SOME OF THE STRUCTURAL ABNORMALITIES PRESENTED BY THE BRAINS OF THIRTY-ONE CERTIFIED MENTAL DEFECTIVES

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INTRODUCTION

In this paper an attempt is made to analyse, by rather different methods than the more purely anatomical, certain macroscopic features of the brains of 31 certified mentally defective patients, details of which are given in Appendix I.

Studies of the macroscopic appearances of the defective brain, in so far as these are causally associated with the resultant mental deficiency, have not been, in the past, very numerous. Fernald, in the *Waverley Researches in the Pathology of the Feeble-minded*, speaks of the 'extraordinary neglect into which this topic has fallen,' and adds that 'the anatomy of feeble-mindedness has not been attacked systematically over a period of years.' Fernald himself endeavoured to bridge this gap by giving complete anatomical descriptions of the brains of 10 defectives. But purely descriptive anatomical methods no longer suffice and are of little or no help to the mental specialist. Even the descriptions given in the most modern text-books of anatomy are of so stereotyped a nature as to afford no measurable standard against which to set supposedly less well developed brains. As the anatomical description of the normal human brain no longer accords with more recently discovered facts of cortical construction it is obviously in need of revision.

Craniologists, on the other hand, have long abandoned purely descriptive methods as applied to human skulls and have substituted therefor a series of accurately recorded drawings from which measurements and angles can be subsequently calculated. From these can be established almost any desired comparison between normal and abnormal.

It is on these lines that it is now proposed to discuss some of the macroscopic details of these 31 defective brains, and for these purposes the dioptrograph, as an instrument of precision, is as well adapted for the brain as the skull, though it is obvious that for both skull and brain dioptrograph tracings have their limitations.

In a previous communication to this JOURNAL the brains of two of the present series (nos. 8 and 9) were macroscopically and microscopically described and illustrated.

The fissuring of the cortex of the whole of the present series is as obviously irregular as was that of these two and is so in very much the same places—

namely, the pre- and post-central cortical areas, the effector precentral gyrus, the opercular cortical areas and the parietal association areas. In fact, the anatomical descriptions previously given of brains 8 and 9 would apply, with but little modification, to most brains in the series. From a naked-eye examination alone of the sulci and gyri and their marked irregularities it would be legitimate to draw the conclusion that these brains are not, and never have been, sufficiently developed to give normal human reactions to normal human environments.

But such general and personal impressions require some more substantial support than the unaided eye of the observer. Hence dioptrographic tracings of the right cerebral hemispheres of the whole of the series were made. On these, lines and angles could be measured, planimeter areas recorded, and the main features of the fissuring studied and measured as future experience might suggest. Whilst this method permits of an accurate comparison between any two or more brains of the series it does not, with very few exceptions, permit of that comparison between abnormal and normal which it is so essential to establish, because the corresponding data for the normal are not yet known.

PERSONAL STUDIES

Commencing first with those facts which do permit of an accurate comparison between these 31 defective brains and those usually accepted as representative of the normal there fall to be considered :

1. The angle of the Rolandic fissure.
2. The length of the Rolandic fissure.
3. The height and index of the skull and brain.
4. The actual size of the cortical hemisphere itself.

1. 'The acute angle which the slope of the fissure of Rolando (central sulcus) forms with the superior border of the hemisphere is known as the *Rolandic angle*. According to Cunningham it averages 71.7° ' (Quain). Eberstaller found that in adults it varied from 70° to 75° . In these defectives the Rolandic angle only averaged 56.5° with a variation of from 31° to 90° , the latter occurring in Case No. 19, which was complicated by ventricular hydrocephalus. In view of the experience of Ariëns Kappers, who found that the Rolandic angle acquires its definite position only about the fifth month after birth, this range of variability in defectives is suggestive of a foetally ill-developed brain retained throughout life.

2. *The length of the Rolandic fissure* measured between the two extremities of the fissure and in a straight line over the arc of the hemisphere is given by Quain as about $3\frac{3}{4}$ inches (8 cm.) and by Symington and Crimble as about 9 cm. in direct line from the ends of the fissure, and 10.4 cm. following the curves of the sulcus.

On the dioptrographic tracings of these defective brains it is the chord, not the arc, which is measured, and this averages only 5.7 cm. with extremes from 4.3 cm. to 8 cm., from which it would appear that these defective brains

show significant reduction in the length of the Rolandic fissure. As this fissure is a cortical infolding between the whole lengths of the important postcentral somæsthetic area and the precentral effector, it follows that a significant reduction in the length of the Rolandic fissure denotes, in these defectives, a diminution in the functional cortical tissue of the greatest importance in mental activities.

3. The *height index* is a ratio between height of skull and length, and is used by craniologists as a standard of racial comparisons. Combined with other measurements and indices it gives some indication of the form of the head and associated size of the brain. The index is expressed by the formula

$$\frac{\text{Basal height} \times 100}{\text{Maximum length}}$$

In general, the lower the index the lower the cranial form, and the index usually ranges from 72 to 77. Skulls with the former reading or less are technically termed platy- or tapeinocephalic. In children's crania the average index is 75.2 (Duckworth).

Diopetrographic tracings of one cerebral hemisphere enable the similar index for the brain to be calculated with the greatest accuracy. In the present series of defective brains the cerebral height index only averages 60.3, with extremes from 54.4 to 72.7—that is, all have lower cerebral indices than the corresponding average cranial figures given by Duckworth for children.

4. *The actual size of the cerebral hemisphere itself.* Notwithstanding the large amount of information for brain weights, cubic content of skull, and similar facts, there is but little available for actual linear dimensions of the normally developed cerebral hemispheres, and without this it is very difficult to institute comparisons of size between defective and normal brains. Several leading English and American text-books of anatomy or neurology seem, however, to make the following fairly uniform statement :

‘The length of the cerebral hemisphere measured from the frontal to the occipital pole varies in the majority of cases from 160 mm. to 170 mm. for the male, and 150 mm. to 160 mm. for the female. The *greatest* vertical diameter is 125 mm.’ (Tilney and Riley). It will be observed that these, at best, but scanty facts afford a range of variation for cerebral length but give only the *greatest* vertical diameter. The corresponding facts for these 31 defective brains and their comparison with the figure just given in the reputedly normal brain may be tabulated as follows :

Normal	Defective
Fronto-occipital length : Males, 160 mm. to 170 mm.	160 mm. (14 males)
Fronto-occipital length : Females, 150 mm. to 160 mm.	152 mm. (17 females)
Greatest vertical height : Both sexes, 125 mm.	100 mm. (31 cases)

As regards cerebral length the defectives stand at the lowest known figure quoted for normality. For vertical height they are 25 per cent. less than the 'greatest' vertical height. This last fact, read in conjunction with standard anthropometric methods of calculating cubic contents of skull from measurements of length, breadth and height, assumes clinical significance because it substantiates other observations on the occurrence of small-headedness (microcephaly) in about two-thirds of certified defectives. In the brains of these 31 defectives the diptrographic measurements of vertical height ranged from 80 mm. to 120 mm. (once) with the average already given.

Male occipitofrontal length ranged from 145 mm. to 180 mm. (once) and the last was pathological. Female cerebral length ranged from 138 mm. to 170 mm.

With the objective of establishing, if possible, some direct comparison of cerebral size between defectives and normals it was determined to multiply

FIG. 1.—Diagram showing the sizes of the 31 defective brains in percentages of the lowest available normal figure, the latter being taken as 100 per cent. Males are shown by continuous vertical lines, females by interrupted ones. The numbers at the bottom are those of the cases as set forth in Appendix I, whilst the percentages are on either side.

cerebral length and breadth together, taking the lowest available figures for normality and regarding the product as 100 per cent., to convert the slightly lower figures for normal females to the same standard by means of the factor 1.067 and then to set against this scale the actual figures for the defectives. The results of this procedure are shown in fig. 1, where the defectives are arranged in chronological order of age—that is, in the same order as that given in Appendix I.

The first and most striking fact brought out by this comparison of cerebral length-height area of size is that the defectives have a brain 20 per cent. less than the lowest figure for normals.

Secondly there is, in these defectives, little if any indication of an association between advancing age and size of brain. It is true that the individual differences are so great that any such association might be obscured, but in addition it is not unlikely that the defective brain does not undergo those increments in size with age that it should.

DISCUSSION OF RESULTS

In so far, therefore, as it is possible to establish comparisons of these defective brains with known data for normals for size of brain, length and angle of the Rolandic fissure, length of the associated cortical areas, and the cerebral height index, all the observations—taken either separately or collectively—go to suggest that the defective brain more nearly resembles that of an eight-months' foetus than of a more normally postnatally developed one. For subsequent post-natal, and, in these cases, slight differences of mental reactions, it seems most likely that microscopic investigation can alone indicate what, if any, are the post-natal cerebral alterations in the brains of defectives as contrasted with normals. Whatever their nature, that they are of an abnormal character seems certain, as does also the fact that, minute though they may be in nature and origin, they are mirrored in the macroscopic appearance of the brain itself.

Passing next to the consideration of the variability of these defective brains within themselves and for which there are insufficient or no data available for comparison with the normal, there may first be discussed the variability in the actual sizes of the defective brains themselves.

It has already been pointed out that in these series of 31, the average defective brain is 20 per cent. less than the lowest normal. Fig. 1 shows that even this low figure is not uniform but displays a considerable range of variation from 61 per cent. of the normal (Case 5) to 103 per cent. in Case 19.

Six cases are only 70 per cent. or less of the normal. Nine cases are between 70 and 80 per cent. of the normal. Eleven cases are between 80 and 90 per cent. of the normal. Four cases are between 90 and 100 per cent. of the normal. One case is above the lowest normal with 103 per cent.

As regards those defectives whose brain size was 90 per cent. or less than that of the lowest normal there does not appear to be any significant or ascertainable association between these variabilities in brain size and the mental ratios during life. Thus Case 31, with a brain size only 70 per cent. that of the normal and a chronological age of 35.11 years, had a Binet mental ratio of 6.10 years, whereas none of the nine cases between 70 and 80 per cent. of the normal, and with ages ranging from 4.8 years to 30.7, had an ascertainable mental age during life of more than three years.

When, however, attention is directed to the five cases with 90 per cent. and upwards of the lowest normal brain size—i.e. to those more nearly approaching normality—some possibly significant facts begin to emerge.

Of these five cases, which are those with the largest brains in the series, one—15—is the highest grade case of all, having been rated during life as low grade feeble-minded with a Binet mental ratio of 6.10 years. Two—Cases 19 and 22—are definitely pathological (ventricular hydrocephalus and tuberous sclerosis), whilst the remaining two (24 and 25) certainly seemed, on microscopic examination, to owe their comparative but relative

bigness of brain to other pathological conditions not obvious to the naked eye but revealed by the microscope, one case being definitely due to an abnormal glial increment.

It would thus seem as though defectives are so on account of their ill-developed and unduly small brain : in this respect they are, even within themselves, extremely variable, and lastly, that those more nearly approximating to normality in brain size only do so on account of a more nearly normal mental ratio or of some underlying pathological condition of a gross or microscopic nature.

FURTHER PERSONAL STUDIES

In view of this large and apparently not insignificant range of variation in the brain size of defectives an attempt was next made to discover, if possible, wherein these differences lay. Was some part relatively over-developed in relation to others ? Were there, for instance, any appreciable or discoverable differences between prerolandic cortex and postrolandic—that is, between frontal lobe and the rest ? To attempt to answer these questions proved by far the most difficult part of the investigation, and notwithstanding the many and varied procedures adopted all seemed to result in consistent failure to produce any result at all—let alone an understandable one.

‘The general conclusion (says Herrick) to be drawn from the entire series of physiological and pathological studies of the cortex is that specific mental entities are not resident in particular cortical areas, but that cortical functions involve the discharge of nervous energy from one or more sensory centres to various and remote regions . . . the destruction of any part of the cortex or of the fibre tracts connected therewith involves first a permanent loss of the particular functions served by the neurones affected, and in the second place a transitory disturbance of the cortical equilibrium as a whole (diaschisis effect).’

Speaking very generally and in terms of the foregoing quotation, it may be accepted that the postrolandic portions of cerebral cortex (the anatomical parietal and occipitotemporal lobes) are more definitely receptor or sensory in function than is the prerolandic frontal lobe. Further, if under normal conditions of cerebral development subsequent destruction of any part of the cortex produces those recognizable effects mentioned by Herrick, then the question arises : does a maldevelopment, or a grossly unequal distribution, of postrolandic and prerolandic cortex produce similar effects and, if so, in what way are the behavioural reactions of the individual altered from those resulting from more normally developed brains ?

As already mentioned, many attempts were made to provide, from the present series of defective brains, some negative or positive answer to these problems, with but little appreciable result. After much discussion with the Medical and Consultant Staffs it was eventually decided to compare the actual

planimeter areas of the pre- and postrolandic cortical regions. There was accurately recorded on the dioptrographic tracings of the lateral (external) surface of each of these defective brains the superior and inferior Rolandic points, the central sulcus of Rolando, the lateral Sylvian fissure, the precise point at which the parieto-occipital fissure cut the superomedial border of the cerebral hemisphere, as well as the main cerebral sulci themselves. With these points and lines accurately located it was easily possible to draw in on the dioptrographic tracing the line of the chord of the Rolandic fissure, the chord of the parietal arc, and the outlines of the external surface of the parietal lobe itself. The anatomical description of the delimitation of this surface of the parietal is so inaccurate and misleading as to be useless for purposes of measurements. In the present series the parietal lobe is delimited by the Rolandic fissure; the superomedial border of the hemisphere from the superior Rolandic point to the parieto-occipital fissure, and completed by a line drawn from that point to the inferior Rolandic point or a line drawn vertically therefrom to the Sylvian fissure. The importance of the enclosed portion of cortex lies in the fact that within it is the parietal association area so closely related, structurally, to the incoming neural streams from the primary visual, acoustic and general somæsthetic senses. These facts are in themselves so important as to necessitate some more accurate method of parietal lobe delimitation than that given in textbooks, hence the method herein suggested and adopted.

The pre- and postcortical areas having thus been accurately delimited on the dioptrographic tracings, as well as the component parts of the postrolandic cortex—that is, its parietal and occipitotemporal constituents—the extent of each area was easily obtained by the planimeter, with the results given in Appendix II.

It is to be noted that this method is open to criticism, inasmuch as it only measures cortex visible on the surface and takes no account of that within the depths of the sulci, usually estimated at twice the amount on the surface. Secondly, it does not deal with the total amount of anatomical lobar cortex, but only with that present on the lateral or external surface of the brain; and, lastly, it records only the linear extent of the areas measured, taking no account of the convex surface involved.

But other methods, such as Wagner's, of covering the whole of the cortex with gold leaf and determining the extent of surface by the amount employed, or Baillarger's attempt at dissecting off the grey cortex from the white matter, are so laborious as to be impracticable over any large series of observations, and if these are not sufficiently numerous the results are of little practical value. It is therefore impossible, at the moment, to establish any comparison of the inequality of cortical areas as between normal and abnormal. The latter, however, proved even with the dioptrographic method to present so many variations as to justify the time and labour bestowed on their ascertainment.

Fig. 2 displays these 31 defective brains in order of the actual extent of their prerolandic frontal cortex (solid black columns). To the immediate left is shown for each case the total amount of postrolandic cortex, the parietal portion of which is indicated by vertical lines, whilst the occipitotemporal is

FIG. 2.—Diagram showing the areas of the pre- and postrolandic cortices on the external surfaces of the brains of 31 defectives, arranged in order of size of area of the prerolandic cortex.

The prerolandic cortex is indicated by the solid black vertical column, with the postrolandic portion to their immediate left. The parietal part of the latter is vertically shaded, and the occipitotemporal unshaded.

The cuneus on the medial surface of the brain is, for diagrammatic purposes, indicated by the cross-hatched lower area.

The line A indicates the average slope of the cuneus area; the line B that of the parietal; the line C the prerolandic cortex; the line D that of the postrolandic cortex; whilst the line E indicates the average slope of the actual sizes of the brains themselves.

For the interpretation of the diagram see text.

left clear. The small cross-hatched area at the bottom of the left-hand column has nothing to do with the amount of cortex measured on the external cerebral hemisphere and will be separately referred to later. As between two of the extremes of the series, Cases 5 and 15, the diagram shows, for example, that the amounts of the pre- and postrolandic cortices were as follows :

	Case No. 5	Case No. 15
Prerolandic	4.54 sq. in.	8.25 sq. in.
Postrolandic	9.13 „	12.37 „
(a) Parietal	2.93 „	5.04 „
(b) Occipitotemporal	6.20 „	7.33 „
Total brain surface	13.67 „	20.62 „

Whatever the mentality, and whether normal or abnormal, it seems difficult to believe that one individual can have almost twice as much effector or prerolandic cortex as another without affecting, in some way, his behavioural reactions to the environment. In the two cases contrasted above, one—Case 5—is that of an imbecile with an assessed mentality during life of less than three years, whereas the other—Case 15—has the highest mental ratio of the series, with a Binet mental age of 6.10 years and a chronological age of 17.7 years.

But the brain of Case 15 is not only one of the best endowed with a prerolandic cortex (Case 22 is excluded because it was definitely pathological) but is also the most evenly proportioned as regards its ratios of parietal and occipitotemporal receptor constituents to prerolandic effector; this was also the case which most nearly approached during life to the lower levels of normal mentality.

The diagonal line D represents the average amounts of postrolandic receptor cortex, whilst the line B represents only that portion of it which is formed by the parietal area. If the parietal cortex—which is very important for the reasons already given—kept pace developmentally with the rest of the postrolandic cortex, the two lines B and D would be more nearly parallel. That they are not so shows that in these defectives the parietal association area is, relative to the rest of the brain, lagging behind in development. This observation is further supported by the fact that the diagonal line C, which represents the average of the prerolandic effector cortex, is so much more nearly parallel with the line D, which is the average for the whole of the postrolandic receptor cortex.

Differently expressed, the proportion of postrolandic receptor cortex to prerolandic is more uniform throughout than is that of the parietal association area. As the latter forms a part of the postrolandic cortex it again suggests that it must be chiefly this part of the brain which is at fault in the defective.

Assuming that it really is the parietal association area which lags behind in development, what meaning—if any—is to be attached to the fact clinically?

It is stated that stereognosis and barognosis—the abilities respectively of recognizing objects by palpation, or of assessing different weights—both belong to a special form of body feeling (Tilney and Riley) and that the

cortical area specially concerned is usually described as occupying the upper portion of the intermediate postcentral area which is bounded below by the horizontal limb of the interparietal sulcus, and in front by the dorsal division of the ascending limb of this fissure. In other words, these two abilities are chiefly mediated by parietal cortex.

In the well-known Binet tests there are two—one for the fifth year and one for the ninth—designed to assess the stereognostic, or, more accurately, the barognostic, sense. That for the fifth year is useless for any purpose, as only two weights are employed, and thus whatever the defective does the chances are always even that he scores a success. That for the ninth year is free from such objection, but unfortunately many defectives never get so far as the nine-year tests. As this perception is, however, supposed to be mediated by parietal cortex, and as this present study suggests that it is precisely this area which is most undeveloped in defectives, it was clearly desirable to ascertain how far, if at all, clinical observations on abarognosis compared with the ill-developed parietal association area as measured after death and suggested by the pronounced microcephaly revealed by head measurement during life.

With this object the Binet records of 113 living defectives of sufficiently high grade to have attempted the nine-year-old weight test were examined, the assumption being that the clinical microcephaly might possibly be indicative of an imperfectly developed parietal lobe, in which case there should theoretically be an associated higher percentage of barognostic failures. The results were as follows :

Percentile of head size	Nos. of cases	Failed at test	Passes
Below zero percentile	23	20	3
0 to 10	38	26	12
10 „ 20	16	9	7
20 „ 30	10	7	3
30 „ 40	2	2	0
40 „ 50	6	6	0
50 „ 60	6	2	4
60 „ 70	2	1	1
70 „ 80	3	0	3
80 „ 90	2	2	0
90 „ 100	3	3	0
Above 100	2	2	0
	113	80	33

These results are shown in fig. 3, in which the percentages of those that fail and those that pass the test are plotted for each level of head size. There is a notably greater proportion of failures amongst the very small heads ;

for heads of more normal size the proportion of successes is somewhat greater, while among the abnormal macrocephalics the proportion of failures again rises.

Apart from any problem of cerebral localization, there would thus seem to be clinical support for the conclusion previously drawn that defectives have feebly developed parietal areas.

Two other problems presented by the investigation—namely, size of cuneus and ratio of corpus callosum to brain—may next be briefly discussed.

FIG. 3.—Diagram showing the percentages of cases that failed or passed the nine-year old Binet barognostic test and the association of failure or success with the percentile size of skull and brain.

The 80 failures are indicated by the continuous line, and the 33 passes by the interrupted line. The figures on the left are percentages, those at the bottom, percentiles.

The Cuneus.—As the cuneus contains within its area parts of the visuo-sensory and visuopsychic cortices it was thought that readings of its planimeter area might show some association—negative or positive—with other parts of the brain, or might even link up with mental differences between these defectives during life. The actual planimeter areas of the cuneus are shown for these cases in fig. 3 by cross-hatched portions drawn at the bottom of the columns for postrolandic cortex.

The cuneus, lying as it does on the medial or internal surface of the brain, has nothing to do, macroscopically, with the postrolandic cortex on the external surface, and is merely shown graphically in this way in order to avoid a multiplication of unnecessary diagrams. But even thus one point emerges. The line A shows the average area of the cuneus in the 29 cases in which it was possible to obtain it (Cases 2 and 19 being unobtainable). It will be observed that cuneus development (line A) does not keep pace with parietal (line B). A permissible neurological inference is, therefore, that as the parietal association area is not being properly fed by visual stimuli, increments therein are more due to somæsthetic or acoustic stimuli.

Assuming the correctness of these inferences—though there may possibly be other explanations not so obvious—they would seem to suggest that defectives, as a class, are more dependent on general somæsthetic impulses from within their own bodies than they are on exteroceptive stimuli from without. They further suggest that these disabilities are structurally present before birth, and if, therefore, these important sensory or receptor stimuli are so lacking, all that can be done after birth is to make the most, therapeutically, of the little the defectives have got.

The Corpus Callosum.—The corpus callosum is composed of the medullated axons of commissural neurones with their cell-bodies in the grey cerebral cortex. As the corpus callosum is the only commissure developed in connexion with the neopallial cerebral hemispheres it follows that, theoretically, there should be some association of size of corpus callosum with neopallial cellular richness. As defectives are presumably deficient in some way or other in such wealth of properly functioning cortical cells, measurable variations in size of their corpora callosa might reasonably be expected. This investigation shows that they do, but it failed to demonstrate any appreciable correlation between size of corpus callosum and size of brain. This negative result is not, however, surprising, for size of brain in defectives varies with many factors other than cortical richness of functioning neurones, and it is this last fact alone which is, or should be, chiefly responsible for callosal structure and size.

CONCLUSIONS

From this series of 31 defective brains two facts definitely emerge: One, that the defective brain is 20 per cent. less than that of the lowest normal, and that even within this low standard the range of variation is considerable; two, that there is a measurable disproportion between the main pre- and postrolandic cortical constituents, and that this disproportion is particularly noticeable in the parietal region.

Fig. 2 shows this inequality of the pre- and postrolandic areas, and particularly the fact that it is the parietal lobe which most lags behind in a development proportionate to the rest. In view of the anatomical position of that lobe relative to the cuneus, superior temporal lobe and postcentral

gyrus—that is, to the cortical areas where are first received visual, acoustic and somæsthetic impulses—it seems impossible to doubt that when the parietal lobe is underdeveloped, as in these defectives, the ‘reception, association and co-ordination of impressions derived from the organs of special sense’ must be so impaired as to result in serious mental deficiency. There is, therefore, an adequate neurological explanation of the mental status of the present series of cases.

Bolton’s insistence, in his *Henderson Trust Lectures* of 1933, on the importance of an adequately developed and well-proportioned prerolandic effector area (frontal lobe) in all normal mental activities fully bears out the present investigation and particularly the facts elicited in fig. 2, where the remarkable irregularities between the frontal effector area and the rest of the brain are readily seen.

In persons with brains so irregularly developed and so lacking in the proper proportions of receptor and effector cortices—that is, in defectives—there must necessarily be a diminished sensibility to, and perception of, the external world around them. They thus have to rely far too much on purely bodily stimuli from within, with results which profoundly alter their mentality and may even make them react to their human environment in an abnormal or asocial manner, and this defectives—especially the lower grade ones—most certainly do.

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APPENDIX I

DETAILS OF THE CASES FORMING THE SUBJECTS OF THE INVESTIGATION ARRANGED IN ORDER OF CHRONOLOGICAL AGE

No. 1. Male, aged 4.8 years. Idiot. Unable to do the simplest test. Wet and dirty (0 marks). Unable to walk (0 marks). Speechless (0 marks). No educational attainments (0 marks). Brain capacity percentile at approximately the two-year-old level.

No. 2. Male, aged 8.0 years. Idiot. Impossible to test. Wet and dirty (0 marks). Unable to walk (0 marks). Speechless (0 marks). No educational attainments (0 marks). Brain capacity percentile not known. Complicated by a vascular pseudoporencephaly.

No. 3. Male, aged 8.3 years. Idiot. Physiological marks 0. Umbilical hernia. Rickets. Enuresis. Extreme hypotonia.

No. 4. Male, aged 8.11 years. Idiot. Physiological marks 0.

No. 5. Female, aged 9.7 years. Mongolian imbecile. Assessed mental age during life less than three years. Clean habits. Walked well. Limited speech. No educational attainments. Physiological marks 40. Brain capacity percentile —45.

No. 6. Male, aged 11.10 years. Imbecile. Brain capacity percentile 4. Binet mental age 4.2 years, Porteus 4 years. Combined I.Q. 37. Physiological marks 40.

No. 7. Male, aged 12.3 years. Mongolian idiot. Brain capacity percentile —9. Mental age less than two years. Wet and dirty. Speechless. No educational attainments. Physiological marks 15.

No. 8. Female, aged 12.3 years. Imbecile. Brain capacity percentile —19. Merrill-Palmer mental age 6.1 years. Physiological marks 40.

No. 9. Female, aged 12.1 years. Idiot. Physiological marks 15. Paralysed and crippled.

No. 10. Female, aged 13.3 years. Imbecile. Brain capacity percentile 5. Merrill-Palmer mental age 3.8 years. Physiological marks 30. Irritable, epileptic, and with phenomena resembling cerebral diplegia.

No. 11. Male, aged 14.0 years. Idiot. Brain capacity percentile 15. Physiological marks 15.

No. 12. Male, aged 15.4 years. Mongolian imbecile. Merrill-Palmer mental age 5.10 years. Physiological marks 25.

No. 13. Female, aged 15.8 years. Idiot. Wet and dirty. Unable to walk. Speechless. No educational attainments. Physiological marks 0.

No. 14. Female, aged 16.10 years. Mongolian idiot. Brain capacity percentile —8. Physiological marks 20.

No. 15. Female, aged 17.7 years. Low grade feeble-minded. Brain capacity percentile 16. Binet mental age 6.10 years, Porteus 11.6 years, and combined I.Q. 57.5. Clean habits. Limited powers of walking. Some educational attainments. Physiological marks 90.

No. 16. Female, aged 17.9 years. Idiot. Brain capacity percentile —7. Physiological marks 20. Paralysis.

No. 17. Female, aged 18.3 years. Imbecile. Brain capacity percentile 19. Merrill-Palmer mental age 2.8 years. Physiological marks 30. Blind, and suffered from fits.

No. 18. Female, aged 19.0 years. Idiot. Brain capacity percentile —1. Merrill-Palmer mental age 2.9 years. Physiological marks 15. Blind and very restless.

No. 19. Male, aged 20.6 years. Imbecile. Physiological marks 40. A pathological case of ventricular hydrocephalus.

No. 20. Male, aged 21.0 years. Imbecile. Physiological marks 40. Brain capacity percentile 4.

No. 21. Male, aged 21.2 years. Idiot. Physiological marks 10.

No. 22. Male, aged 21.5 years. Idiot. Brain capacity percentile 43. Physiological marks 10. A pathological case of tuberous sclerosis.

No. 23. Female, aged 22.6 years. Brain capacity percentile 7. Physiological marks 70.

No. 24. Male, aged 23.8 years. Idiot. Merrill-Palmer mental age 1.8 years. Physiological marks 5. Dull and vacant expression, with blindness of left eye.

No. 25. Female, aged 24.0 years. Imbecile. Brain capacity percentile 11. Merrill-Palmer mental age 6.6 years. Speechless. No educational attainments. Physiological marks 25.

No. 26. Female, aged 24.5 years. Imbecile. Merrill-Palmer mental age 6.6 years. Physiological marks 40.

No. 27. Female, aged 24.9 years. Feeble-minded. Physiological marks 85. Encephalitis lethargica.

No. 28. Male, aged 27.3 years. Idiot. Excitable. Physiological marks 25.

No. 29. Female, aged 30.7 years. Imbecile, high grade. Physiological marks 60.

No. 30. Female, aged 30.10 years. Imbecile. Brain capacity percentile —13. Merrill-Palmer mental age 4.0 years. Physiological marks 30.

No. 31. Female, aged 35.11 years. Imbecile. Merrill-Palmer mental age 6.6 years, Binet 6.10 years, Porteus 5 years, and combined I.Q. 37.

Cases 2, 19 and 22 were associated with definite and easily recognizable pathological conditions. These respectively were pseudoporencephaly of vascular origin, ventricular hydrocephalus, and tuberous sclerosis.

The physiological system of markings referred to was devised by the author for the early assessment of oligophrenia and is on the following arbitrary scale :

	Minimum	Intermediate	Maximum
<i>Habits</i>			
Wet and dirty	0		
Clean			10
<i>Walking</i>			
None	0		
Limited or impaired		5	
Good			15
<i>Speech</i>			
None	0		
Limited		15	
Good			30
<i>Education (reading, writing, counting)</i>			
None	0		
Limited		15	
Fair			45
	0	35	100

The uses of the system, as well as its practical value, are easily assessed from a study of the cases themselves.

As regards the brain, the technical procedure included injection through the orbits, as soon after death as possible, of a 10 per cent. solution of formol saline, with removal of the brain at the earliest opportunity. After further hardening, one cerebral hemisphere was specially preserved and mounted for macroscopic study, including dioptrographic tracings, and the other retained for microscopic investigation.

APPENDIX II

PLANIMETER AREAS OF BRAIN IN SQUARE INCHES

No. of case	Parietal	Occipitotemporal	Frontal	Total brain
1	3.44	9.53	6.39	19.38
2	1.95	6.87	6.87	15.69
3	4.14	7.83	7.36	19.38
4	3.50	8.30	7.91	19.69
5	2.93	6.20	4.54	13.67
6	3.79	6.50	7.03	17.35
7	4.44	7.46	6.18	18.06
8	2.47	6.30	5.86	14.63
9	3.37	8.15	7.91	19.40
10	4.05	7.77	7.90	19.72
11	4.60	8.82	6.91	20.31
12	3.54	7.49	6.70	17.75
13	3.22	7.06	5.30	15.58

PLANIMETER AREAS OF BRAIN IN SQUARE INCHES—*Continued*

No. of case	Parietal	Occipitotemporal	Frontal	Total brain
14	3.33	7.17	5.84	16.31
15	5.04	7.33	8.25	20.62
16	2.98	6.07	5.92	14.96
17	3.16	6.24	6.39	15.79
18	3.33	8.01	7.14	18.46
19	3.00	11.63	8.06	22.69
20	3.38	7.62	6.52	17.51
21	3.10	7.73	6.11	16.91
22	4.57	8.87	8.54	22.07
23	3.83	7.09	6.62	17.55
24	4.82	8.97	8.18	21.93
25	4.93	9.09	7.80	21.82
26	3.08	6.80	5.43	15.31
27	3.90	7.80	7.02	18.70
28	3.37	9.45	8.16	20.97
29	4.36	8.12	7.05	19.51
30	3.67	7.28	5.65	16.60
31	3.57	6.62	5.58	15.76

NOTE.—For check purposes the total brain area was recorded separately from the three which make it up ; there is, therefore, an occasional slight difference of not more than 0.03.