Focal and General Unilateral Brain Atrophy: Effects upon the Corpus Callosum

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WITH PLATES

FOCAL AND GENERAL UNILATERAL BRAIN ATROPHY: EFFECTS UPON THE CORPUS CALLOSUM.¹

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(With Plates 2-6.)

A FAVOURABLE opportunity for pathological investigation of the human corpus callosum is afforded by cases of infantile hemiplegia. Institutions for epileptics not infrequently show marked examples, perhaps because whatever caused the hemiplegia (prenatal lesions, cortical hemorrhagic encephalitis, perhaps poliomyelitic or the primary acute type described by Strümpell, or possibly other still more obscure causes) at the same time provoked the epilepsy.

The object of this study was to utilise epileptic material in the interest of the knowledge of the corpus callosum. This material was derived from the Monson State Hospital, and analysed anatomically and by means of photographs, with the aid of the Massachusetts State Board of Insanity. Its most striking result dealing with (a) two cases of relatively complete unilateral cerebral atrophy, and (b) three cases of sharply defined focal unilateral atrophy, is the demonstration that

(1) Complete unilateral atrophy results in a general diffuse thinning of the corpus callosum, and

¹ Read at meeting to review twenty years' work at the Monson State Hospital, Palmer, Massachusetts, May 4, 1916.
(2) Partial unilateral atrophy entails a partial thinning of the corpus callosum in a region precisely opposite to the location of the cerebral lesion. Accordingly, one may suppose that whatever the ultimate origins or terminations of the corpus callosum fibres, they run in an approximately transverse direction while within the corpus callosum. Since our knowledge of the exact location of the cells of origin and the terminal arborisations of the corpus callosum fibres is at the present time very slight, it is clear that such a study as this may finally add to our knowledge of a very important problem—the physiology of the commissural system. The results of this study incline the writer to agree with von Monakow and others, that the parts of the callosum unite symmetrical areas of the cortex, rather than with Cajal, who believes that the corpus callosum is much more complex, and unites dissimilar areas. It may be that the corpus callosum does, by means of collaterals, effectively unite dissimilar areas of the hemispheres.

**Material.**

In 1913 the Monson State Hospital had its collection of formalised brains photographed as a permanent record, providing a possibility for detailed study from the anthropological-morphological point of view. At that time there were fifty specimens available. Twelve photographs were made of the surfaces of each brain; six with the pial membrane intact, including top, base, two lateral surfaces, and two mesial surfaces. After removal of the pia, the photographs were duplicated. In addition to this, in twenty-five cases the brain was cut in frontal sections, one centimetre or more in thickness, according to the proportions of the brain, and the posterior surface of each of these sections was photographed. Altogether, this makes a detailed, permanent record of a series of epileptic brains which is at the disposal of any one who may wish to make an investigation of the gross morphology at any time. Later, a much more detailed piece of work has been begun, in which sections from twenty-nine areas of the cortex of twenty-five cases have been prepared for histological examination, together with sections from cerebellum, medulla, and high cord. From the entire number five specimens have been chosen for whole-brain sections. These five are those which are the subject for special consideration in this paper.
GENERAL UNILATERAL BRAIN ATROPHY

Each one of these cases presents a gross lesion mainly of one hemisphere. In detail they are as follows:

CASE DESCRIPTIONS.

Case I. (11-26).—Female, 19 years of age at death; duration of epilepsy, seventeen years. Mental classification, moron. On entrance, physical examination showed complete paralysis of left arm and hand, with contraction of wrist and fingers. The left knee was bent, but could be straightened. Left knee-jerks much increased and Babinski reflex positive.

The history of the epilepsy is, that at two years there was an illness called meningitis, at which time the patient had convulsions. Previously she had been as bright as the ordinary normal child of her age. Following this there were no further epileptic manifestations until the age of 6 years, when she had the measles. During this illness there was one distinct convulsion, and later she began to have staring spells, and slight convulsions which grew progressively more severe as she grew older. On admission to the hospital, four to ten attacks were occurring daily. They were said to be preceded by tachycardia. During the hospital residence of five years, there was a continuance of convulsions, varying in frequency and severity, accompanied by progressive mental enfeeblement. Death was due to status epilepticus.

The brain weighed 1,260 gm. It showed an extreme degree of asymmetry, due to much tissue destruction on the right side, involving the greater part of the central convolutions. Of this area, only the upper portion of the post-central gyrus, and the posterior half of the para-central lobule remain at all in a normal condition. Further, there is complete destruction of the supramarginal gyrus, and a marked degeneration of the frontal operculum. This corresponds exactly with the paralysis present, and accords roughly, perhaps, with the degree of mental deterioration. The left hemisphere shows nothing remarkable in the gross. On palpation the cortex is soft and springy, suggesting a considerable degree of oedema, especially over the central convolutions.

On the mesial surface the most notable finding is the marked narrowing of the corpus callosum, for a certain definite segment of the horizontal portion, exactly corresponding to the cortical destruction in the right hemisphere; this includes the posterior
two-thirds of the callosal body. In this part the width is only 2 mm., while in the anterior third it is 5 mm. in width. This apparently represents the inter-hemispheric fibres, arising from the area of the cortex destroyed in the right hemisphere.

Case II. (09-14).—Female, 22 years of age at death; duration of epilepsy, fourteen years. Mental classification, imbecile. Physical examination at the time of entrance to the hospital showed a left hemiparesis. Patient is able to use her hand in grasping objects, but cannot execute fine movements. There was a dorso-lumbar scoliosis, and some evidence of a left-sided facial paralysis. Reflexes increased. Sensory disturbance more marked in upper than in the lower extremities. An indefinite history suggests anterior poliomyelitis in childhood. During the six years of hospital residence the convulsions varied from four to forty-five monthly.

The brain, which weighed 970 gm., is fairly developed, but extremely simple in configuration. On inspection a marked degree of atrophy of the main part of the post-central gyrus is found, involving also the entire inferior parietal lobule, and all except the most anterior marginal portion of the superior parietal lobule. Frontal section through this area shows almost complete loss of substance, the wall of the hemisphere being reduced to the thinnest possible layer of tissue. The findings apparently correspond with the clinical signs. The motor disability probably depended more directly upon the sensory disturbance, as evidenced by the post-central gyrus degeneration, than upon absolute destruction of motor fibres. The loss of cortex in the inferior parietal lobule would result in loss of muscle sense, as shown by inability to execute fine movements with the corresponding hand, while retaining the ability to use it in a general way, more or less reflex in nature, as in merely grasping objects. Cause of death, status epilepticus.

On the mesial surface one may distinguish a sharply localised area of thinning of the corpus callosum, limited to its most posterior portion, and mainly affecting a very narrow segment of the body adjoining the splenium; the latter is also somewhat thinned. Thus one may easily suppose that this narrowed portion of the corpus callosum corresponds to the fibres from the atrophic area in the right hemisphere, crossing to the opposite side.
Fig. 1.—Top of Case 1, showing asymmetry due to destruction of tissue in central region.

Fig. 2.—Mesial surface of Case 1, showing destruction of tissue in the region of the operculum, and the corresponding thinning of the posterior two-thirds of the body of the corpus callosum.
Fig. 3.—Top of Case 2, showing atrophy of the post-Rolandic region, and slight consequent asymmetry.

Fig. 4.—Mesial surface of Case 2, showing thinning of the corpus callosum just anterior to the splenium.
Fig. 5.—Top of Case 3, showing marked asymmetry, due to lack of development of the left hemisphere.

Fig. 6.—Mesial surface of Case 3, showing remarkable thinness of the entire corpus callosum, corresponding to the size of the left cerebral hemisphere.
Fig. 7.—Top of Case 4, showing marked asymmetry due to lack of development and retrograde changes in the left hemisphere.

Fig. 8.—Mesial surface of Case 4, left hemisphere, showing atrophy of hemisphere, and corresponding thinness of the entire corpus callosum.
Fig. 9.—Basal surface of Case 5, showing areas of softening in the left temporal lobe, and on the left orbital surface.

Fig. 10.—Mesial surface of Case 5, left hemisphere, showing marked thinning of the middle third of the body and subependymal portion of the corpus callosum, corresponding to the softened areas in the temporal lobe and on the orbital surface.
Case III. (11-10).—Female, 18 years of age at death; duration of epilepsy, thirteen years. Mental classification, idiot. Physical examination on entrance to the hospital showed a right hemiplegia; further details are lacking, and there is no early history, as there were no known relatives nor friends, the patient having always been, as far as known, an institution inmate. Cause of death recorded as epilepsy, probably not status epilepticus.

The brain weight was 740 gm. The convolutions vary notably in width and lack a rounded contour. The pattern is rather a simple one throughout. There is marked asymmetry, due to the very small size of the left hemisphere. There is no focal tissue degeneration, but a general lack of development, affecting mainly the post-central half of the hemisphere. Corresponding to this very small hemisphere, there is also a very thin corpus callosum, and this is true of the entire structure, including knee, body, and splenium; the rostrum is least affected.

Case IV. (11-7).—Male, 30 years of age at death; duration of epilepsy, twenty-eight years. Mental classification, imbecile. On entrance to the hospital thirteen years previous to death, the physical examination showed a triplegia, involving the entire right side and the left lower extremity. The brief history states that the onset of paralysis and epilepsy was in childhood, and there was complete inability to walk. The patient was also entirely devoid of intelligence. Death was due to epilepsy.

The brain weight was 965 gm. The right hemisphere shows a fair degree of development; the convolutions are fairly broad, but not well rounded. The pattern is simple. The left hemisphere is very much smaller than the right, involving all lobes. The convolutions are narrow, increased in consistence, and on section the cortex forms merely a thin shell, the thinning being particularly marked in the temporal and post-central lobes. The corpus callosum in this case shows similar appearances to those of the preceding one, but somewhat more marked in degree, with extreme thinning in all parts, corresponding to the general cortical degeneration of the entire left hemisphere.

Case V. (09-23).—Male, 18 years old at death; duration of epilepsy, twelve years. Mental classification, imbecile. Physical examination on admission to the hospital showed partial paralysis of the right side, with some difference in the degree of development of the two sides. Muscular co-ordination is said to have been
fair; knee jerks increased. Unfortunately this brain was sectioned before the photographic work was done, so it is impossible to reproduce it, but a reconstruction from whole brain sections shows an extensive degeneration of the entire operculum, and the underlying first temporal gyrus, resulting in multilocular cyst formation, and an accompanying reduction in size of the entire left hemisphere. By the same means it is found that the corpus callosum is thinned out both in location and degree corresponding to the area of tissue destroyed in the left hemisphere.

To complete the picture of focal thinning of the corpus callosum, a case (15-63) is included which does not represent epilepsy, but criminal insanity. By the photograph it may be seen that with a great degree of softening of the cortex over the left temporal lobe and the greater part of the left orbital surface, the callosum is correspondingly thinned in the middle third of the horizontal portion, with the greatest degree in the posterior part of this area, and the rostrum also is markedly decreased in thickness.

Discussion.

The corpus callosum is made up of nerve fibres passing from one hemisphere to the other, in a transverse direction. The fibres of the posterior extremity (the splenium) are seen to connect the occipital lobes; just anterior to that, or the most posterior part of the horizontal portion, arise the parietal lobes; just anterior to this, the fibres have their origin in the temporal cortex; the middle part represents the central convolutions; the anterior portion, including the knee, pass between the convexity of the frontal lobes, and the subgenual part, or rostrum, is made up of fibres having their origin in the orbital surfaces. It has been said that this portion represents fibres only from the gyri recti, but it is plainly demonstrated by case 15-63 that this is not a fact, for here the gyrus rectus of both sides is only partly involved in the degeneration, while the remainder of the left orbital surface is very largely destroyed, with corresponding thinning of the rostrum.

Concerning the question of epilepsy in connection with these findings, it is interesting to review Strümpell's description of the development of infantile cerebral paralysis. He says that children of one and a half years and over, who previously have been both physically and mentally perfectly well, suddenly develop fever
and headache. A few hours, or oftentimes a number of days thereafter, cerebral symptoms develop in the form of convulsions, unconsciousness, or other similar phenomena. After a varying number of days the child becomes perfectly well again, except for a persistent hemiplegia, varying in extent. The clinical picture points to an affection of a focus in the cerebral cortex, suggested by the paralysis with convulsions; the former is often confined to one side, and the epilepsy persists, and may be accompanied by athetoid movements. According to Oppenheim, the extent of the encephalitic focus may vary between very wide limits; it may be so small as to be recognised only with the microscope, or, in the case of infantile encephalitis, an entire hemisphere may be involved.

The onset of the disease is very indefinite in all of the five cases in this series; only in one, 11-26, is there a history of sufficient detail to indicate any febrile disturbance. The ages at onset, however, which vary between two and eight years, suggest very strongly that a focal or general infection of the brain cortex may have been the starting-point, resulting in a corresponding degree of paralysis, and causing epilepsy. Gowers says, "Of all the regional diseases of the brain in man, lesions of the convolutions stand almost alone as a cause of convulsions." The lesions of the convolutions in the cases considered here are quite apparent, and in these, as in other specimens, may find a definite indicator in the corpus callosum, when there is sufficient involvement to include the commissural fibres.

**Summary.**

Five cases are considered, all of which were hemiplegic, epileptic, and mentally defective. The onset in these cases was between the second and the eighth year. Examination of the brains in every case shows a lesion of one hemisphere; focal in three cases, and involving the entire hemisphere in two. The location and extent of the cortical defect is indicated by an exactly corresponding thinness of the corpus callosum.

**Literature.**

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