THE ETIOLOGY OF AMENTIA.

THESIS

by

Ernest Arthur Milner, M.B., C.M.

The Royal Albert Institution,
Lancaster.

A.D. 1913
THE ETIOLOGY OF AMENTIA.
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
*
* INTRODUCTORY. *

A survey of the present state of knowledge concerning the causes of Amentia (or Mental Deficiency) showed a very chaotic state of affairs. It is true that most writers are agreed on certain general points; a number of conditions are accepted by all as factors in the production of this condition; but the agreement ceases when it becomes a question of estimating the relative value of the different causes; the figures quoted in support of the various opinions are nearly all drawn from too small a number of cases to give reliable data, and only emphasise the different points of view of their authors. The matter is further complicated by the absence of a common system of grouping the cases, so that similar groups are not identical in composition and are therefore not strictly comparable. Even in cases where the groups are identical an extreme divergence of numerical and proportional results is often found. This is especially noticeable when the figures compiled in America are compared with those relating to European countries.
Again, although there is general agreement as to what may be considered causes, it is only very general; a number of factors are accepted by some and rejected by others, and it would seem that some even of the most generally accepted are relics of an earlier and obsolete pathology, and will not stand critical examination in the light of present day knowledge of Morbid processes, more especially in their application to the Nervous System and its functions.

All that seemed possible, therefore, was to group the cases available on the lines adopted by the recognised authorities, in the hope that some additional light might be thrown on an obscure subject; or that at any rate the Data might be available for other investigators; and to draw from these figures such tentative conclusions as appeared to be justified by the percentages obtained.

CLASSIFICATION.

With the exception of conclusions regarding certain well defined types, no attempt has been made to deal with the question of etiology from the stand point of a classification of varieties of Amentia for two reasons – In the first place no system of classification has yet been propounded that conforms to the recognised scientific standard. The most satisfactory are only groupings of more or less well marked clinical types in which the group of "other cases" or "unclassified" bulks largely, and which one and all lack a common pathological or anatomical denominator.
In the second place a comparison of the individual with his casebook record failed to show any connection between the probable cause and the resulting variety, and proved conclusively that the same cause would produce very different types of Ament, and that same type of Ament might be the result of widely different causes.

The classification of mental defectives adopted by the Royal Commission is of course entirely useless, being merely a convenient division of cases for practical and legal purposes.

An attempt to classify the cases under the heading of causes failed for the additional reason that in a considerable majority the operation of more than one etiological factor could be clearly traced.

It is easy to assign a minority of the cases to one or other of two or three well defined varieties of causes.

1. Some cases were of the truly and purely hereditary type. The defect was evident at or soon after birth; the stigmata of degeneration and other physical abnormalities were well marked, and the patients' pedigree furnished the necessary corroboration.

2. Others again, with an apparently untainted descent, with no stigmata, and with a history of some accident or disease as the starting point of the defect, following an initial period of normal development, belonged to the accidental or secondary type of Ament.

3. Some possibly belong to a third class where the Amentia is the result of adverse conditions affecting the health and reproductive energy of the mother. These have been called by some writers developmental cases.
4. The majority of cases however, as stated above, would have to be included in two or even in all three classes, evidence being fairly conclusive of the co-operation of several factors.

In view of the fact that some cases appeared to be due to only one cause while others were the result of several, it seemed impossible to divide the causes into direct and remote — or primary and secondary because in many cases what might fairly be termed the remote or predisposing cause was the only or at all events the most direct exciting cause also.

Some few cases caused further confusion on account of the impossibility of deciding the relative importance of the several factors.

The only attempt at classification that has been made is a rough grouping of the various causes under three heads viz: 1. Hereditary influences. 2. Influences acting directly on the Patient and 3. Influences affecting the health of the Parents.

It is not pretended that this grouping is either strictly scientific or accurate, it is adopted purely for convenience.

________________________

GENERAL OBSERVATIONS.

A perusal of the records of cases of Mental Deficiency reveals certain general features which it may be of use to indicate clearly at the outset.
1. While positive statements regarding the patient's relatives may be accepted as accurate, negative statements must be received with the greatest caution. For instance the statement that a certain member of the family was epileptic, insane, intemperate etc. would no doubt be true, but further investigation has shown that the statement that no other cases of mental or nervous disease were known to occur in the family or that the parents or brothers and sisters of the patient were of normal health and intelligence was in a considerable number of instances erroneous. This as pointed out by Sir W. Gowers may be due either to the ignorance of the informant or to the well recognised unwillingness of people to own to facts of which they are ashamed; and the feeling that mental disease of any kind is in some way a disgrace to the family, is still almost universal. This detracts greatly from the accuracy of percentages calculated from the results of "information received" at the same time it enhances greatly the relative value of all positive information.

2. Again the personal history of the patient is frequently defective and inaccurate and the chronology confused. Still, these defects are more or less inherent in all similar records, and in spite of them the results obtained may help to throw some light on this obscure subject.

The following figures and the conclusion drawn from them are the result of a careful analysis of the records of the cases admitted to the Royal Albert Institution, Lancaster, since its opening in 1871.
Of the 3254 cases in the case books, 894 were rejected owing to absence of history or other particulars. In 185 other cases there is nothing in the information given either concerning the family or personal history of the patient to throw any light on the case.

There remain therefore for statistical purposes 2155 cases; and the percentages have been calculated from this total except where otherwise stated.

To these have been added for purposes of comparison the statistics published by previous investigators with their conclusions, and the opinions of the recognised authorities; a list of the authorities quoted and of the works consulted will be found at the end of this thesis.

1. HEREDITARY INFLUENCES.

THE NEUROPATHIC INHERITANCE.

As has been already pointed out, abundant evidence was obtained that hereditary played a very important role in the production of Mental Deficiency, but was not in all cases the sole cause.

In a large number in which it was a factor it appeared to act - as Dr. Mott has recently shown in relation to insanity - by the transmission of a hereditary weakness or tendency rather than a definite morbid condition and was insufficient of itself to determine the occurrence of mental deficiency without the aid of some other intercurrent exciting cause such as injury to the head, meningitis, etc.
It seems probable that the occurrence of disease or injury to the brain of a child with a strong neuropathic heredity would be more likely to produce permanent impairment than in the case of one with a sound pedigree—but proof of this would be difficult to get, and is obviously not here available.

In 1003 cases (46.5 per cent) other defectives were noted in the family—distributed as follows:

Generation 1.

1. Direct (Grandparents of patient) 95 cases.
2. Indirect (Brothers & sisters of 1.1.) 95 " "
3. Not particularised or more remote 30 " "

Generation 2.

1. Direct (Parents of patient) 619 cases.
2. Indirect (Brothers & sisters of 2.1.) 202 " "
3. Remote (Descendants of 1.2.) 36 " "

Generation 3.

1. Direct (Brothers & sisters of patient) 374 cases.
2. Indirect (Descendants of 2.2.) 55 " "

Note:—Defectives includes persons described as imbecile weak or feeble-minded, insane, epileptic, neurasthenic, peculiar, not normal &c &c.

Other observers have found a hereditary taint in from 20 to 76 per cent.

L. Dahl found 50 per cent in his 109 cases.
Langdon Down says he failed to find a neuropathic heredity more or less direct in only 16 per cent, and 15 per cent of the mothers and 16 per cent of the fathers were feeble-minded.

Fletcher Beach found the hereditary taint in 59 per cent.

Shuttleworth found it in 41.3 per cent.

Koch in approximately 60 per cent.

Kerlin in 100 families found 27 per cent.

Rogers gives 25 per cent and H. Piper 18 per cent in the cases he examined from Dahldorf.

Graham states that 65 per cent of his 800 cases were congenital and that in 18 per cent a hereditary taint was admitted; but considers the true figure to be much larger.

Ireland gives no figures but is convinced of the probability of a feeble-minded woman having imbecile children.

Barr believes that the transmission of imbecility from generation to generation is the most potent cause of inherited amentia but concedes the power of producing Idiocy in the children to both epilepsy and insanity, of which he considers epilepsy the more potent.

Tredgold finds a Neuropathic Heredity in 80 per cent of his selected cases.

The following statistics of direct transmission of amentia from parent to child were found. In a few cases the defect was traced back to the grandparents and in one case it appeared in members of four successive generations.
153 imbecile mothers had 686 children: -
  these were - imbecile        274 = 1.8 each.
  - normal                     159 = 1 each.
  - not described              253.

64 imbecile fathers had 333 children: -
  these were - imbecile        114 = 1.78 each.
  - normal                     141 = 2.2 each.
  - not described              178.

30 imbecile couples had 167 children: -
  these were - imbecile        58 = almost 2 each.
  - normal                     33 = 1.1 each.
  - not described              76.

In all, the parents of 320 patients were described as imbecile, but the information as to the number and condition of the other children was lacking in the remaining 73 cases.

A few family histories fairly complete for three generations show that mental deficiency is sometimes only one of several manifestations of what is called a neuropathic inheritance, and were comparable to that of the celebrated Jukes family, wherein epilepsy, insanity, drunkenness, pauperism, prostitution, criminality, neurasthenia, and imbecility occurred alternatively in successive generations.

The following selected pedigrees serve to illustrate the various forms of Defective Heredity met with in the family histories of imbeciles:
Practically all forms of defect (mental & moral) are to be found alternating in these pedigrees.

Note: - These pedigrees are all of them the family histories of Patients in the Royal Albert Institution, Lancaster, and have been compiled from the results of special enquiries of the nearest relatives. A form was supplied to be filled up in order to obtain the particulars required with the greatest possible accuracy.
PEDIGREE N° I

Generation I. 1. 2. Imbecile
Generation II. 1. unknown. 3-4. died in infancy. 2-5-6 Imbecile
Generation III. 1. Imbecile.

PEDIGREE N° II

Generation I. 1. Imbecile. 2. Insane
Generation II. 1. 2. Imbecile.
PEDIGREE No. III

I  O1  O2  O3  O4  O5  O6  O7
II

Generation I  1-7 Normal  8. Epileptic


PEDIGREE No. IV

I
II

Generation I  1. Imbecile  2. Intemperate  Prostitute  possibly defective  3. unknown

Generation II  1.2. 3. 4. 5. Imbecile
PEDIGREE NO. V

Generation I
1. Normal
2. Normal
3. Normal
4. Normal

Generation II
1. Normal
2. Normal
3. Normal
4. Normal
5. Normal
6. Normal

Generation III
1. Died in infancy
2. Insane
3. Epileptic
4. Epileptic
5. Epileptic
6. Epileptic

PEDIGREE NO. VI

Generation I
1. Normal
2. Epileptic
3. Normal

Generation II
1. Intemperate
2. Normal

Generation III
1. Epileptic
2. Imbecile
3. Stillborn
PEDIGREE No. VII

Generation I: 1. Intelligent. 2. 4. 5. 6. Insane. 3. Inane + Intelligent. 8. 9. Normal.


Generation III: 1. 2. 5. Normal. 4. 6. 7. Imbecile. 3. Died in Infancy.

PEDIGREE No. VIII


PEDIGREE No. IX


PEDIGREE No. X


Generation II: 1. Normal. 2. 3. Epileptic. 4. Imbecile.
PEDIGREE N° XI


PEDIGREE N° XII

Generation I: 1. Insane. 2. 3. 4. No Information.

Generation II: 1. Insane. 2. Epileptic. 3-4. Imbecile

PEDIGREE No XIII

Generation I. 1. Normal. 2. 4. Intemperate

Generation II. 1. Imbecile. 2. 3. Intemperate. 4. Insane. 5. Normal.

Generation III. 1. 3. 4. 5. Imbecile. 2. Stillborn.

PEDIGREE No XIV

Generation I. 1. 3. 4. 6. No information. 5. Insane.


Generation III. 1. 4. Normal. 3. Imbecile. 2. 5. Died in infancy.
PEDIGREE N° XV

Generation I 1. Normal, 4. Intemperate, 2, 3. Normal
Generation II 2, 10. Imbecile, 5, Insane, Other Normal
Generation III, 1, 2. Imbecile

PEDIGREE N° XVI

Generation I 1, 2. Normal, 3. Imbecile
Generation II 1, 2. Normal, 3, 4. Insane, 5, 6. No Information
Generation III 1, 2, 6, 7, 8. Normal, 3, 4. Insane, 5. Imbecile, 9. Intemperate
Generation IV 1. Imbecile
PEDIGREE N° XVII


PEDIGREE N° XVIII


PEDIGREE N° XIX

I

II

III


Generation III. 1. Invalid. 2.3.4. Normal or no information.

PEDIGREE N° XX

I

II

Generation I. 1. Invalid. 2. Epileptic. 3.4.5.6.7. Normal.

7 & 8. Twins.
PEDIGREE NO. XXI

Generation I. 2. Inbreed. 6. Intemperate. 1. 3. 4. Normal

Generation II. 1. 8. Epileptic. 6. Inbreed. 2. 4. 5. 7. Normal

PEDIGREE NO. XXII

Generation I. 2. Inbreed. 1. 3. 4. Normal

Generation II. 3. Intemperate. 6. Neurotic. 1. 2. 4. 5. 7. Normal

Generation III. 2. Inbreed. 1. 3. 4. N. Information
Generation I. 1. insane. 2. normal.

Generation II. 2. epileptic. 3. intemperate. 1. normal.

Generation III. 3. 4. alcoholic. 1-2. normal.
2. THE TUBERCULAR INHERITANCE.

Most writers on the subject of Mental Deficiency have spent much time in working out the percentages of patients with a Phthisical Heredity, but when it is realised that about 90 per cent of the total population suffer from tuberculosis at some period or other of their lives, and that almost everyone has lost one or more relatives from this disease, the efficacy of this so-called heredity as a predisposing cause of imbecility will be heavily discounted.

As a fact, hardly a single family history complete for even two generations can be found in the Royal Albert Institution case books that does not show the incidence of this scourge somewhere.

This being the case, tuberculosis in any form has been entirely disregarded in estimating the causes of Amentia.

It is true that some family histories show an undue proportion of deaths from various tubercular affections, and in view of the well-known susceptibility of the feeble-minded to tubercular infection, some connection might reasonably have been suspected in the days when Tuberculosis was considered hereditary.

The fallacy however lies in this, that the low vitality of the Ament is the reason for his susceptibility to tubercle in common with all other infections. This weakness or susceptibility is in no sense a cause of his mental condition but may be regarded as a consequence of it or as a closely associated condition equally dependent on the original cause.
Pedigrees showing unusual incidence of tuberculosis, where a common source of infection could not be postulated are not numerous.

The figures given by the authorities are quoted if only to show that in this matter their divergence is even greater than usual.

Shuttleworth and Fletcher Beach allow a Phthisical Heredity as efficacious in 28.3 per cent.

The Elwyn Table (M. W. Barr for America) gives only 7.57 per cent.

Kerlin found Phthisis in one or both parents in 56 per cent.

Piper found it in parents or near relations in 23 per cent.

Langdon Down gives 25 per cent of fathers and 20 per cent of mothers.

W. Grabham allows 22 per cent.

Rogers finds it in 9.8 per cent but does not consider it a cause.

II CAUSES ACTING DIRECTLY ON THE PATIENT.

1. EPILEPSY AND INFANTILE CONVULSIONS.

A history of fits or convulsions in infancy occurs in at least 36.3 per cent of all admissions to the Royal Albert Institution.
Owing to the fact that epileptics are not eligible for admission, this figure is probably much below the total percentage in Amente generally, for it is well known that most epileptic imbeciles have a history of convulsions in infancy. It is a common experience here that children admitted with a history of infantile eclampsia, frequently become epileptic after puberty.

What is not so easily determined is the relationship between eclampsia and mental deficiency.

All observers are agreed that it is impossible to draw a hard and fast line between true epilepsy and infantile convulsions. The tendency is to describe as epileptic those attacks which recur periodically during a long period; while those attacks which are solitary or occur during one short period of illness only are considered eclampsia.

For this reason and for the additional reason that, epilepsy being a bar to admission here, there is a natural tendency to minimise its occurrence and to describe all convulsive attacks as "teething fits", no attempt has been made to separate epileptic from eclampsia cases.

A careful scrutiny of the records shows that the large majority of cases give evidence of some mental defect or backwardness before the onset of the fits, whether the fits were attributed to teething or to some inflammatory disease.

In a considerable number of cases where the child was stated to have been normal previously, the fits were dated from an attack of one of the exanthemata or of meningitis or from an injury to the head.
Many cases of injury at birth or asphyxia Neonatorum were marked by the occurrence of fits within a few hours of birth.

A residuum of cases remains however in which no pre-existing defect or disease can be traced.

When those cases have been eliminated in which the absence of discoverable cause is due to insufficient information, only very few cases remain.

It will thus be seen that the eclampsia is rather to be regarded as an expression of cerebral disease or defect than as a cause of Amentia, and that both the Amentia and the fits are due to a common cause.

The occurrence of epileptic dementia makes it probable that even a moderately severe attack of infantile convulsions may greatly intensify a pre-existing amentia and suggests that in some cases the mental defect was previously latent or was so slight as to escape untrained observation.

Tredgold and others estimate the cases in which fits are wholly responsible for the mental defect as constituting not more than 3 or 4 per cent of all aments, but they appear to be doubtful as to the propriety of considering them true aments; in fact, although classed as, and treated with aments, it would seem more correct to describe them as juvenile epileptic dement.

Dr. A. R. Moon found that 25 per cent of his cases of infantile convulsions showed some defect of intelligence and nearly 50 per cent defects of character or morals.
His conclusions agree with those above stated viz:
that the convulsions were the outward expression of a
pre-existing cerebral or mental morbid state, and were
not a cause of mental deficiency.

Fully 33 per cent of the eclampsic cases in the
Royal Albert Institution showed distinct evidence of a
neuropathic heredity. This may be compared with Gowers' figures for epilepsy. In a series of very carefully
investigated cases he found 35 per cent amongst hospital
cases and 46 per cent amongst private patients.

Dr. Moon found the same heredity in 40 per cent of
his cases.

It is of interest to note in this connection that
Shuttleworth states that more than 25 per cent of all
amints develop epilepsy at some period of their lives.

There is a well marked type of epileptic idiot, but
the epilepsy does not usually show itself until puberty,
whereas the mental defect was plainly evident in infancy;
there can be no doubt in these cases that the idiocy and
the epilepsy are the effects of a common cause. In
several of the cases examined this appeared to be
meningitis or similar diseases.

The occurrence of fits in the history of amints has
been found in very varying proportions by their observers.

Shuttleworth in an earlier series of cases from the
Royal Albert Institution case books found a history of fits
in 52.5 per cent.
Grabham found a history of convulsions in 2 per cent.
Rogers gives 4.8 per cent.
Barr who includes epilepsy, only found 5.9 per cent.
Fletcher Beach at Darenth found 27.39 per cent.
Ireland quotes Shuttleworth's figures for Earlswood Asylum as 14 per cent.
Langdon Down includes convulsions and convulsions and epilepsy as causes, but gives no figures.

2. DISEASES OF THE BRAIN.

Cases of amentia directly traceable to disease of the brain are fairly common, and constitute one of the most obvious classes of secondary or accidental amentia.

Of the cases examined 176 were found with a history of meningitis or other cerebral disease, a percentage of 8.17 of the total.

The morbid conditions included in this group are:

(a) Tubercular meningitis.
(b) Septic Meningitis (secondary to otitis media &c.)
(c) Epidemic Cerebro-Spinal Meningitis.
(d) Infantile paralysis or acute poliomyelitis.
(e) Hemiplegia and paraplegia.
(f) Meningitis or Encephalitis occurring as a sequela of scarletina, measles, whooping cough, pneumonia &c.

Several cases of hemiplegia and paraplegia were traced to the occurrence of some form of meningitis. Others were due to injuries at birth and are omitted from this and included in the appropriate group.
It is well known that by no means all cases so called infantile paralysis show evidence of mental deficiency; and on the other hand paralytic aments are not markedly common, at any rate in this Institution.

That these inflammatory conditions sometimes cause amentia may be accounted for by the disease being unusually severe and extensive or attacking the areas of the cerebrum concerned in the higher intellectual processes; and by the fact that fully one third of the cases show a marked neuropathic heredity. Any pre-existing instability or defect would be readily intensified by such disease.

That paralytic aments are not more common may be explained on the supposition that an attack severe enough to cause definite mental deficiency is more often than not fatal.

Statistics comparable with the above are not very numerous.

For the combined causes Shuttleworth and Fletcher Beach give 6.88 per cent in their table.

Barr in the American Table gives 8.42 per cent.

Rogers find a history of acute disease in 22 per cent of his cases but does not specify the particular diseases.

Tredgold appears to treat Meningitis as a very unimportant cause, and in the only case cited by him seems to blame the patients' Neuropathic Heredity rather than the disease. He also attaches but slight importance to the exanthemata, though he describes a class of secondary Amentia caused by various toxic conditions among which he includes scarletina, measles &c.
He describes the macroscopic and microscopic changes caused by various morbid processes in the brain but gives no indication of his estimate of their value as causes of Amentia.

3. INJURIES TO THE HEAD.

Injuries to the head caused by falls or blows and causing concussion account for 149 cases = 6.9 per cent.

A history of falls or blows on the head is very frequently given by parents to account for the patient's condition; but very few children reach adolescence without suffering many such accidents, while permanent after effects are uncommon.

No cases have been included in this group unless there is a very definite history of concussion or serious injury, so it is possible that a few have escaped inclusion; but as will be seen the figures tally closely with those of other observers.

The fact that in nearly 40 per cent of these cases there is neuropathic heredity may help to explain the serious consequence of apparently slight accidents.

Barr gives 6.36 per cent.
Shuttleworth and Fletcher Beach give 6.17 per cent.
Rogers only found traumatism in 2 per cent of his cases.
Tredgold finds it is undoubtedly the cause of a few cases but gives no figures.
4. INJURIES DURING PARTURITION.

In these cases there is a history either of precipitate labour, the child being projected on to the floor, or of extremely prolonged labour with subsequent asphyxia neonatorum. In many of these cases there is a further history of convulsions occurring within a few hours of birth. There were 295 cases = 13.7 per cent.

There is no doubt that a history of asphyxia at birth is very common in the records of amits. In a few cases where the question was asked it was found that the asphyxia was of the so called pallid type, which is not true asphyxia such as occurs when the cord is tightly twisted round the neck of the child or is knotted or prolapsed, but consists rather in a defect of the respiratory reflex such as might be caused by long continued or extreme pressure on the central nervous system.

It has been held by many that this condition is most often found in first born children; and is asserted to support the theory that the first born is more likely to be defective or delicate than the subsequent ones. It certainly sounds reasonable, as the first labour is notoriously slow and lingering in the great majority of cases - but I have been unable to find any confirmation of this in the Royal Albert Institution records where the position of the patient in the family is carefully noted whenever it can be ascertained.
Shuttleworth and Fletcher Beach found a history of injuries during labour &c in 19.01 per cent and Fletcher Beach found a history of asphyxia also in about two thirds of them or 12.96 per cent of the total number examined.

Barr records only 1.81 per cent.

Langdon Down gives 20 per cent for all imbeciles and 40 per cent for first born imbeciles.

Tredgold found 16 per cent, but states that the majority had a morbid heredity also and is of opinion that it is the immediate cause of only 1 or 2 per cent. He allows however that it may be a very important contributary cause.

5. SHOCK.

A few cases (55 = 2.5 per cent) seem reasonably attributed to severe nervous shock in the shape of burns and scalds, outrage, fright &c, and a small number (10 or 12) give a history of neglect, continued illusage or the repeated adminstration of sleeping draughts, soothing syrups, beer, spirits &c in early infancy. Total 3 per cent.

Barr finds a history of abuse, neglect, exposure, only .66 per cent, accidents other than head injuries 1.67 per cent and abuse of drugs in .82 per cent - a total of 3.15 per cent.

Rogers gives 3 per cent for the first, 1.4 for the second and 1.4 for the third group of causes - a total of 5.8 per cent.
Shuttleworth and Fletcher Beach attribute 3.06 of their cases to fright or shock, but do not mention the other groups of causes as given above.

6. RICKETS.

Rickets, does not appear to exert much, if any, influence on brain development. Very few patients in this Institution show signs of the disease, in spite of the fact that a few years ago it was very prevalent amongst the mill operatives and colliers of the Northern Counties. It does not appear as a cause in any Statistics to which I have access and where mentioned is treated as being of no effect.

Tredgold says it may have some causal effect but appears very doubtful.

On the other hand, Gowers quotes with approval the opinion of Sir Wm. Jenner that Rickets is the most potent agent in the production of infantile convulsions and subsequent epilepsy.

If the above apparently contradictory opinions are correct, they furnish further confirmation of the view expressed in dealing with the relationship of epilepsy to Amentia and strengthen the conclusion that epilepsy is not in any true sense of the word a disease but is merely a symptom of an abnormal condition of the cerebral tissue which has been brought about by various unrelated causes; and it cannot therefore be seriously considered as a etiological factor in the production of mental defect.
Congenital Syphilis has not been shown to be a prominent factor in the production of Amentia. A very small number of cases in the Royal Albert Institution show signs of the disease and only rarely does a pedigree point to the occurrence of syphilis in the parents. In fact it would almost seem to be less common in amongst aments than it is usually stated to be amongst the normal population. The reason may be that a defective child born of syphilitic parents would probably be more gravely affected than an otherwise normal child and therefore would either be still born or would die in early infancy.

This agrees with Mott's view "that there would undoubtedly be a considerable larger proportion of defective children from this cause were it not for the high rate of sterility, miscarriages, still born and short lived offspring that it produces."

A careful examination by Shuttleworth of the cases in this Institution showed evidence of syphilis in only 1.71 per cent.

Barr could find it in only .20 per cent.

Tredgold found evidence in 2.5 per cent but attributes only .5 per cent to this cause acting alone.

Langdon Down and Kerlin give 2 per cent.

Grabham and Ireland also arrive at the same conclusion but give no figures.
Syphilis has been given as a cause of Mongolism by Sutherland but I cannot find any other observers have confirmed his conclusions.

CAUSES AFFECTING THE HEALTH OF THE PARENT.

1. PLUMBISM.

Plumbism either accidental or as the result of taking abortifacients containing lead, seems to have a deleterious effect on the brain of the foetus that survives to term. A history of plumbism in one or other parent corresponding to the period of gestation is given in eleven cases.

Paul, quoted by Tredgold, gives very striking statistics of the fatal effect of paternal plumbism on the foetus, and Tredgold himself considers that lead poisoning may be a cause of amentia in a few cases but gives no figures.

Barr does not mention plumbism as a possible cause nor do other authorities appear to consider it sufficiently important to deserve special mention.

2. INTEMPERANCE.

Marked intemperance in one or both parents is noted in 375 cases = 17.4 per cent. In these cases the coincidence of the intemperance with the intrauterine life of the patient has been carefully noted. The large majority of these cases show evidence of a neuropathic heredity as well, so that the parental intemperance has rather served to develop a hereditary defect than to actually cause amentia.
Barr gives only 4.46 per cent for intemperance in the parents amongst American Idiots.

Tredgold finds a pronounced family history of alcoholism in 46.5 per cent.

Shuttleworth and Fletcher Beach give 16.38 per cent.

Ireland considers it much over rated as a cause but does not give figures.

Langdon Down on the other hand considers it a factor of great importance but also gives no figures.

C. T. Wilbur of Illinois found very few (8 in 365).

Howe found nearly 50 per cent of Idiots had intemperate parents.

C. Loof gives only 3.7 per cent and Kind 11 per cent.

Rogers finds it a factor in 15.45 per cent.

Kerlin 38 per cent.

The figures of other observers vary as greatly one from another as do the above.

3. ABNORMAL CONDITION OF MOTHER DURING GESTATION.

Other conditions that adversely affect the health of the mother may interfere with the normal development of the child's brain. A history of worry and anxiety, chronic ill health, acute illness, over work and starvation &c during pregnancy is given in 116 cases = 5.4 per cent and no doubt in some cases has contributed to, if it has not actually caused the mental defect.
These conditions variously estimated and grouped are accepted by previous writers, but the majority appear to consider seriously and include the very large number of cases in which there is a history of sudden fright shock or fall or an account of some alleged maternal impression. In cases where these accidents have produced premature birth it is possible that they may have contributed to the mental defect, but in the large majority little credit can be given to such causes and the circumstantial details serve merely to increase one's scepticism as to the power of maternal impressions, so called, to produce this or any other abnormality.

A few cases taken at random that permitted of personal examination both of the patient and of his parents rendered unnecessary the admission of this type of cause in face of the very obvious other causes that had been at work.

Cases of Mongolism and of harelip attributed to shocks or falls within a month of the birth of the patient are not uncommon.

From the following figures it will be seen how greatly these mental cases predominate over physical ones; but a proper comparison of these figures with those given above is not possible owing to the different standpoint adopted by the compilers.

Shuttleworth gives 15.92 per cent for abnormal mental states in the mother apparently including both worry and shocks and frights and 5.5 for abnormal physical states.
The American Table gives 8.49 for the combined causes. Fletcher Beach gives 38.47 per cent for the combined causes in the tables drawn from Larenth cases.

Langdon Down finds Mental Disturbance in 32 per cent and physical disturbance in 20 per cent.

Graham found a history of shock or grief in 16 per cent.

Rogers gives 11.6 and Kerlin 7 as the percentages for abnormal states in the mother.

In this connection the question of "Siege babies" is of interest, most observers have noted an increased percentage of abnormal or defective children born during and shortly after a prolonged Siege.

Baron Percy found 57 defectives out of 92 children born during or shortly after the siege of Landau in 1793.

A similar state of things is said to have occurred in consequence of the siege of Paris.

In these cases it is impossible to separate the effect of the physical from that of the mental stress undergone by the mothers, and the cases should be included under those caused by long continued strain rather than by sudden shocks or frights; all observers agreeing that the continued anxiety and insufficient food are more distressing than the occasional alarms or the disturbance caused by a heavy bombardment.
This concludes the list of causes that have been generally accepted as contributing to the production of Amentia; and careful scrutiny of a large number of family and personal histories has failed to indicate any other probable cause; neither have the writings of previous observers given any indications that could be followed up. So far as I have been able to go, all the cases that admitted of sufficient detailed examination could be accounted for by one or more of the causes enumerated above.

Certain types of Ament are so readily distinguished by their physical characteristics from the generality of defectives and from one another - forming in fact a number of distinct varieties - that it is reasonable to expect and search for a cause common to each variety.

This search has been fairly successful in some cases, but some of the problems remain unsolved.

The types referred to are the Microcephalic, Hydrocephalic, Cretinous and Mongolian or Kalmuc.

1. MICROCEPHALIC.

Too few cases of Microcephaly were available for examination to enable any satisfactory conclusions to be drawn as to their etiology; more particularly as various authorities hold different views as to what constitutes true microcephaly.
Shuttleworth has rightly insisted that it is not merely a question of the size of the head, but that microcephaly is a definite condition with distinct features, physical, anatomical and mental.

Ireland on the other hand relies entirely on the cranial measurements and includes all heads under 17 inches in circumference.

All authorities agree that it is a condition of defective brain development, but do not seem to have seriously considered the question of what cause or causes lie behind the anatomical condition.

No cases were accepted that did not answer fully to the classical description of the condition and in these the details of the family histories were mostly meagre. There was evidence of a strong neuropathic taint in nearly all and the only other circumstance to be noted was that they were in several cases members of large families.

Neither of these conditions separately, or both combined, are peculiar to microcephaly and nothing was found to indicate the determining factor.

The following pedigree is that of a family of which two members - both well marked microcephalics - are inmates of this Institution. I am informed that the other two imbeciles were "very like these" and that the heads of all the children were small. I was unable to get any information about either of the mother's husbands or their relations, the mother herself being but little better than an imbecile but one of her brothers and one sister were insane; the remainder were described as normal or delicate.
The third child died of acute meningitis. The ninth and thirteenth children had very small heads and died of convulsions when 5 months and fourteen days old respectively.

PEDIGREE NO. XXIV [Microcephaly]

II. HYDROCEPHALUS.

Marked cases of Hydrocephalus are not often admitted to the Royal Albert Institution, probably because they rarely attain the necessary age or if they do, are of such extremely feeble physique as to be unsuited for reception into an Institution for the training of the feeble-minded.

Lesser degrees can hardly be diagnosed with certainty except Post Mortem, and unfortunately the material is too scanty to be of use.

Amongst the earlier cases, hydrocephalus—acute or chronic—is not infrequently given as a cause of death but an examination of the Post Mortem records raises a suspicion that some, at least, were tubercular meningitis.
In any case the anatomical condition is paramount, the mental being purely secondary and the etiology is a question for pathologists.

The writers consulted all agree that the condition is the result of disease but they differ as to the exact pathological condition that results in the accumulation of fluid within the cranium, and no light is thrown on this subject by any material at hand.

It would seem however that heredity plays little if any part in the etiology of this disease.

III. CRETINISM.

Sporadic Cretinism has been noted by previous observers to be extremely uncommon in the Northern Counties of England; this accounts for the very small number of cases admitted to the Royal Albert Institution; too small to throw much light on the causes of this condition. Further, as a coincidence, the histories of most of them are so scanty as to lead to their inclusion amongst the cases that has to be omitted from the totals on account of insufficiency of data.

They were all of the Sporadic Type, no cases of Endemic Cretinism being now known to occur in the district served by the Royal Albert Institution.

The Literature of Endemic Cretinism is very large and complete but except for a very full and accurate description of Sporadic Cretinism by Ireland the condition does not appear to have attracted much attention from earlier observers.
One reason no doubt is that it is a rare condition and consequently did not offer much field for investigation.

The discovery of the immediate cause—absence or deficiency of the Thyroid Gland—no doubt attracted much attention to the condition some years ago; but all theories to account for the defect have been tentative and have failed to receive general support.

Some American authorities suggest syphilis, while Shuttleworth hints that it is evidence of a hereditary taint; but I find no other attempted explanations; in fact Tredgold confesses himself completely at a loss as to the cause.

The fact that two of the cases in the Royal Albert Institution were the youngest members of large families suggests a possible relationship to Mongolism—a condition to which it presents some superficial resemblances—but beyond this very tentative suggestion I am unable to go.

IV MONGOLISM.

Mongolism is one of the commonest types of Amentia in this district, and an examination of the records of a large number of cases (nearly 150) strongly confirms Shuttleworth's conclusion that the determining factor is an exhaustion or deficiency of the reproductive energy of one or both parents; that Mongols are, in fact, "unfinished children."
At least 5.3 per cent of all admissions to the Royal Albert Institution are described as mongols.

In 72 per cent of them the patient was the youngest child, in 16 per cent there was a history of ill health or acute illness of continued worry in the mother during pregnancy. In a few cases the patient was the next to the youngest coming at the end of a series of rapidly recurring pregnancies. The youngest, if born after a considerable interval, being normal - if not, either also a mongol or still-born.

In 30 per cent the mother's age was 40 years and upwards. In 2 cases the father's age was over 75 years, in 4 cases where the patient was the eldest child there is a history of worry and anxiety owing to the child being illegitimate.

As showing the complex nature of the etiology of so well defined and apparently simple a condition, 54.4 per cent showed distinct evidence of a neuropathic heredity, in 50 per cent of these one or both parents were themselves defective, in 16 per cent one or both parents were markedly intemperate.

Three mothers had each two mongol children.

The sister of one mother had also a mongol child. In this case the mother was twice married and the youngest child of each family was a mongol.

The following is a pedigree of these three mongols. Apparently there was no neuropathic heredity. The causes of death were various but in no way remarkable and there is almost an entire absence of Phthisis. Most members of the earlier generations lived to a good age.
Ireland says that 3 or 4 per cent of all imbeciles are of the mongolian type; but offers no suggestions as to the cause, merely disagreeing with Langdon Down's theory that they are the product of a marked tubercular heredity. This theory obtains no confirmation from the families histories in the Royal Albert Institution Case Books nor is it accepted by Barr, Tredgold, Wilbur and others.

Barr found only .09 per cent amongst his cases; only one case was the youngest born, and only one had aged parents. He is unable to discover any common factor amongst them that might throw a light on the cause.

G. A. Sutherland (Practitioner 1899) suggests syphilis as a probable cause, but this theory obtains no support from other writers nor from the histories of my cases.
Tredgold ascribes it to "uterine exhaustion" which agrees with the conclusions drawn from the Royal Albert Institution cases, but postulates also a strong Neuropathic Heredity. He however gives no figures for either condition.

W. B. Hill quotes with approval Weygandt's conclusion that "the majority were the last children of a large family and that the parents were often old," and the histories of his eight cases fully confirmed this.
CONCLUSIONS.

It will be seen that the etiological factors which go to produce Amentia are many and various, and their relative importance still remains to be gauged by the value of the various statistics published from time to time. The comparison of nearly all the previously published groups of observations with the large number of cases available for the purposes of this thesis, should be of service in helping to elucidate some of the problems of this important subject.

There seems little room for doubt that heredity plays the most important role in the production of Amentia. The heredity may be homogeneous i.e. the mental defect of the parent is reproduced in the child; it may be heterogeneous, where an insane or epileptic parent has imbecile children, or it may be diffuse or indirect - where the neuropathic strain shows itself in a marked susceptibility to nervous or cerebral disease or mental breakdown as a result of comparatively slight causes.

This generalised or diffuse neuropathic strain is frequently shewn in an unusual number of deaths from nervous diseases or the occasional production of drunkards, prostitutes or unemployables in earlier generations. In these cases the parents may pass through life without showing any evidence of defect and the children are considered normal until the occurrence of some acute disease or accident leaves behind it effects from which children with sound heredity would escape.
I have searched carefully for, and failed to find any convincing proof of the importance of a so-called tubercular heredity, or that the occurrence of numerous cases of Pthisis was at all strikingly evident in the family histories of Aments.

The occurrence of two or even three deaths from tubercular affections in previous generations can now-a-days hardly be considered sufficiently uncommon to bolster up a theory based on an alleged hereditary susceptibility to tubercular infection; and yet it is evident from the writings of earlier observers that the occurrence of even one case amongst near relatives was held to justify the inclusion of the case in the class of hereditary Aments and in the subdivision of Scrofulous or tubercular cases.

Epilepsy is now considered to be of the nature of a symptom or an expression of a morbid condition of the brain rather than a disease and therefore it is difficult to understand how it can act as a cause.

That the defective brain is unduly prone to epileptic and eclampsic discharges is universally admitted, but to say that because the mind of the confirmed epileptic may eventually show signs of degeneration (though this is not an absolute rule) therefore epilepsy is the cause of the degeneration, and may therefore also arrest the development of the mind in infants savours too much of the "post hoc ergo propter hoc" type of argument to merit unhesitating acceptance.
The fact that less than half the children who suffer from convulsive attacks in infancy show signs of mental impairment to even the slightest extent further discounts the value of this disease as a cause of the impairment.

There are many analogies drawn from the histories of adults which supply arguments in favour of ascribing certain cases of Amentia to injuries and diseases affecting the brain. The dementia which follows apoplexy and extensive accidental laceration of the brain substance; the loss of memory or alteration of character sometimes seen after severe concussion, and the progressive mental deterioration that accompanies some forms of chronic inflammatory or degenerative nervous disease, have all their analogies in various types of Amentia.

For the rest - I have found it easier to ascribe Amentia to abnormal physical than to abnormal mental states in the pregnant mother and to conclude that where the abnormal maternal factor was purely mental the effect on the foetus was secondary to its effect on the maternal metabolism.

The etiology of Mongolism seems fairly well established now, and the cases examined for the purposes of this thesis merely go to confirm very strongly the accepted theory.
The pathology and morbid anatomy of Hydrocephalus offer a practically unexplored field for research. Such modern Brain Pathologists as Bevan Lewis, Ford, Robertson, Tredgold and Mott have entirely ignored this disease, and works on general pathology are very little more informing.

So, also, much remains to be done in investigating the causes of Cretinism and Microcephaly. It is probable that heredity plays a large part, and possibly the maternal health is an important factor, but the determining cause has so far not been found.
BIBLIOGRAPHY.

Figures and opinions from the following works have been quoted:

(1) Mental affections of Children by W.W. Ireland, Edinburgh 1898.


(3) Mental Deficiency (Amentia) by A.F. Trédgold, London 1908.

   (a) Artes: on Imbecility & Idiocy by J. Langdon Down.
   (b) Artes: on Imbecility & Idiocy &c by G.E. Shuttleworth & Fletcher Beach.

(5) Desyndsyge i Norge by Ludwig Dahl, Christiania 1859.

(6) Causes and prevention of Idiocy by S.G. Howe, Boston 1848.


(8) The origin varieties and termination of Idiocy, by G.W. Grabham, London 1875.

(9) The ascribed causation of Idiocy, by A.C. Rogers, Iowa 1885.

(10) Epilepsy by Sir W. Gowers, London 1901.


(14) Plumbism and the foetus, by Constantine Paul, Paris 1861.

In addition the following Authors have been consulted.

Idiocy and its treatment &c, by Ed. Segun, New York, 1866.
The feeble-minded, by E.B. Sherlock, London, 1911.
The Pathology of Mental Diseases, by W. Ford-Robertson, Edinburgh, 1900.
Arts: by F. Warner, L. Guthrie, E. Jones &c.
Art: Idiocy by J. Langdon Down.