(7) The predisposition to microbic diseases afforded by acidosis, above all to tuberculosis. As regards children, there are questions which can best be answered perhaps by the general practitioner: for instance—What is the relationship between "biliousness" in children and subsequent tuberculosis? Are the sexes equally subject to acidosis? Does it throw any light on the greater mortality of male children? With regard to tuberculosis: Is it a question of the optimum reaction of the medium necessary for the growth of the tubercle bacillus? In other words, do certain individuals, as a result of errors of nutrition or faulty cell-metabolism, offer a more favourable pulmonary or lymphatic culture medium for the growth of the tubercle bacillus than do others?

Finally, is this whole question of acidosis, within limits, at the bottom of what we understand by heredity in respect to disease processes? Is it an effort on the part of the organism in some cases to autolyse itself? All these questions are of extraordinary interest, and the whole subject may bring us vastly nearer a proper comprehension of certain processes which up to the present have been shrouded in mystery.

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An Analysis of 200 Cases of Mental Defect.[1] By J. E. MIDDLEMISS, M.R.C.S.Eng., L.R.C.P.Lond., Medical Officer to the Leeds Committee for the Care of Mental Defectives; Late Assistant Medical Officer, Gartloch Mental Hospital, N.B.

THE cases dealt with in the present paper came under review during the course of my duties as Medical Officer to the Leeds Committee under the Mental Deficiency Act. They comprise examples of the four varieties of mental defectives defined by that Act, *viz.*, idiots, imbeciles, feeble-minded, and moral imbeciles, and include most of the clinical types described by writers on the subject. The commonly accepted

[1] A paper read at the Annual General Meeting, July 23rd, 1919.

division into primary and secondary groups has been adopted; a third group, containing cases which appear to combine the characteristics of both types, and a fourth group containing "doubtful" cases, in which the data are insufficient to allocate them definitely, being also added. It has seemed useful, too, to give columns showing the number of cases corresponding to the accepted clinical types, those complicated by epilepsy, those exhibiting pronounced stigmata of degeneration, as well as those showing a definite family history of neuropathic affections, tubercle, or alcohol respectively. It is obvious that many of these cases will figure under the several heads. The following analysis then shows the proportion of cases under each of these heads of the total number of cases examined, *viz.*, 200:

CLASSIFICATIONS.

Primary amentia.	Secondary amentia.	Combined primary and secondary.	Doubtful.	Cases associated with epilepsy.	Cases showing stigmata.	Cases showing neuropathic history.	Cases showing alcoholic history.	Cases showing tuberculous history.
146 73 *per cent.*	27 13·5 *per cent.*	10 5 *per cent.*	17 8·5 *per cent.*	58 29 *per cent.*	126 63 *per cent.*	97 48·5 *per cent.*	27 13·5 *per cent.*	54 27 *per cent.*

Feeble-minded.	Moral imbeciles.	Imbeciles.	Idiots.	Hydro-cephalic.	Cretins.	Mongolians.	Micro-cephalic.	Sclerotic amentia.
75 37·5 *per cent.*	5 2·5 *per cent.*	103 51·5 *per cent.*	17 8·5 *per cent.*	4 2 *per cent.*	2 1 *per cent.*	8 4 *per cent.*	3 1·5 *per cent.*	3 1·5 *per cent.*

Although the accepted division of cases into primary and secondary amentia is adopted, some reservations have to be made which will, at the same time, explain the inclusion of a third, the "combined" group. It may be said that the classification into two broad divisions was originally adopted in obedience to current procedure. But as the analysis progressed a conviction gradually grew in the writer's mind that such a classification was arbitrary and unsatisfactory, for the following reasons.

The criteria of primary amentia are the following:

(1) That the mental defect should have dated from birth, in other words, that there should be no period, however short, in which the mentality was normal.(2)

(2) The frequent association of physical stigmata referred to the various systems, but which may be broadly described as developmental anomalies.

(3) Evidence of a neuropathic tendency as shown in the family history.

(4) The absence of history of trauma, including in this term not only accidents (pre-natal, post-natal, and at birth), but such bodily disorders, *e.g.*, meningitis, hemiplegia, epilepsy, etc., as are usually associated with mental defect as a cause.

Cases of secondary amentia, on the other hand, frequently show a period of normal mental development, with the history of a fall, febrile or other illness, from which the symptoms of mental defect date; or, if dating from birth, disclose a history of accident or injury at birth, or of the existence of disease or debilitating influences acting on the child through the mother before birth. In either case the physical anomalies frequently associated with primary defect are wanting; but, on the other hand, there is usually evidence of gross brain-lesion, as exmplified in loss or impairment of function of one or more limbs, associated with alterations in the reflexes or sensory disturbance in the affected parts, tremors, tics, or choreiform movements.

So much for the broad definition of the two types. When one comes to an actual analysis of cases, however, the distinction between the two groups is found to be not nearly so precise and well defined as the above description would suggest. Cases occur, for example, with a definite history of meningitis or encephalitis subsequent to birth, but which are associated with stigmata, such as narrow palate, irregular dentition, malformed ears or cranium, suggesting a primary defect. When, in addition, there is a neuropathic family history and delayed development before the onset of the "illness," the primary factor becomes relatively more prominent. Such cases would accurately be described as combined primary and secondary amentia, and they are by no means uncommon. One is struck, too, by the frequency with which a history of meningitis or convulsive affection occurs (and no doubt plays a causative part in the mental defect) as a complication in cases where there is undoubtedly primary defect. Such cases are generally described as cases of primary amentia complicated by meningitis, epilepsy, hemiplegia, etc. Even in those cases where development appears to have proceeded on normal lines, up to the period of the "illness" the alleged cause of the defect, and where there are few or no stigmata indicating primary deficiency, not infrequently a history is elicited showing undoubted neuronic defect affecting several members of the family and rendering the classification, to say the least of it, doubtful.

CASE 1.—The case of H. H— is one in point. Walking and talking developed at usual age. There was a definite history of some cerebral affection, "inflammation of the brain," at 6 years of age, lasting some weeks, during which he lost his sight and speech. Mental defect noticed subsequently. Ears are large, forehead small and narrow, the cranium generally small in relation to face.

Family history.—(1) Father, said to be mentally slow. (2) One brother, a mental defective in an institution. (3) A second brother, æt. 22, low in intelligence, cannot read or write, poor at figures; vagrant habits. (4) Two other brothers died of infantile convulsions, aged, respectively, 16 and 8 months. (5) A fifth brother, æt. 7, has epileptic fits of major type, infrequent but severe. (6) A paternal uncle died of pulmonary tuberculosis. (7) A maternal uncle (died *circa* 14), a mental defective. Such a case as this is a good example of the difficulty I refer to. The primary neuronic defect as evidenced by the occurrence of amentia and

epilepsy in other members of the family does not need emphasising, and yet superficially it would be described as secondary amentia.

CASE 2.—Another, K.B— (imbecile), æt. 6, said to be normal up to 3 years, when she developed epileptic fits which continued ever since. One aunt died insane. Another aunt was epileptic and paralysed. She herself shows evidence of left hemiplegia and drags one foot. Superficially this is a secondary ament but might with equal propriety be regarded as primary.

CASE 3.—Another case, I. B— (imbecile), developed normally up to 6 years of age, when she had a fall. Later minor epilepsy supervened, which gradually merged into major attacks. She exhibited symmetrical malformation of the hands, *viz.*, the little finger and the thumb of each hand were remarkably short. The index was nearly as long as the middle finger, and the ring finger, instead of being next in length to the middle finger as is normally the case, was much shorter than the index. Further, she had a symmetrical cubitus valgus, and signs of right hemiplegia.

History: Father drank. Maternal uncle, æt. 38, is an epileptic and cannot work. Paternal grandfather had epilepsy up to 40 years of age, and died of dementia æt. 72.

Such a case, one would imagine, would come under Tredgold's definition of "Delayed primary amentia."

CASE 4.—Case, L. I— (imbecile), has never talked. At 3 years of age had a right-sided hemiplegia. Went to bed all right, woke up with loss of use of right arm and leg. Recovered use of leg to some extent but was unable to walk for six months. Has had one or two similar attacks since. Has had "fits" of some kind all her life, chiefly clonic spasms and very frequent, also typical epileptic seizures of major type. Present type post-hemiplegic chorea affecting right side and head. Walks with limp, right arm is flexed and contracted. Bilateral talipes equinus, scoliosis of spine, genu valgum of right side.

Family history.—(1) Father, æt. 41, lately in asylum seven months. (?) Manic-depressive insanity. (2) Brother died, æt. 2, of convulsions. Mental defective. (3) Father's brother has been ill for four years—some form of brain disease, nature unknown. (4) Mother's uncle died, æt. 34. Feeble-minded. Good example of combined type.

CASE 5.—M.A.H—, walked at 3 years, began to talk at 3. Had "meningitis" twice, once at 1½ years, again at 6. Said to have lost use of legs at the second attack. At the age of 8, epilepsy developed. Fits have continued up to present age (9), (serial type, occurring about once a month).

Description.—Notably anæmic. Head hydrocephaloid and globular in shape. Left internal strabismus. Thorax much deformed. Scoliosis of spine. Double genu valgum. Aortic incompetence, patellar reflex is present slightly. Apart from above, no stigmata.

Family History.—(1) Mother was epileptic; fits set in about menopause. Severe major type. (2) Maternal grandfather, died æt. 70. Epileptic all his life, no mental impairment. (3) Sister, æt. 30, has strabismus and is mentally retarded. Stays at home. (4) Sister, æt. 16, mentally retarded, stays at home.

The two sisters referred to have never gone out to work, and though not examined in detail were obviously subnormal in intelligence.

This also is a good example of the mixed or combined type.

CASE 6.—A. H—, talked at ordinary age. Not thought to be backward. At 3 years of age had illness described as "congestion of the brain" during which he had convulsions, which have continued ever since, but are now infrequent. Mental defect said to date from this illness. Never recovered speech and did not walk for a year after. Present time: Marked paresis of upper limbs and double genu valgum. Finger-joints are lax and hyper-extensible. Teeth gapped. Fits have decreased in severity and frequency—average about one per annum.

Family History.—Child illegitimate. (1) Mother, epileptic and very nervous. (2) Maternal grandfather epileptic. Fits as a boy, decreasing in severity as he grew older. Died æt. 65. Cerebral softening. (3) Mother's children by another father: (1) Son, æt. 28, in asylum, ? mental defective or insane. (2) Son, æt. 20, well. Has had "brain fever" twice. (3) Son delicate, stammers, æt. 18. (4) Son, died æt. 10 months, convulsions. "Was born blind."

Here, though the history is imperfect, a decidedly neuropathic strain is evident.

The primary character of the defect is evidenced further by the presence of stigmata. Good example of combined type.

Cases might be multiplied indefinitely, but the above are fairly typical and are sufficient to show the artificiality of the distinction between primary and secondary type as usually defined. The fact is, one encounters every degree and variety of combination of the primary and secondary factors, ranging from a comparatively pure primary type in which the inherent defect is the outstanding feature, to the type in which it may be almost if not quite excluded. Moreover there is reason to believe that the tendency to develop toxic inflammatory or vascular affections of the cerebrum or meninges is considerably greater in members of neuropathic families than in normal individuals. At any rate one is impressed by the frequent occurrence of such affections in the family history of aments, whether associated or not with normal development prior to such attack. The coincidence of similar affections in other members of the same family, the frequent association with insanity, epilepsy or amentia, as shown in the direct or collateral lines, suggest more than a chance relationship between the two.

Similarly with regard to epilepsy as a causative factor. Epilepsy itself being the expression of a primary neuronic defect (3)—even recent metabolic theories do not traverse this point of view,—it seems rather absurd to distinguish a type of amentia as epileptic amentia, meaning by that secondary to epilepsy. Again it is only a question of degree, and such cases may be regarded as primary aments "at the second remove." For example, where there is a definite family history of neuropathic affection, including perhaps epilepsy, and where epilepsy develops at some period during childhood, it is difficult to exclude a primary defect of the nervous system, even though mental defect as such may not have been manifest before the onset of fits. On the whole, a broad division of cases into two main types—*viz.* (1) cases which have a decided neuropathic strain as shown by the family history, and (2) cases which do not—would prove more useful than the present one of primary and secondary.

It must be remembered in this connection that the presence of stigmata is only useful in so far as it implies injury to the germ-plasm or to the fœtus, but this, as a recent writer (3) suggests, may be due, not to primary defect, but to constitutional disease, and especially to syphilis. If this contention is correct, many of the cases hitherto regarded as primary in origin will eventually come under the category of secondary amentia. Further, when one considers the cases of Mongolism and certain other types associated with a condition of infantilism and hitherto included in the primary group, which are probably dependent on some disturbance of function of the endocrine glands, it is evident that

many other cases which satisfy the present definition of primary amentia will eventually have to be relegated to the secondary group.

Relationship of Amentia to Epilepsy.

Of the 200 cases dealt with, no less than 58 (or 29 *per cent.*) have suffered from epilepsy at some period of their lives, which differs very little from Sherlock's (4) figures. Out of a total of 1,600 mental defectives examined, this writer found 466 (or approximately 29 *per cent.*) epileptics.

As might be expected, the fits, though broadly designated epileptic, included all known types, *viz.* major, minor, Jacksonian or focal, nocturnal, diurnal, or combined, and occurred singly or serially, or occasionally as a *status epilepticus*. In a certain proportion of cases the fits started at the second or third year of life, and diminished in frequency and severity as the child grew older, in many cases ceasing altogether. In a small proportion of cases the fits increased in severity with the age. In a considerable number epilepsy started with an acute febrile illness, in which the child became more or less comatose, and which left it with a monoplegia, paraplegia, or hemiplegia, and gross mental deterioration. In most of the cases the mental defect was dated from and was attributed to the febrile illness, and clinically they would come under the class of secondary amentia. Some of these gave a subsequent history of Jacksonian or focal epilepsy affecting the paralysed or paretic limbs, with gradual transition into ordinary major attacks. It should be mentioned that a considerable proportion of the cases gave a history of infantile convulsions gradually merging into true epileptic attacks.

Considering the large proportion of the total number examined in which fits occurred some time in their career, the number in which epilepsy could be definitely assigned a causative *rôle* was negligibly small. In by far the larger number, epilepsy was merely an accompaniment, or occurred in conjunction with amentia as a sequel of gross brain disease.

Tredgold (5), it may be remarked, in his series of cases, found that not more than 3·5 *per cent.* of aments owed their defect to epilepsy, this being an approximate estimate.

Apart from the fact that at the time of examination (usually from the ninth to tenth year in my series) the fits had often ceased, or were at least more infrequent; the characteristic appearance and features of idiopathic epilepsy were notably absent, *viz.* the mental hebetude, slow mental reaction, so-called epileptic facies, and dull, torpid expression. In short, the clinical picture presented, however diverse in detail, conformed rather to the ament type than to the chronic epileptic. It was notable that in a few isolated cases in which one would infallibly

have diagnosed epilepsy without any knowledge of the previous history, the fits had supervened comparatively late (at or about puberty). Moreover the history shows a fair development of intelligence up to the onset of the fits, followed by progressive deterioration and loss of such mental functions as had been acquired.

Although, as has been stated above, the causal relationship between epilepsy and amentia is comparatively negligible, the close association between the two conditions is of such universal recognition that it calls for further discussion.

The following analysis shows the distribution of epilepsy in relation to the three main classes of aments. There were no cases amongst moral imbeciles.

For purposes of comparison, the corresponding percentage distribution is shown for the whole number of cases examined.

	Epileptic.		Aments.	
Feeble-minded	13	22·4 *per cent.*	75	37·5 *per cent.*
Imbeciles	35	60·3 „	103	51·5 „
Idiots	10	17·3 „	17	8·5 „
Moral imbeciles	—	— „	5	2·5 „
Total	58	100·0 „	200	100·0 „

It will be noted that the incidence of epilepsy amongst the three types of aments is relatively higher amongst the imbeciles and idiots and lower in the feeble-minded than one would expect if all the types were equally affected. Moreover the disparity is greatest in the case of the idiot class, the percentage of cases having epilepsy being more than double as many as there would be on the basis of an equal distribution. This is shown more clearly in the following analysis, which shows the proportion in which epilepsy occurs in each group:

Total number of idiots examined.	Idiots with epilepsy.	Percentage.
17	10	59·0
Total number of imbeciles examined.	**Imbeciles with epilepsy.**	**Percentage.**
103	35	34·0
Total number of feeble-minded examined.	**Feeble-minded with epilepsy.**	**Percentage.**
75	13	17·3

These figures may be compared with Tredgold's (6), who found that convulsions occurred in 11 *per cent.* of the feeble-minded, 42 *per cent.* of imbeciles, and 56 *per cent.* of idiots (all institution cases). The greater incidence of epilepsy amongst the most degenerate types of

aments is, of course, only what one would expect, and is here shown to increase progressively the lower one descends in the scale of development. This also accords with Shaw Bolton's conclusions (7), who found in 94 cases of low-grade amentia 37·2 *per cent.* of epilepsy as compared with 189 cases of high-grade amentia with 12·7 *per cent.* of epilepsy. Again, he found a larger proportion of cases of epilepsy in aments with marked stigmata of degeneration than in those without. It must be borne in mind that the term "low-grade" amentia, as used by this writer, includes all the three types—feeble-minded, imbeciles, and idiots—and is not limited to the lower grades. Similarly the "high-grade" aments, as defined by him, comprise cases of mental disease which do not come within the provisions of the Mental Deficiency Act, and are not commonly regarded as mental defectives, but (8) "which form the connecting link between the mildest type of imbecile (the mental defective of the non-alienist) on the one hand, and the ordinary 'sane' individual of average intelligence and mental stability on the other," and which therefore differ only in degree from the cases here discussed.

As regards the period of onset of fits, in more than two-thirds of the cases (40 out of 58) the fits started before the age of four years, and in less than half (32 out of 58) either ceased altogether after a variable period, or diminished in frequency and severity. As before stated, the fits are of the most varying type, both in character and in severity, ranging from a minor convulsion affecting part of the body, to a typical major fit. Quite frequently there is a history of ordinary infantile convulsions gradually merging into true epilepsy, or there may be minor attacks followed by or alternating with major attacks. At different periods one type or other may predominate; there may be remissions for a long interval, or a combination of typical attacks with localised clonic spasms between the attacks. Some cases again are associated with chorea, athetoid movements, or motor tics.

Perhaps the most striking feature of the convulsive seizures of the ament is their association with an attack of encephalitis or meningitis occurring during the first few years of life. Sometimes there is a history of "fits," more or less severe, starting in infancy and culminating in a definite illness which marks an epoch in the child's career and in the mind of the relatives, and therefore is generally remembered. (I say this advisedly, because it is always a difficult matter to obtain a correct medical history from relatives, who frequently are uneducated and apt to romance.)

The history is, as a rule, that the child was suddenly taken ill, had a series of "fits," lay unconscious for several days, and subsequently was found to have deteriorated mentally, not infrequently being paralysed, and having lost whatever mental acquirements it may have attained.

Very often, indeed, the mental defect is referred to the illness. Where there have been no "fits" previous to the illness, they generally supervene, becoming less frequent and less severe as the child grows older, and frequently, as stated above, ceasing altogether later in life. Occasionally one gets a history of two or more attacks of this type, each so definite as to have memorised the date in the mind of the relatives. Apart from the marked mental arrest which ensues from such an attack, there is frequently a loss of the power of speech and partial or complete paralysis of one or more limbs. It is generally recognised, of course, that encephalitis or meningo-encephalitis occurring in infancy is one of the causes of secondary amentia. Tredgold, in his authoritative work (9), describes the underlying pathological process "as either a lepto-meningitis or a polio-encephalitis (as described by Strümpell), and, as pointed out by Oppenheim, the latter closely resembles the acute inflammation which occurs in the anterior horns of the spinal cord."

On this point Col. E. Farquhar Buzzard, in a recent paper (10), may be quoted as follows: "He thought that the medical profession had never realised that encephalitis or inflammation of the brain was by no means an uncommon condition. He was convinced from his own experience that a large number of cases of epilepsy, of mental deficiency, of hemiplegia, and of diplegia were the permanent results of attacks of encephalitis occurring in early childhood, many of these disabilities dating from an illness occurring in the first few years of life, the history being that a healthy child had been taken ill suddenly with convulsions, fever, vomiting, etc., and that the diagnosis of meningitis, gastritis, or teething had usually been made; he thought that this large group of cases could be properly attributed to the virus of poliomyelitis." There is reason, therefore, to believe that such sudden illnesses during early childhood are due to a definite infection of the encephalon. The points I wish to emphasise, however, are the following: that unless a careful and searching inquiry be made into the previous history of the case the occurrence of such an affection may be overlooked; that these affections are frequently preceded by and nearly always followed by "fits" of some type, and are therefore loosely designated as epilepsy; and finally (for reasons given in an earlier part of this paper) that aments in general, and especially the lower grades, are peculiarly liable to toxic and infective affections of the encephalon. So far indeed from occurring in a previously healthy child, there is good reason to believe that convulsive disorders which affect aments, whether of the idiopathic or infective type, attack particularly individuals with a pronounced neuropathic ancestry, and are largely the physical expression of a subnormal cerebral development.

An analysis of the cases included in the present series will perhaps make the matter clear. Of the 200 aments examined, no less than 65

(that is, 32·5 *per cent.*) had convulsive disorders of one type or another at some period in their lives. Of these 55 showed on investigation marked stigmata of degeneration or evidence of a neuropathic ancestry either in the direct or collateral lines. Frequently both conditions obtained or were associated with a family history of tuberculosis.

Aments suffering from convulsive disorders.	Aments suffering from convulsive disorders and with neuropathic ancestry.	Aments suffering from convulsive disorders and showing stigmata.	Aments suffering from convulsive disorders and with tuberculous ancestry.	Aments with convulsive disorders and neuropathic and tuberculous ancestry.
Total number, 65 (32·5 *per cent.* of cases examined.)	38 (58·4 *per cent.*)	42 (64·6 *per cent.*)	20 (30·8 *per cent.*)	10 (15·4 *per cent.*)
Total number of aments examined.	Total number of aments with neuropathetic ancestry.	Total number of aments showing stigmata.	Total number of aments with tuberculous ancestry.	
200	97 (48·5 *per cent.*)	126 (63 *per cent.*)	54 (27 *per cent.*)	

The lower columns give the total number of cases examined, with the corresponding figures and percentages for each group except the last.

It will be noted that the percentage of cases with a neuropathic inheritance is considerably higher in the convulsive group than in the whole series. As it has been shown above that it is the lower type of ament (idiot or imbecile) which is more prone to convulsive disorders, which type is presumably more likely to be the offspring of neuropathic progenitors, this is only what might be expected. One would expect, however, on a similar reasoning, that the percentage incidence of stigmata would be much higher in the convulsive group than in the general series, instead of which it is very little higher, there being only 64·6 *per cent.* in the former as against 63 *per cent.* in the latter. The disparity in the figures may partly be accounted for by the fact that the criteria as to what constitutes stigmata was not quite the same in the two groups of cases. The convulsive group, for instance, included a number of cases of encephalitis with secondary paralysis and contractions. These were not included as stigmata. In the general group the number showing stigmata was swelled by the inclusion of some such cases. The differentiation between physical anomalies due to injury and maldevelopment of the germ-cell and those due to gross cerebral disease occurring after birth is admittedly difficult, and the fact referred to would in any case only partially explain the disparity in the figures mentioned above.

One must guard against attributing to the figures too great a signifi-

cance, but the suggestion offers itself that the form of inheritance which results in the proclivity to convulsive affections, though neuropathic in character, may differ in degree and in type from that which results in those gross anomalies of anatomical structure and of function which are regarded as stigmata of degeneration. In other words the type of ament with gross stigmata may represent a lower grade in the scale of neuronic defect than the one which exhibits convulsive disorders. The matter will be referred to later when the question of neuropathic ancestry in general is discussed.

Of the total number of 65 cases, 24 presented evidence both of stigmata and of neuropathic history, and 20 (32·8 *per cent.*) gave a history of familial tuberculosis. This latter may be compared with the figures for the whole series examined, *viz.*, 24 *per cent.*, and again with the percentage of normal children as given by Potts (11) and quoted by Tredgold, *viz.*, 17 *per cent.* Of the 20 with a history of tuberculosis, 7 gave no other history, 9 gave a neuropathic history in addition, 2 showed also a neuropathic and an alcoholic strain, and finally 2 disclosed alcoholism as the only additional factor. Altogether 7 cases gave a history of alcoholism, either alone or complicated by tuberculosis or neurosis.

Neuropathic Inheritance.

A slight acquaintance with the family history of aments establishes the prominence and importance of a neuropathic ancestry, and in the series here dealt with one has a definite history of some form of neurosis or psychoneurosis in either the direct or collateral lines in no less than 97 cases (48·5 *per cent.*). There is every reason to believe that this represents a decided under-estimate of the conditions actually obtaining. The difficulty of obtaining an approximately accurate family history even in cases of physical disease is recognised by all inquirers. In the type of case before us this difficulty is enhanced for various reasons. Firstly, the informant is himself frequently an illiterate person and often of subnormal intelligence; secondly, there is a natural tendency to conceal or gloss over incidents or illnesses which are associated with a certain social stigma, or at the best an implication of inferiority; and lastly the history, to be of any value, must go back at least two generations, and for that very reason is frequently fragmentary, vague and inconclusive. The figures here given represent then the minimum number of cases with admitted neuropathic inheritance, either direct or collateral, and must be read in the light of this limitation. Neuropathic ancestry here includes not only amentia, epilepsy and insanity, but also such minor neuroses as chorea, neurasthenia, motor tics, etc., and also cases exhibiting pronounced criminal or immoral propensities, where there is no direct evidence of neuropathic affections as such. Alcoholism as

an antecedent factor is dealt with separately, although it is recognised that it is in many cases merely an indication of the neuropathic constitution, and should in such cases be included also under this head.

The figures here given may be compared with the corresponding figures of various writers. These, as quoted by Tredgold (12), range from 24 *per cent.* (Beach and Shuttleworth) to 65 *per cent.* (Goddard). Tredgold himself found a neuropathic inheritance in over 80 *per cent.* of cases (13). "In 64·5 *per cent.* the ancestral conditions took the form of amentia, insanity, or epilepsy, whilst in 18 *per cent.* they consisted in a marked family tendency to paralysis, cerebral hæmorrhage or various neuroses and psychoses." Other authorities quoted by Tredgold (14) include Lapage (48·4 *per cent.* of feeble-minded children in Manchester special schools with neuropathic inheritance), Dr. W. A. Potts, 45·6 *per cent.* of children in Birmingham special schools, and a Commission appointed by the Legislature of Connecticut who found neuropathic heredity to be the undoubted cause in 65 *per cent.* of cases.

It may be noted that Goddard (15), in his work "Feeble-mindedness: Its Causes and Consequencees," found (1) feeble-minded ancestry in 54 *per cent.* of 300 cases, (2) 11·3 *per cent.* which he groups as "Probably Hereditary," also with feeble-minded ancestry, and (3) 12 *per cent.* with neuropathic ancestry, whose family history shows relatives suffering from various brain affections, such as paralysis, apoplexy, "brain disease" and the like, epilepsy, insanity (so described), blindness, deafness, and other neurotic conditions. If all these groups be included under neuropathic ancestry, the total amounts to 77·3 *per cent.*, which approximates to Tredgold's estimate of 80 *per cent.*

The figures quoted are not strictly comparable. Those for the children in special schools, for example, include a disproportionate number of low-grade aments, as in my experience a considerable proportion prove incapable of being educated even in a special school. Again, Goddard, in his series, gives particulars of on an average 200 or more individuals in one family. Tredgold conducted a similar exhaustive research in the case of 200 individuals. It is probable that the searching inquiries of these two writers would account for their higher figures.

Apart from the incidence of a neuropathic history, it must be remembered in this connection that a calculation of the actual number of cases with neurotic strain gives only a relative estimate of the importance of this factor. It takes no account, for instance, of the number of individuals with mental affection in a given family, or the degree to which they are affected. This, of course, can only be shown by genealogical tables. A family with several individuals suffering from psychoses or epilepsy has obviously a much stronger neurotic strain than a family with one such member, but this fact would not emerge

where the ordinary methods were employed. A proper quantitative estimate of the neuropathic inheritance would necessarily take account of these facts.

Apart from the actual incidence of neuropathic ancestry in the whole group of cases, it is interesting to study its relative incidence in the three grades, *viz.*, feeble-minded, imbeciles, and idiots.

Of the 97 cases showing a neuropathic strain 39 were feeble-minded, 50 imbeciles, 7 idiots, and one moral imbecile. If the neuropathic inheritance were evenly distributed through the different grades the figures would be: feeble-minded, 36·4; imbeciles, 49·9; idiots, 8·3; moral imbeciles, 2·4, which is approximately what one finds to be the case. If anything the discrepancy in the two series suggests that the neuropathic factor assumes a greater significance the higher one ascends in the type of amentia, instead of a lesser as one might expect. It would be interesting to know whether these findings would be confirmed if a larger number of cases were analysed.

The moral imbecile class may for this purpose be excluded, as they are hardly comparable, differing as they do from the other types qualitatively rather than quantitatively.

Alcoholic Inheritance.

Of the total 200 cases examined only 30 (15 *per cent.*) gave a definite history of familial alcoholism. What has been said with regard to the obtaining of authentic records applies here with even greater force. Apart from the reluctance to admit an addiction to alcohol, there is the personal factor, which varies with each observer, to take into consideration. Data are frequently vague and difficult to standardise, and are further vitiated by the varying interpretations given to the same data by different observers. Here, again, the number quoted represents the absolute minimum and may be regarded as a considerable under-estimate.

The numerical incidence of alcoholism in the different groups is as follows: feeble-minded, 14; imbeciles, 12; idiots, 3; moral imbeciles, 1. If alcoholism were evenly distributed through the groups the numbers would be: feeble-minded, 11·2; imbeciles, 15·4; idiots, 2·5; moral imbeciles, ·75. One finds, therefore, that alcohol as a factor plays a relatively greater *rôle* in the case of the feeble-minded and a lesser one in the case of the imbeciles, whereas in the case of the idiot and moral imbecile the distribution is approximately proportionate.

Stigmata of Degeneration.

The term "stigmata," as here used, includes all those anomalies of structure and of function which are so frequently found in association

with amentia, as to suggest that they are part of the germinal blight of which the mental defect is itself only a special manifestation. They mostly date from birth or from the early developmental period, and do not include those structural deformities and imperfections which occur in secondary amentia as the sequelæ of gross organic disease of the cerebrum. The distinction may appear somewhat artificial and in practice is a little difficult, but in many cases the clinical picture presented and the definite history of onset enable one to decide as to the nature of the defect.

Numerical incidence.—Of the total number of aments under review (200), no less than 126 (63 *per cent.*) present stigmata. In the great majority of the cases these are multiple in character, and very few cases are included which do not exhibit more than one stigma. Their numerical distribution among the four types of aments is as follows: Feeble-minded, 37; imbeciles, 71; idiots, 15; moral imbeciles, 3. If the stigmata of degeneration were distributed proportionately through the different types according to the prevalence of each type, the incidence would be as follows: Feeble-minded, 47·25; imbeciles, 64·9; idiots, 10·7; moral imbeciles, 3·1.

It will be seen, therefore, that the number of moral imbeciles presenting stigmata is approximately what it would be with a proportional distribution; in the idiot class it is much greater (nearly 40 *per cent.*), in the imbecile class it is a good deal greater (9·4 *per cent.*), whilst in the feeble-minded class it is much less (21·7 *per cent.*). In other words, excluding the moral imbecile class, the incidence of stigmata shows a steady rise as one passes from the higher grade to the lower grade ament. This accords with the findings of most writers on the subject, who agree in regarding the presence of stigmata as a measure of the degree of neuronic degeneration.

Character of Stigmata.

The anatomical anomalies encountered may be classified as follows:

(1) *Variations in the shape of the external ear.*—(*a*) Asymmetry of the ears: This is perhaps the commonest anomaly met with, and is almost invariably associated with bilateral deformity. That is to say, one rarely finds that either ear approximates to the normal. Where both are deformed, the deformity is greater on one side than the other, or is of a different type. In my experience asymmetry of the external ear is more frequent than gross deformity and has at least as great a significance.

(*b*) Abnormally shaped ears: These include ears which are too large or too small relatively to the size of the head, ears which project laterally, and ears whose abnormality consists in variations in the shape and development of the various folds and hollows which constitute the normal contour. Perhaps the commonest deviation is in the helix, which may be hardly developed at all, resulting in a thin-edged ear, especially at the tip. Again, the incurved helix may be compressed or flattened on itself, and when this is the case the compression or flattening is rarely equal on the two sides. This represents the commonest type of asymmetrical ear. Another type commonly met with is the long, narrow ear, where the disparity between the vertical and horizontal diameters is greater than usual although other-

wise the ear may be normal. An excessive development of the concha at the expense of the anti-helix, so that the fossa triangularis, fossa scaphoidea and the concha become one common cavity is frequently seen. This type of ear in the writer's experience usually projects laterally. The lobules may be almost absent, abnormally large, or adherent to any degree. In one case the scaphoid fossa was continued as a groove into the lobule, which was not so full and pendulous as usual The case was a Mongolian imbecile who presented the typical stigmata, the ear abnormality being bilateral.

(2) *Variations in the hard palate.*—These include varying degrees of highly-arched and narrow palates. The great majority of highly-arched palates are also narrow, the narrowing being much more pronounced in front than behind. In a majority of cases the narrow palate becomes progressively more narrow as one approaches the incisor teeth.

In a number of cases there is a sudden narrowing about the level of the first molar.

In both types there is overcrowding of the canine and incisor teeth. Occasionally one sees a broad flat palate, where the arching is subnormal.

There was no case of cleft palate in the series.

(3) *Variations in the face and jaws.*—Prognathism, receding chin, loose gaping mouths are commonly seen, as also are flattening or absence of the bridge of the nose. Unusual patency or direction of the nostrils, so that the latter look forwards rather than downwards, are met in special types. The same may be said of the radial striæ or grooves seen in the lips of the Mongol imbeciles. Close setting of the eyes, the obliquity of the palpebral fissures of the Mongol and the presence of the epicanthic fold require mention.

Skin affections of the face, especially dryness or seborrhœic dermatitis, are unusually common. Adenoma sebaceum is rarely seen and is not necessarily associated with mental defect. In a typical case seen by the writer the child, an epileptic imbecile, had had the disease from three years of age. His mother, a woman of average intelligence, had also had the complaint from early childhood. A brother of the patient had epilepsy and was mentally defective.

Asymmetry of the face, so much emphasised by many writers, was rarely observed in the present series.

(4) *Variations in the shape of the cranium.*—Craniums which conform more or less to the classical types, brachycephalic or dolichocephalic, are fairly common, though as a rule the skull of the defective is much more irregular and asymmetrical than in any normal type. Apart from the extreme types, such as hydrocephalic or microcephalic, the commonest deviations from normal are due to sub-development of the frontal region—receding and narrow foreheads are the rule—whilst a sharply rising occiput with practically no backward projection is very common apart from the typical bullet head of the Mongol. A flattened vault with projecting bossy forehead is also seen, though probably due to rickets in many cases. In a large proportion of aments the cranial capacity is markedly diminished, though the condition is frequently obscured by bony hypertrophy due to rickets or syphilis.

(5) *Variations in the length and shape of the phalanges.*—Abnormal shortness and stumpiness of the fingers as well as incurving of the little finger have been described among the stigmata found in aments, but the subject has hardly received the attention it merits. It may be remarked that in the normal individual there is a fairly constant relationship in the length of the digits, the second finger being the longest, the ring finger being slightly shorter, the index coming next, and the little finger being the shortest, the thumb of course being shorter than any of the fingers. Normally the tip of the little finger extends to the last interphalangeal joint of the ring finger. In a considerable proportion of aments the little finger and thumb are relatively much shorter than in the normal hand. Frequently the tip of the little finger does not reach beyond the centre of the second phalanx of the ring finger. An abnormally short little finger and thumb frequently occur in association, and in practically all cases the abnormality is bilateral and symmetrical. Not infrequently, too, one finds an abnormality in the relative lengths of the other fingers. In a case in point there was abnormal shortness of the little fingers and thumbs (bilateral and symmetrical). The middle finger as usual was the longest, the index came next, and the ring finger third in length. In fact the ring and

index fingers were transposed as regards their relative lengths. In this case it was noteworthy that the mother's hand showed an identical abnormality of the little finger and thumb, but the relative lengths of the other three fingers were normal. The abnormality was identical on both sides in both mother and child. As has before been mentioned, the abnormal shortening of the little finger is frequently associated with pronounced incurving. Similar variations are found in the length of the toes, though the relative lengths of the digits are not so fixed or constant in the normal individual as in the case of the hand. Instead of a gradually tapering off in length from the great toe to the little toe, one sometimes finds two or three toes of approximately the same length, or the normal disparity in the size of the great toe and the rest much less evident than usual. Another not uncommon abnormality is for one toe to be out of alignment with the rest, giving the appearance of two toes of the same length. A partial syndactylism is not uncommon, a partial polydactylism is more rarely seen, and in these and in all the other abnormalities described the condition as a rule is symmetrical. Partial syndactylism, or a setting of one toe out of alignment with the others, is not uncommon in normal individuals, and in all cases observed the condition was symmetrical. Moreover it usually occurs as an isolated phenomenon and not in association with other stigmata as in aments.

Hyperextensibility of the joints of the hands and fingers is so common as only to require mention. Apart from modification in the relative lengths of the fingers a variation in the absolute length of the digits is very common. A short squat hand with thick stubby fingers, poorly developed nails, atrophy of the muscles, and flatness of the thenar and hypothenar eminences resulting in a simian hand have all been described and are of common occurrence. In short, one rarely sees a hand normal in shape and contour in the lower-grade mental defective. In the higher types, as one would expect, the departure from normal is not nearly so evident.

(6) *Variations in the teeth.*—These have been described in such detail as only to require brief mention. Overcrowding is the rule where there is a narrow V-shaped palate. Malposition and eruption at different planes of the alveolus, abnormally small teeth, rotation of the vertical axis so that the tooth faces forwards and backwards instead of outwards and inwards and serration of the edges are the chief abnormalities.

(7) *Variations in the eyes.*—Close setting of the eyes is fairly common. Refractive errors are much more common than in normal individuals, though the present series were not examined from the point of view of the particular defect of vision. Strabismus occurred in a large percentage of cases. No case of ptosis was recorded. Iridoplegia was not seen; in most of the cases the pupils reacted normally to light and accommodation. Eccentric and irregular pupils occurred but rarely and the same may be said of speckled irides, though Shaw Bolton I believe regards them as a frequent occurrence in the ament. Rotary or lateral nystagmus was noted in a number of cases.

(8) *Variations in the deep and superficial reflexes.*—It is to be remembered that the cases with a definite history of organic disease of the cerebrum or meninges with resultant secondary amentia are not included in the present consideration. This rules out immediately the class of case where one would expect to find anomalies in the reflexes. Even so the writer has been struck by the variations in response in both deep and superficial reflexes.

The reflexes chiefly examined were the patellar, the plantar, the epigastric, hypogastric, the cremasteric, and the pupillary reflex. It is not here proposed to give a detailed statistical analysis of the results obtained; this it is hoped will form the subject of a future paper.

In the first place it may be said that most of the anomalies relate to the condition of the superficial reflexes, which, in view of the frequent disorders of ordinary sensation met with in aments, is not remarkable.

As the integrity of the superficial reflex arc is dependent upon the function of sensation, it affords indirectly a gauge to the impairment or modification of the sensory function, especially as the mental condition often precludes one from measuring the latter by direct methods.

The variations in the superficial reflexes include:

(*a*) In a fairly large proportion of cases the superficial reflexes are all absent, or only present to a minimal degree. Generally a diminution in the abdominal and

cremasteric reflexes is associated with a diminution of the plantar reflex, but there is no constant relationship between the two. When one considers the activity of the superficial reflexes in the normal child, the diminished or total lack of response of the ament assumes a striking significance. This is perhaps the commonest deviation from the normal.

(*b*) In a smaller proportion of cases the superficial reflexes are exaggerated, though, as in the preceding case, they are not uniformly affected.

(*c*) The tendency of the reflex to "overflow" as it were is frequently exemplified in the case of the cremasteric reflex. In extreme instances of exaggeration, when the inner side of the thigh is stroked, not only is there a retraction of the testicle of the same side, but it is accompanied by a sharp contraction of the abdominal muscles as far as the umbilicus or beyond it, and sometimes involving both sides. This, by the way, is not peculiar to aments, but is frequently found in normal individuals. In such cases there is sometimes an extreme degree of ticklishness, so that a slight stroking of the skin of the abdomen evokes a fit of laughter as well as the characteristic muscular response.

This suggests that alterations in the superficial reflexes are chiefly due to changes in the afferent path, and immediately to abnormalities of sensation. Finally, it may be remarked that gross anomalies in the superficial reflexes are frequently associated with a normal condition of the deep reflexes.

It is to be regretted that the scapular reflex was not systematically examined as it generally disappears in the normal child at or about puberty, and a comparative study of the condition of the reflex in the ament might be expected to yield interesting results. One's general impression is that the reflex is not nearly so active in the ament as in the normal child. Frequently it is altogether absent. The scapular reflex (16), it will be remembered, is obtained by stroking the skin in the interscapular region, when a contraction of the scapular muscles ensues. If the chest-piece of a stethoscope be placed to the back of a child during ordinary examination of the chest there will be a sudden sharp bending of the spine towards the affected side. In the writer's experience the sensory receptive area is not limited to the interscapular region but extends as low as the last dorsal vertebra. This reflex it is, however named, which is mostly frequently diminished or lost in aments.

(9) *Modifications in the motor nervous system* are so common in aments and have been described in such detail as to require little more than mention. Some imperfection in the motor apparatus may almost be said to be the rule in the lower grade ament. Ranging from a total or partial paralysis of one or more limbs in the severe types to a mere inco-ordination or handlessness (to use a graphic Scotticism) in the less pronounced, one meets innumerable and varying degrees of imperfection and want of adaptation to the normal needs of the individual. Apart from gross paralysis, the lack of fine adjustment, shown in the inability to dress or fasten buttons or execute any delicate movements, is strikingly common. The clumsiness of gait, the frequent occurrence of deformities of either extremities, the laxity and hyperextensibility of the distal joints, the frequency of coarse or fine tremors, chronic chorea, motor tics, and habit spasms, all testify to the presence of some abnormality or imperfection of the motor system. The effect of emotion or attention in developing or reinforcing a muscular tremor is very evident. Chorea, motor tics and athetoid movements are often found as a sequela of hemiplegia or paraplegia, though quite frequently there is no evidence of such antecedent event. A fine tremor affecting the whole or greater part of the body elicited on movement and absent in repose is frequently noted.

(10) *Gross deformities of the trunk, spinal axis or limbs* occur in the overwhelming majority of the lower-grade aments, generally as the result of paralysis in early or intra-uterine life. In view of the frequency of some degree of paralysis, or paresis of the limbs, this is to be expected. They include genu valgum and genu varum, pes cavus, pes equinus or equino-varus, and pes valgus. Pes planus, the commonest foot deformity of the normal individual, is comparatively rare in aments. Secondary scoliosis of the spine is frequently associated with these deformities, and kyphosis and lordosis are fairly common. Cubitus valgus or varus is occasionally seen and when present is bilateral. Rickety curvature of the limbs and chest deformities are, I think, not more common than in normal individuals, and the same applies to the cranial abnormalities due to rickets.

(11) *Variations in the external genitalia.*—Under or over-development of the

external genitals are found in a fair proportion of aments. In the males non-descent of the testicle on one or both sides is perhaps the commonest anomaly. In a certain number of cases, on the other hand, the external genitals and especially the penis are ridiculously large in relation to the size of the body. In some male cases the bodily contour conforms more to the female than to the male type, the stature being short and stumpy, the genitals undeveloped or rudimentary, and there being an excessive deposit of subcutaneous fat throughout the body. Another feature of these cases is the diminution of the normal curves of the spinal column so that the back is short and straight. The condition in fact resembles that found in the so-called dystrophia adiposo-genitalis, although no case was seen which presented the extreme signs of infantilism exhibited by typical examples of this disorder. None the less, it is not improbable that these cases are merely minor examples of what Schafer describes as hypopituitarism (17), and are due to disease or atropy of the pituitary glands. As the diminution in stature is related to atrophy of the anterior lobe of the pituitary gland, and excessive fat formation and deficient sexual development to deficiency of the posterior lobe, it is to be supposed that in this case the whole gland is concerned. This is merely a suggestion thrown out, of course, and would, if correct, account only for the bodily conformation as described.

Similarly, some female aments are characterised by an unusual bony and muscular development, a bodily conformation resembling the male type with a notable absence of subcutaneous fat, with the roundness of contour characteristic of the female. The absence of mammae, presence of hair on the limbs or trunk, and occasional absence of hair from the pubic area combine to heighten the resemblance to the male. Such individuals are generally tall, and have a pelvis of the male type, and unusual length of limb.

In one such case, that of a female, æt. 33, the facies resembled that seen in acromegaly. The nose and upper jaw were relatively large; the thorax was of the male type with absence of mammae. There was scantiness of the pubic hair, the limbs were long and muscular, and there was a marked kyphosis in the dorsal region and a notable diminution of the subcutaneous fat. She was stated never to have menstruated. Her mental state was that of a feeble-minded person. This case may be contrasted with the condition of infantilism discussed above, and the condition may be attributable to hypertrophy of the anterior lobe of the pituitary gland so far as the somatic variations are concerned.

Hypospadias and epispadias are occasionally though comparatively rarely seen, and no case of hermaphroditism was met with in the series.

The above represent the main types of variations in the anatomical structure found in the series of aments under review, and their significance lies in the fact that though any of them may occur singly in the normal individual they occur far more frequently in aments, and in the vast majority of cases are multiple in character.

A Comparison of the Numerical Incidence of Stigmata and Neuropathic Inheritance Respectively.

As already stated, it has been found that the incidence of stigmata shows a steady and notable rise as one descends in the scale from the higher-grade to the lower-grade ament, whereas a neuropathic inheritance is, if anything, slightly more common in the higher grades of amentia than in the lower. This perhaps hardly accords with one's expectations, and certainly not with the views currently held. Most writers, indeed, on this and kindred subjects regard stigmata as indicative alike of the degree of neuronic degeneration and a measure of neuropathic inheritance, the implication being that one increases *pari passu* with the other.

Whether true or not, this view is certainly not confirmed by the findings of our present analysis, although it is recognised of course that the analysis of a larger number of cases might yield very different results.

Aldren Turner, for example (18), speaking of stigmata, says—"They are of immense value as an index of the intensity or degree of the hereditary predisposition." Moreover, he finds (19) "that of 200 cases of epilepsy 42 *per cent.* presented well-marked evidences of structural stigmata, although no hereditary neuropathic history could be obtained; while of those in whom such a history was known, only 24 *per cent.* showed stigmata." He goes on to say (20): "It is therefore obvious that the absence of a family neuropathic history is of little account in face of the well-marked structural signs of an inherited degenerative disposition which many of the cases presented. Moreover it is clear that if the family history could have been probed more deeply, a large percentage of those with stigmata of degeneration would have made mention of some inherited degenerative psychosis." Upon which one may comment that the postulated relationship between stigmata and hereditary predisposition is by no means established—at least so far as this writer is concerned.

Special Clinical Types.

(*Mongolian and Cretin Imbeciles.*)

Two groups of mental defectives, *viz.*, the Mongolian and the cretin types, stand out pre-eminently from the general body by virtue of their strongly marked physical characteristics and the pronounced resemblance to one another of the individual members of the class in question. Although the ætiology of the cretin may be regarded as settled, whilst that of the Mongolian imbecile is still *sub judice*, the general resemblance between the two groups is such as to suggest a similarity in origin, *viz.*, a disturbance of function of the endocrine glands.

Out of the 200 cases examined only 8 were Mongolian imbeciles, whilst 2 were cretins, all 10 cases being typical examples of their class.

Mongolian Imbeciles (8 in number).

The whole eight were males.

Case 1.—C. P—. The child was fifth in a family of five, there being ten years' interval between birth of patient and the last preceding child. No neuropathic history. Age of mother at birth of patient 40.

Case 2.—G. F—. Patient was the only child and was born two years after marriage. Father was "nervous" and unstable, and maternal uncle was insane. Mother's age at birth of patient 32.

Case 3.—I. B—. Patient was last born in a family of nine. No neurosis in family. Mother's age at birth of patient 40.

Case 4.—E. A. G—. Patient was last born in a family of five. Maternal aunt became insane at climacteric. Age of mother at birth of child 40.

Case 5.—J. W—. Patient was third child in family of four. Maternal grandfather drowned himself (melancholia). Age of mother at birth of patient 37. Patient was an epileptic and had hypospadias.

Case 6.—N. A. W—. Patient was third in family of three. No neurosis in family. Age of mother at birth of patient 32.

Case 7.—F. B—. Mental status that of idiot. Patient is eighth child in family of eight. Father drank heavily for twenty-six years. Age of mother at birth of child 46.

CASE 8.—J. G—. Patient is second child in family of four. Father and paternal grandfather addicted to drink. Age of mother at birth of patient 30.

To summarise, all eight cases were males. In five cases the child was the last born in the family. The youngest mother was aged 30 and the oldest 46, the average age of the mother at birth of child being $37\frac{1}{7}$ years.

In three cases there was a history of insanity in either the direct or collateral lines, in two a history of alcoholism, whilst in three there was no evidence of neurosis.

Although the number of cases was small the evidence would suggest that the comparatively advanced age of the mother is a probable factor, with its possible corollary—an exhaustion of the reproductive function; and secondly that neuropathic inheritance probably plays an equally important *rôle*. The disharmony or disturbance of function of the endocrine glands which is now regarded as the essential and proximate cause of Mongolism may be related to the age of the mother, but the mode of operation of the neuropathic factor is less direct.

Cretin Imbeciles (2 in number).

Both cases were typical examples of cretinism.

CASE 1.—H. T—, male. The mother was "nervous" and the maternal uncle was an inmate of the imbecile ward of a workhouse hospital. Patient was the sixth child in a family of twelve.

CASE 2.—A. M. I—, female. Patient was eldest child of six. No history of neurosis, but maternal grandmother died at 52 "at the change of life." Age of parents at birth of patient was 25. The patient herself, although 16 years of age, was only a little over 4 ft. in height.

The number of cases was too small to prove anything as to ætiology beyond the obvious fact of thyroid deficiency.

TUBERCULOUS INHERITANCE.

Of the 200 aments examined, 48 (that is, 24 *per cent.*) gave a history of tuberculosis either in the direct or collateral lines. Tredgold (21) found a tuberculous history in the families of 34 *per cent.* of cases investigated, and quotes Beach and Shuttleworth as finding close upon 30 *per cent.*, Langdon Down 22·5 *per cent.*, Kerlin 56 *per cent.* and Potts 43·2 *per cent.* of defectives as compared with 17 *per cent.* of normal children. My figures include tuberculosis of all types, pulmonary, glandular, intestinal, and tuberculosis of bones, and as in the case of neuropathic ancestry probably represent a considerable under-estimate of the actual facts. One is repeatedly struck by the co-existence of tuberculous affections in several members of the family with insanity, epilepsy, or mental defect in others. The two strains, the neuropathic and the tuberculous, are rarely combined in the same individual, but in extreme cases the distribution is such as to suggest an alternation or substitution of the two diatheses in different members of a family.

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(4) Sherlock.—*The Feeble-minded*, p. 261.
(5) A. F. Tredgold.—*Mental Deficiency*, p. 277.
(6) *Op. cit.*, p. 221.
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(8) *Op. cit.*, p. 162.
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(10) "Lethargic Encephalitis."—Paper read before Med. Soc. of London by Col. E. Farquhar Buzzard, *Brit. Med. Journ.*, December 24th, 1918.
(11) W. A. Potts.—Quoted by Tredgold, *Mental Deficiency*, p. 47.
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(13) *Loc. cit.*
(14) *Loc. cit.*
(15) Goddard.—*Feeble-mindedness, its Causes and Consequences*, p. 437,
(16) Hutchison and Rainy.—*Clinical Methods* (*Superficial Reflexes*). p. 477.
(17) E. A. Schäfer.—*The Endocrine Organs*, p. 110.
(18) Aldren Turner.—*Epilepsy*, p. 31.
(19) *Op. cit.*, p. 38.
(20) *Op. cit.*, p. 38.
(21) Tredgold.—*Mental Deficiency*, p. 47.

(2) The so-called delayed primary amentia, in which there is a latent period of normal mentality, is after all only a sub-division of primary amentia, in which the potentiality for normal development is rather greater than in the typical cases of this group.—(3) "The co-existence of epilepsy and mental disease is thus of such a character as to indicate that both conditions are symptomatic of cerebral degeneracy" (*The Brain in Health and Disease*, by J. Shaw Bolton, p. 199).

Anxiety States Occurring at the Involutional Period.(1) By D. K. HENDERSON, M.D.Edin., F.R.F.P.&S.Glas., Senior Assistant Physician, Royal Asylum, Gartnavel, Glasgow.

A WIDOWER, æt. 69, was admitted recently to the Glasgow Royal Mental Hospital in an anxious, apprehensive, excited, restless state. The history of the case showed that he had come of a good stock, and that he had been a strong, healthy man. For a period of forty-six years he had been employed by the same firm of lawyers, and latterly had been their cashier. He had married twice; there were four children from the first marriage and two from the second. He had divorced his second wife on account of her unfaithfulness. In January, 1919, he resigned his position, sold his home, and made plans to live with his daughter. Three days after his home and furniture had been sold he made a determined attempt on his life by cutting his throat. One month later he was admitted to the Glasgow Royal Mental Hospital. Following his admission he continued in a state of abject misery, he

(1) Paper read at the meeting of the Scottish Division, March 19th, 1920.