am unable to understand how it can be suggested that mental defectives who have been certified and found to be incapable of managing their affairs or to need care, supervision and control for their own protection can be supposed to be able to decide voluntarily on the question whether or no they should be sterilized. I do not include those defectives found to need care, supervision and control for the protection of others. Obviously they cannot be discharged in any case and their decision on the matter cannot be considered trustworthy. The label voluntary as applied to the sterilization of a certified defective seems to me, in fact, to be a contradiction in terms, and I do not think even the highest grade of them are capable of forming an opinion on the matter. Defectives, however, are notoriously suggestible. The very great majority of them could be induced to accept operation or not by anyone they trusted. I venture to say I should not be fitted to hold my present office of medical superintendent

of an institution for the care of the mentally defective if I could not induce practically every one of my patients to be operated on or to refuse operation just as I myself might wish.

In spite of the various objections, administrative, humanitarian or biological, which have been brought forward in opposition to a proposed legislation for sterilizing mental defectives, the fact remains that in many States in America such laws have been passed, and also in other countries. The legislation can in no case be said to have advanced much beyond the experimental stage, and the examples of the workings of such Acts in some instances have not been very encouraging from the eugenic point of view. For a full account of the question the reader is referred to Landman's [1] historical summary. Landman shows clearly that the dreams of the eugenic prophets as to the value of sterilization have not been realised though, of course, it is too early yet to make any judgment on the ultimate effect of these laws. He reports that, since 1907, in all, some twelve thousand sterilization operations have been performed in the United States for eugenic purposes out of a total population of some 137 million. The majority of these operations appear to have been performed on insane persons. In certain states the legislation has been a failure. For example, in New York, legislation was enacted in 1912 allowing for the sterilization of criminals, feeble-minded and epileptics. Writing in 1923, Davies [2] described the sequence of events thus :

The net results of the New York sterilization statute were practically nil. Thirty state institutions were subject to the act and yet only one operation was performed under its authority. In the spring of 1915 a test case was introduced in the New York State courts and the statute was held unconstitutional and invalid by the State Supreme Court—Albany County. The case was appealed and continued in litigation until the statute was repealed on May 10, 1920.

In view of the great difficulties which confront the legislators in bringing in this type of law and also in carrying it into execution, it is rather puzzling sometimes to understand

[1] Landman, J. H., *Human Sterilization*. Macmillan, New York, 1932.
[2] Davies, S. P., *The Social Control of the Feeble Minded*. National Committee for Mental Hygiene, 1923.

why it should have such popularity in so many quarters. It must be obvious to the majority of clear-headed people that the eugenic effect of such measures will be exceedingly small as compared with the labour expended in making them operative. It would be less troublesome to deal with the few people who would be covered by such a law by the recognized methods of segregation. In some cases, as has already been pointed out, the demand for legislation providing for the sterilization of mental defectives may be the result of humanitarian motives. It may be supposed, for example, that defectives in institutions would be allowed more holidays if they were sterilized, even if the operation would not make them fit to go back permanently into the community. But there is no evidence of any such necessity for sterilization before ordinary holidays or paroles are granted.

In some cases the demand for this kind of legislation may be a political subterfuge to further humanitarian or eugenic interests in other quarters. When we are dealing with other diseases than those associated with mental deficiency, entirely different considerations come into the question of the value of sterilization. For example, a man suffering from a severe physical deformity, such as split-hand, but who was entirely normal mentally, would be aware that half his children would be likely to be affected in a similar way, knowing the deformity to be inherited as a mendelian dominant. This man would have a right to demand from society that he should be allowed to be voluntarily sterilized so that he could enjoy the privileges of matrimony without the serious consequences which might otherwise follow from this action. Now it is possible that political opinion might be unfavourable to the introduction of legislation for the purpose of voluntary sterilization of mentally normal persons afflicted with hereditary diseases, or known to be carriers of such diseases, but that it might be quite favourable to suggested legislation of the same kind where the mentally defective are concerned. If so, this still leaves the problem unsolved as to why public opinion should be specially attracted to the idea of sterilization of mentally defective, rather than physically defective, individuals.

A great deal of the agitation for sterilization of the mentally defective is certainly not the outcome of humanitarian views. At the back of the minds of many people is the notion that sterilization should be a punitive measure. In certain countries, Denmark for instance, punitive laws are actually in force. The following extract from an American source shows the length to which the aggressive impulses of persons proposing this type of legislation may carry them.

> Whenever a person shall have been convicted of murder (not in the heat of passion), rape, highway robbery, chicken stealing, bombing, or theft of automobiles, the judge trying the said case, shall immediately upon final disposition of the case, if conviction is upheld, appoint a competent physician, resident of the county in which the conviction was had, to perform on said convict the operation known as vasectomy or oöphorectomy for the purpose of sterilizing such convict, so that the power to procreate will be forever destroyed.[1]

The operation of castration has often been recommended as a punitive measure for sexual offenders. About twenty such cases have been dealt with in Michigan since 1925, when the Sexual Sterilization Act was passed there. A young man, for instance, it is reported, who was found lying in wait for his schoolmistress, was ordered by the Court to be castrated in a State Hospital. After the operation he went home, and now works quietly and happily on the farm with his mother. Before the operation this young man was quite unmanageable.[2] It is unbelievable that such a case could not have been dealt with in other ways more in keeping with modern humanitarian standards.

The greatest psychiatrist of modern times, Sigmund Freud, has pointed out very clearly that some of the most serious troubles affecting civilization come from man's imperfect mastery over his aggressive impulses against his neighbour.[3] It is a well-known psychological mechanism that hatred, which is repressed under normal circumstances, may become manifest

[1] Section 1 of House Bill No. 290, introduced by Mr. Bellew of the Missouri State legislature. Cf. *Mental Hygiene Bulletin*, April, 1929.

[2] Pocock, H. F., *Eugenics Review*, Vol. XXIV, No. 2, July, 1932, pp. 127–30.

[3] Freud, S., *Das Unbehagen in der Kultur*. Internationaler Psychoanalytischer Verlag, Wien, 1930.

in the presence of an object which is already discredited in some way. Conscience, super-ego or whatever it may be termed which preserves the individual from assaulting his neighbour or expressing a public wish to mutilate his private enemy, is removed when a socially or politically abhorrent class of persons is concerned. An excuse for viewing mentally defective individuals with abhorrence is the idea that those at large enjoy themselves sexually in ways which are forbidden or difficult to accomplish in the higher strata of society. The association between the idea of the supposed fecundity of the feeble-minded and the need for their sterilization is apparently rational, but it may be emphasized by an unconscious desire to forbid these supposed sexual excesses. It has been pointed out that the advocates of sterilization never desire it to be applied to their own class, but always to someone else. In the present writer's view, unconscious motives have contributed largely to the prominence of the question of sterilization of mental defectives in the public eye.

Actually, when put in its true proportion to other world problems, " the menace of the feeble-minded " is a question of very moderate dimensions. At present public funds provide an annual expenditure of one to one and a half million pounds on mental deficiency. It might cost the state three or four times as much to provide, without charitable aid, institutional accommodation for all the mentally defective who need this kind of care. But one hundred million pounds or thereabouts are spent annually on the fighting services. It would be possible to cope with the whole problem of mental deficiency in England by a comparatively small reduction in the expenditure under this heading alone.

Conclusion.—Since mental deficiency is not a clinical or biological entity, but only a legal concept useful for social and administrative purposes, the problems concerning the subject have both a social and political as well as a scientific and medical significance. From the scientific point of view, these problems can be coped with systematically after sorting out the different types of individuals to be found legally classified as mentally defective. But the problems have also to be

tackled from the social point of view. We have to remember that there will always be, in any human society, brilliant people and simpletons, and what to do with the simpletons will always remain a social problem. This is a question which civilization has to face. A society which is ideally conducted will have to make arrangements so that the simpletons can find a useful purpose in their existence.

The pathological types of mental deficiency remain, under any circumstances, a problem primarily of interest to the medical scientist. The research worker in this field may well be thankful sometimes that it is customary to preserve these oddities, who are thus available for study and are not put by law into a lethal chamber at birth.

GLOSSARY

Achondroplasia—a congenital abnormality affecting cartilaginous bones : the limbs are very much shortened.
Acromegaly—excessive growth of the lower jaw and the extremities, due to pituitary hypersecretion.
Agglutinogen—forerunner of a substance which causes agglutination of red blood corpuscles.
Amaurosis—progressive visual failure.
Amniotic Sac—innermost membrane enclosing the fœtus.
Anaphylaxis—sensitivity to specific proteins, as in hay fever, asthma, etc.
Anencephaly—a condition in which the brain and the cranial bones are rudimentary : affected infants do not survive.
Ataxia—unsteadiness in movement.
Athetosis—involuntary squirming movements.
Auditory Meatus—channel of the ear.
Autosome—any chromosome which is not an X or Y (sex) chromosome.

Brachycephalic—short-headed.

Catatonia—stupor associated with peculiar rigidity of the limbs.
Choroiditis—inflammation or degeneration of the pigmented lining of the eye.
Chromosome—filaments peculiar to the nucleus of a living cell : in man there are twenty-four pairs to each cell nucleus.
Coloboma—maldevelopment of the inner structure of the eye, leaving a cleft.
Cornea—thin transparent anterior covering of the eye.
Cutaneous—superficial ; in the skin.

Dehiscence of Teeth—shedding of the first teeth at the second dentition.
Dementia Præcox—a common type of mental disease often commencing at puberty : an affected person lives in a world of his own.
Diathesis—constitutional predisposition.
Diplegia—paralysis of both legs.
Dolichocephalic—long-headed.

Echolalia—mimicry of words.
Echopraxia—mimicry of actions.
Encephalitis Lethargica—epidemic encephalitis, an infective disease of the nervous system usually associated with fever, lethargy and ocular palsies, popularly spoken of as "sleepy sickness" : physical after effects are sometimes known as "Parkinsonism."

Endocrine—secretion of ductless gland.

Flaccid Paralysis—weakness of a limb with loss of power of movement and muscular wasting.

Gene—that part of the chromosome which is identified with a particular factor of inheritance.
General Paralysis of the Insane—mental and physical changes progressing to complete dementia and paralysis: due to syphilis.
Germ Cell—part of an organism specialized to develop into a new organism (ova, sperms).

Hæmoglobin—the red substance in the blood which contains iron: the Hæmoglobin Index is a measure of the amount of hæmoglobin in the blood.
Hallux—the big toe.
Hemiplegia—paralysis of arm and leg on the same side.
Heterozygous—when a gene is present on one chromosome of a pair but not on the other, the individual is said to be heterozygous for the character difference determined by such a gene.
Homozygous—when a gene is present in duplicate, an individual is said to be homozygous for a character difference determined by such a gene.
Hormone—internal secretion conveyed by the blood.
Hutchinson's Teeth—peg-shaped or notched incisors.
Hypertelorism—mental deficiency associated with abnormal width between the eyes.

Intelligence Quotient—the ratio of mental age to chronological age expressed as a percentage.
Ideational Inertia—difficulty in moving from one idea to another.

Keratitis—inflammation of the cornea.

Lenticular Degeneration—a disease of a group of nerve cells in the mid-brain associated with hardening of the liver.
Leptocephalic—narrow-headed.
Luetic—due to syphilis.
Lymphocytosis—increase in the number of lymph cells in the blood.

Macula—the most sensitive part of the retina.
Maxilla—upper jaw.
Meninges—membranes covering the brain and spinal cord.
Monoplegia—paralysis of one limb.
Morbid—diseased.
Motor Nerves—these carry impulses from the brain and spinal cord causing muscular contraction.
Mucous Membrane—surface lining of any cavity of the body which communicates with the exterior.
Myxœdema—thyroid deficiency beginning in childhood or later.

Narcolepsy—sudden sleep produced by excitement.
Nasopharyngeal Cavity—inside of nose, mouth and throat.
Nystagmus—rhythmic oscillations of the eyes, occurring especially on looking to the side.

Obsessional Neurosis—prevalence of impulses compelling actions which the subject knows to be irrational.
Occiput—the lower part of the back of the head.
Oculogyric Crises—uncontrollable upward movement of the eyes.

Palmar Fascia—tough membrane under the skin of the palm.
Palpebral Fissure—the slit between the eyelids.
Parity—the number of children born to a woman.
Pathogenic—causing disease.
Perseveration—involuntary repetition.
Petit Mal—transient unconsciousness with recovery after a few seconds.
Phalanx—segment of finger or toe.
Plantar Response—a reflex obtained by stimulation of the sole of the foot.
Porencephaly—the occurrence of lacunae in the tissues of the brain.
Presentation—child's posture in the maternal pelvis during birth.
Pseudohypertrophic Muscular Dystrophy—progressive weakness of certain muscles with initial swelling and subsequent wasting.
Psychosis—severe mental disorder : insanity.
Ptosis—drooping of the eyelids.
Pubis—groin.
Pyramidal Tract—fibres which convey impulses from the cortex of the brain to cells in the spinal cord.

Renal Dwarfism—retarded development associated with kidney disease.
Reflex—muscular action in response to sensory stimulus.
Retinitis Pigmentosa—degeneration of the retina associated with deposition of abnormal quantities of pigment.
Rhabdomyoma—tumour made of striped muscle cells.
Rachitic—due to rickets.
Rhagades—scarring at the corners of the mouth or round the anus.

Sagittal Sinus—blood vessel running from front to back beneath the skull.
Scaphocephaly—keel-shaped head.
Sebaceous Adenoma—overgrowth of glands in the skin.
Sex-linked Inheritance—inheritance associated with genes located on the X chromosome.
Sibs or Siblings—brothers and sisters : members of the same fraternity, irrespective of sex.
Spastic Paralysis—loss of power of voluntary movement with rigidity due to increase of muscle tone.
Status Epilepticus—continuous epileptic seizures.
Strabismus—squint.

Subarachnoid Space—a vascular region beneath the middle of the three membranes lining the central nervous system.
Syndactyly—fingers or toes joined together.

Threatened Abortion—uterine bleeding early in pregnancy.
Trauma—injury.
Tuberose Sclerosis—fibrous nodules in the brain sometimes resembling candle gutterings.

Vascular System—blood vessels and heart.
Vasovagal Attack—slowing of the heart leading to faintness.

INDEX OF NAMES AND AUTHORS

Complete references will be found at the pages referred to in heavy type

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