ON A CASE OF CONGENITAL PORENCEPHALUS
IN WHICH THE PORENCEPHALIC AREA CORRESPONDED TO THE
AREA OF DISTRIBUTION OF THE LEFT MIDDLE CEREBRAL ARTERY.

BY

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INTRODUCTORY.

WHILE the condition of porencephaly—either congenital or acquired—is not particularly rare, it is remarkable how rarely opportunity presents itself for the full and satisfactory study of any individual case. Most often there are pronounced psychical disturbances so that it is impossible to obtain from the patient any history of the development of his condition ; while further, it most often happens that such cases enter an asylum and there the little time that the asylum physician can afford to any individual case is against any adequate investigation of the history of the case prior to entry ; while, if the patient has shown no marked psychical disturbance, the chances are much against obtaining an autopsy upon a private patient, and if this be obtained, either the examination is made too long after death for satisfactory histological examination of the nervous system, or intercurrent disease masks the primary lesions in the various tracts.

It thus happens that, to my knowledge, there does not exist any complete study of a case of porencephaly by any

4 Congenital Porencephalus.

English speaking observer. There are, it is true, important papers by Wigglesworth, Ross, and others,* who have brought to light valuable facts, but not one of these observers has been in the position to work out all the details of his cases. Mott's admirable papers upon Cerebral Hemiatrophy [1] and the Degeneration of the Neurone [2] afford much information, which has been of material aid to me in the following pages, but porencephalus is merely treated incidentally. The same may be said of the article by James S. Collier and Risien Russell upon Cerebral Diplegia.[3] It is, perhaps, only right that I should here state that this paper was almost completed and my results obtained in 1899 before either of the two last papers were published, and that thus to a very large extent the observations of these observers did not so much direct my work as afford confirmation of facts already noted by me, and I may add, announced in a preliminary communication before the Montreal Medico-Chirurgical Society in December, 1899.

What is true of publication in English is also to a very large extent true as regards French and German literature With the exception of the admirable monographs by Andry,[4] Schattenberg [5] and Otto,[6] the continental literature consists of but partial studies and descriptions, although here mention should be made of Kundrat's [7] valuable collection and criticism of a large number of cases.

In the following pages I have not so much attempted to review or base my work upon previous literature, as I have endeavored to describe adequately a thorough study of one individual case and the results obtained therefrom. Such a full study has seemed to me desirable from several considerations. Cases of congenital porencephalus in man afford us the fullest knowledge of the course of various tracts and the relationships between various centres — afford us in short information equivalent to that obtained in the lower animals only by means of experiments : these considerations alone render it at least worth while to record here in detail the results of a prolonged study of a case of

* Wigglesworth — Brain, XX., 1897, p. 200; Clinch—Journal of Mental Science, XLV., 1899, p. 246; Norman and Fraser—Journal of Mental Science. XL., 1894, 9, 649; Ross, J.—Brain, V., 1882-3, p. 343; Warner and Beech—Brain, II., 1880, p. 576; Clark—Journal of Mental Science, XXV., 1879, p. 329; Anderson—Trans. Royal Soc., Edinburgh, 1898, II., p. 326. There is yet Abercrombie, frequently mentioned by other English writers, but for whose paper I have been unable to obtain the exact reference.

congenital porencephaly in which the loss of substance affected a very large portion of the motor area on the left side without inducing any well-marked psychic disturbance ; in which again the cause of the atrophy and loss of nerve substance is clearly seen to be associated with obliteration of a certain extent of the middle cerebral artery and its branches, in which the autopsy was performed within a few hours after death, so that the brain and cord were obtained in an excellent condition of preservation, and in which, lastly, I was fortunate enough to be able to obtain a singularly full and interesting history of the case from the relatives.

The case here recorded came under my notice at a post-mortem examination performed by Dr. Adami upon a woman aged seventy-six who had died of pneumonia in the Royal Victoria Hospital, Montreal. The patient was brought in in a moribund condition and was dead in twenty-four hours. Thus no clinical notes could be taken, but from her brother and sister-in-law, whom I saw later, I obtained the following history :—

HISTORY OF CASE.

The parents of Mrs. X. were both healthy and wealthy, and the patient was the sixth born in a family of eight. While the mother was pregnant her health was not in a very satisfactory condition, but it is not known that her ailments were other than those common to the pregnant state. It is, however, remembered in the family that at about the seventh month she received a severe shock to her nervous system from the upsetting of a lamp and that the following day signs of abortion presented themselves, but with careful treatment she was able to carry the child to full term.

At birth the child seemed to be in every way normal, and it was not until a week after it was born that the wet nurse perceived a stiffness in the right arm and fingers. This was all that was noticed at the time. Yet as the child grew up it was seen that its intellectual capacity was poor ; she did not walk until she was three years old, and it was a year later before she was able to talk. She was not put to school like her sisters and brothers, but the mother took her education in hand and with great perseverence taught her to read and spell and to write with the left hand. It was at this period

that her general backwardness, mentally, was most marked,
but under the guidance of the mother, who was able
to devote much of her time to her, she was able to
gain a fairly good foundation in elementary education. At
the age of thirteen she was sent to school, but as her parents
found the excitement too much for her she was allowed to
remain at home and finish her education there. As she grew
up to womanhood she seemed to be in all respects a woman
with an average amount of mental and physical capabilities.
Her friends state that she never suffered from epilepsy or fits
of any kind at any time.

The right arm and hand were seldom used, and when
she was occupied with household duties she employed the left,
the right, although powerful, being simply brought into use as
a support; for instance, when washing and cleaning dishes
she would rub and clean with her left hand and support the
dish with the elbow and hand of the right side, and for all
purposes the left hand was as good as any normal right hand;
in fact, she was an extremely active needlewoman.

The right foot was inclined to drag a little, but it did not
seem to hinder her progress to any extent, for she was
fond of walking and used to take long walks out into the
country. In stature she was not very upright, but had a
rather cramped attitude, and was inclined to stoop (kyphosis),
the right shoulder being lower and more forward than the
left. Her vision was poor in the left eye.

She was married twice, but had no family by either
marriage.

When she attained the age of from 56 to 60 her mental
faculties began to weaken rapidly. By this time she had lost
her second husband and she seemed to worry much over her
loneliness. She became very religious, obstinate and eccen-
tric, living by preference more or less a hermit's existence
apart from her friends. At this period she suffered from
illusions and delusions, imagining that her relations ill-used
her and desired to do her bodily harm. These spells were
transitory, and when she was feeling better she would return
to her former way of living.

It was in one of these paroxysmal delusionary states that
she was seized with pneumonia. At the time she was living
alone in her home, looking after her own household affairs
unattended by any one, and existing in a rather miserable

and dirty state, this not on account of poverty, for she was very comfortably settled, but on account of the above mentioned habit of mind. While thus laid down by sickness a neighbour happened to call in, and recognizing how seriously ill she was, called the ambulance and saw that she was removed to the hospital where, as above stated, she died within 24 hours.

A distinct family history of neurasthenia and of arterio-sclerosis was obtained. Thus, of the seven other members of the family, two brothers and one sister died of apoplexy, and the brother to whom I am indebted for this history had himself a stroke of apoplexy two years ago and is at the present time suffering from its effects ; he is extremely nervous, sleepy and mentally somewhat enfeebled.

POST-MORTEM EXAMINATION.

Mrs. X., æt. 76, admitted under Dr. C. F. Martin ; autopsy 2 hours after death, performed by Dr. Adami.

The body was that of a spare old woman, below normal height, eyes sunken, left pupil distinctly smaller than right, the right shoulder lower and smaller than the left ; muscles of the right upper extremity smaller than those of the left and bones shorter. The right hand claw-like, the flexor muscles atrophied, the thumb turned inwards, hyper-extension of the first interphalangeal joints of the second and third fingers with slight flexion of the terminal joints (Fig. 1). The right chest also was smaller than the left, and the muscles of the right lower extremity smaller, although there was no pronounced shortening of this leg.

Measurements : (D.A.S.)

Right leg, from ant. sup. spine to ext. malleolus, 28 in.
Left leg, " " " " 29 in.
Humeri : Right, 9¼ in.
Left, 10½ in.
Ulnæ : Right, 7½ in.
Left, 8½ in.
Circumference of leg (3 in. below gluteal fold) :
Right, 10 in.
Left, 12 in.

Head : The skull cap was very thin and easily cut through, the dura slightly adherent. The longitudinal sinus was normal. There was a fair amount of fluid beneath the dura and round the brain which on the left side appeared shrunken and markedly smaller than on the right. Upon removing the dura a remarkable and extensive condition of porencephaly was recognisable on the left side. The area of loss of substance was of a sausage-like shape, extending along the Sylvian fissure from the mid-frontal area backwards well into the temporo-

occipital region and involving evidently the centres of speech, face and upper extremity. Here the brain matter was completely wanting and was replaced by a membrane distended with clear fluid. The destruction of tissue leading to the cavity formation did not seem to be so great as it really was until the lobes were separated from each other, then the considerable loss of substance which had taken place was clearly appreciated.

Weight of brain, 930 grm. (as against normal for female, 1235).

Spinal Cord : The cord was removed down to the end of the dorsal region and upon section presented, especially in the cervical region, extensive atrophy of the white matter on the right side.

FIG. 1.

Thorax : The pectorals on the right appeared to be paler and flabbier than on the left ; the intercostal muscles and ribs were also smaller on the right side. The sternal cartilages were somewhat ossified. There were extensive old thready adhesions all over the right lung, and a condition of grey hepatisation affecting the whole of the upper lobe which on section yielded abundant diplococci ; there was a condition of splenisation affecting the left lower lobe rather more than the right lower lobe. The heart was senile with considerable fatty infiltration ; the aorta showed atheroma with a tendency to ulceration, more especially in the lower third. As is usual in cases of pneumonia, there were firm, well-formed blood clots forming casts in the pulmonary arteries and their branches and in the heart cavities.

The *abdominal viscera* presented nothing worthy of note ; the internal genitals were senile and the uterus contained two small sub-

mucous polypi, the larger, situated near the fundus, being 2 cm. in diameter.

Brain :　The brain having been carefully removed was given into my charge for hardening and further study.

GROSS ANATOMY OF THE BRAIN.

The difference in size and development between the two hemispheres was very marked ; upon looking at the external surface of the left hemisphere, along the longitudinal fissure, the mesial border of the frontal, parietal and occipital regions of the right hemisphere could be seen to a considerable extent.　The right hemisphere weighed 560 grammes, the left 302 grammes, but contrariwise the right half of the cerebellum was markedly smaller than the left.

The Right Hemisphere :　The right hemisphere was found normal and exceptionally rich in convolutions which were large and well formed and along with the fissures followed a typical course.　For the size and age of the woman this hemisphere was singularly well developed, and it was distinctly above the average in weight for a woman of her stature.

The Left Hemisphere :　The left hemisphere had a strikingly abnormal aspect ; the convolutions of the mesial aspect and of the orbital surface appeared to be intact and well rounded off, but on the whole they were very poorly developed, though not simplified, their general outlines being distinctly smaller than the corresponding convolutions of the right side.

(*a*) *Fissures :*　Mesial Aspect :　The fissures of the mesial aspect could be clearly made out and followed a typical course, with the exception of the parieto-occipital which was exceptionally deep mesially and extended externally for more than an inch on the external aspect of the hemisphere.

External Aspect :　The horizontal, ascending and posterior branches of the fissure of Sylvius were absent, a large cavity, the remains of a cyst, occupying this region, the cavity being formed by the destruction of a considerable portion of the convolutions of this area.　Its walls and floor were collapsed and puckered, drawing the surrounding convolutions in towards its centre so as to obscure the normal arrangement of the parts.　On account of this

puckering the fissure of Rolando was very hard to make out
but by tracing up the calloso-marginal sulcus on the mesial
aspect I found it possible to locate accurately what
remained of the fissure.   This was found to lie further back
posteriorly than normal, and its course was extremely short,
not more than a quarter of the normal length.

The precentral fissure and the inferior part of the inter-
parietal were absent, but in the frontal area, the superior frontal
and the anterior half of the inferior frontal fissures could
easily be made out.   In the temporal region the 2nd tem-
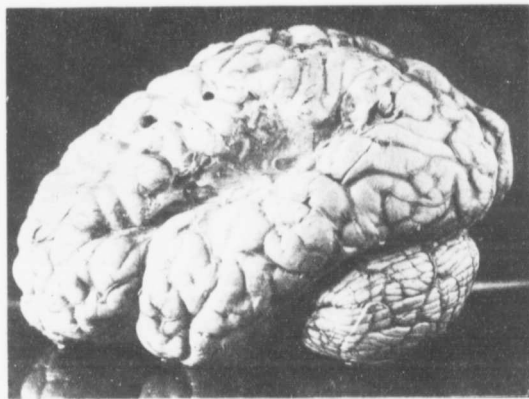poral fissure and the anterior third of the 1st temporal (or

FIG. II.

parallel) fissure could be seen.   In the occipital region all
the fissures were present.

(*b*) *Convolutions :*   External Aspect :   The cerebral
substance of the third frontal convolution was entirely absent
save for a very small part of its anterior and inferior end
which could be made out on the under surface of the anterior
lobe external to the posterior orbital convolution.   The
posterior third of the middle frontal convolution had com-
pletely disappeared as had also the lower two-thirds of the
precentral and post-central convolutions, the supra-marginal

and the angular. The first temporo-sphenoidal and the posterior half of the second temporo-sphenoidal were wanting, but the inferior temporo-sphenoidal and the superior middle and inferior occipital as again the superior frontal convolutions were still to be made out.

Under Surface : The hippocampal convolution with its uncus, the lingual and the fusiform were present, but like the others, were poorly developed, and tilted upwards and outwards so that the fusiform and the lingual presented to a slight extent on the external surface of the hemisphere and gave the impression that three temporo-sphenoidal convolutions were present.

The superior, and part of the middle frontal, the remains of the precentral and the post-central, as again the superior parietal were drawn downwards and inwards round the edge of the cavity. On account of this the destruction of cortical tissue seemed to be much less than was really the case.

Coming now to the under surface of the anterior lobe of the left side, here, taking together the convolutions and the fissures, the sulcus olfactorius and the triradiatus, the anterior fissure of Reil, the internal, anterior and posterior orbital convolutions could be clearly defined, as could also the anterior inferior part of the inferior frontal and that portion of the Island of Reil which lies anterior to the sulcus centralis insulæ and is called the precentral lobe, its post-central lobe, which normally lies behind, being absent. The former of these corresponds to or may be regarded as a part of the frontal lobe, the latter, to the parietal and temporal lobes. The absent post-central lobule of the Island of Reil normally, I should add, lies external to the lenticular nucleus. The anterior and posterior orbital and the inferior end of the inferior frontal convolutions were displaced outwards and appeared to a certain extent on the external surface of the hemisphere.

(c) *The Porencephalic Cavity :* On dissecting off the roof of the cyst which consisted of the arachnoid, it was found that the floor and the sides were formed of a thickened membranous layer of pia mater in which could be recognized scattered areas or flakes of grey matter. There was no communication between the lateral ventricle and the cavity of the cyst.

Sagittal Section of the Brain : After separating the two hemispheres and dividing the brain into two by a sagittal section, the following points could be made out on subsequent transverse section of the two hemispheres :—The corpus callosum was fairly well developed ; the lenticular and caudate nuclei of both sides were normal and about equal in size ; the optic thalamus on the left side was distinctly and uniformly smaller than the one on the right. The left inferior quadrigeminal body was also the smaller ; the left inferior brachium was poorly developed. The corpus geniculatum mediale was absent, as was also the inner or medial root of the optic tract ; the lateral ventricle was normal on the right side and on the left anteriorly not greatly changed, but the posterior horn was very much dilated and to a less extent also the descending horn, while further, the left choroid plexus was enlarged and markedly cystic. The middle commissure of the third ventricle was absent, but the velum interpositum, the infundibulum, the pituitary and the pineal gland were normal.

The left crus was much smaller than the right. The pons and medulla were asymmetrical, the left again being smaller than the right, while the left olivary body was much more prominent ; the right anterior pyramidal tract also was well formed and rounded, that on the left side being absent. The right and left olfactory nerve and bulb were found normal ; the left optic nerve was distinctly smaller than the right.

The Cerebellum : The right cerebellar hemisphere was considerably atrophied in all dimensions, the left of good form and size.

Condition of the Blood Vessels : Immediately after removal of the brain its vessels were carefully examined and there was found very little evidence of atheroma. The internal carotids were normal and of the same size, as were also the anterior cerebral, the posterior cerebral, and the posterior communicating. The basilar and vertebral arteries were normal ; the middle meningeals, as again the venous sinuses in the dura mater, showed no deformity.

The right middle cerebral was found normal, but on the left side, shortly after the antero-lateral ganglionic branches were given off (to enter the anterior perforating space and supply the greater part of the caudate, lenticular and ophthalmic

nuclei and internal capsule), the vessel rapidly narrowed and became completely obstructed, so that it could be traced in its further distribution as a mere thread. Its branches, external and inferior frontal, the ascending frontal, the ascending parietal and the parieto-sphenoidal, could not be found.

It is obvious from the above description that the area of absence of cerebral substance corresponds with the area of the obliterated portion of the left middle cerebral. The accompanying diagram, showing as it does the extent of this distribution and the area of the loss of substance, shows very clearly this characteristic relationship.

I need not say that it is impossible to determine from microscopical examination what was the precise cause of the obliteration, but seeing that the first portion of this artery is normal and that all the other cerebral arteries are normal in distribution and in condition, it would appear most probable that the cause was either an inflammatory stenosis, or embolism, and the absence of any evidence of intrauterine inflammation elsewhere is, if anything, in favour of the latter of these two possibilities.

Now conditions in the offspring due to inflammatory or obstructive valvular disease are not in the same category with monstrosities, or even with nævi and birth-marks due to imperfect development of the vascular walls in one or other area. While we are, with reason, more than suspicious of any attempt to explain the latter by nervous or other maternal disturbances during intrauterine life, the same should not hold with regard to the former. It is worthy of note that I only obtained the history of this case after I had fully worked out the histology of the cord and brain, and had given an account of the case before the Montreal Medico-Chirurgical Society, now two years ago, and after I had before that society stated that, from the development of the various tracts, the destruction of the tissue of the left hemisphere must have occurred during the last two months of pregnancy. Given in this history the "threatened abortion about the seventh month," it is at least possible to explain the closure and subsequent atrophy of the left middle cerebral artery and the production of porencephaly, as being brought about by placental hæmorrhage, localised thrombosis of the fœtal vessels in the placenta, detachment
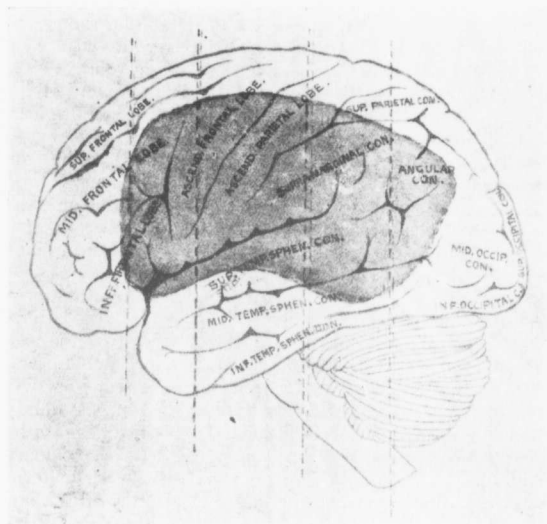
FIG. III.

Side View of Left Hemisphere [after Quain].

The shaded area maps out the area of destruction; the vertical dotted lines indicate the position of the sections made in this case.

FIG. IV.

Distribution of the Middle Cerebral Artery [after Charcot].

1. Perforating arteries. 2. Frontal branch. 3. Ascending Frontal.
4. Ascending Parietal. 5. Temporo-sphenoidal branches.

of a portion of the thrombus, and embolism of this one special vessel—the left middle cerebral.*

STUDY OF VERTICAL SECTIONS THROUGH THE CEREBRUM.

To describe in detail the naked-eye appearances of the medulla and cord in various sections would seem unnecessary, since a fuller and more accurate knowledge of the degenerative changes is gained by a study of sections under the microscope. It will, however, be well for me to rapidly point out the main facts shown by making a series of vertical sections through the cerebrum, and my description can be well followed by an examination of figures 5, 6, 7, 8 and 9.
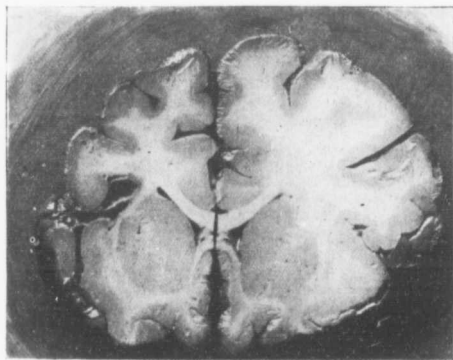

FIG. V.

First transverse vertical section, posterior aspect.

.The corpus callosum, the anterior commissure, caudate and lenticular nuclei, the external capsule, claustrum and the anterior lobe of the Island of Reil, are to be seen on both sides ; the *left* internal

* When we take into account that the patient herself afforded during her life singularly little indications of the extreme cerebral change found post mortem, that death occurred at the age of 76, that the patient's sister and brother neither knew nor were in a condition to appreciate the pathology of the case, and that this history was obtained without the employment of leading questions, I cannot but regard the history given in the first portion of this article as a most interesting example of the singular capacity shown by certain members of certain families to retain and treasure up the minute details of the life histories of individual members of those families.

capsule has not so many fibres as has the right ; the *left* centrum ovale is the smaller.   On the *left* side we can just see (below) the anterior inferior end of the inferior temporal convolution, and the remnants of the cyst wall.
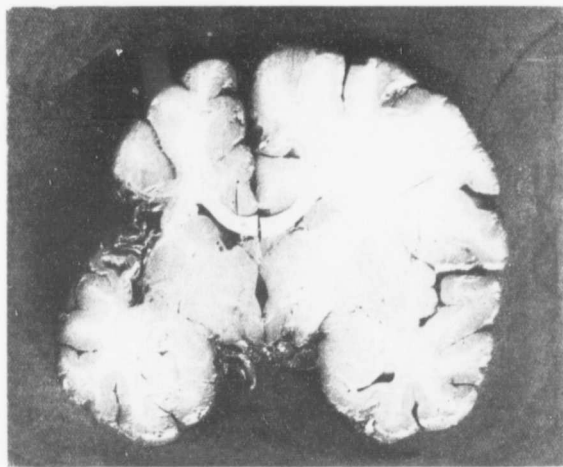

FIG. VI.

Second section : posterior aspect.

In this plate we see more noticeably the difference in size of the two cerebral hemispheres ; on the *left* side the 3rd frontal and part of the 2nd frontal convolutions have become atrophied.   The posterior lobe of the Island of Reil, claustrum and external capsule, are absent, and here also are the remnants of former convolutions.   The caudate nuclei on both sides are normal.   A marked atrophy of the *left* thalamus is apparent ; the internal capsule is to be seen in all its richness of fibres on the right side, but on the *left* only a few scattered fibres can be made out.   The corpus striatum, corpus callosum and fornix are much the same on both sides.
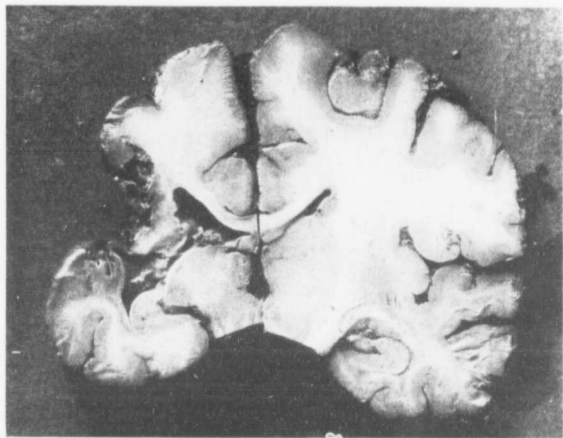
FIG. VII.

Third section : posterior aspect.

The difference in size of the two hemispheres is very noticeable although the halves of the section are not quite on the same plane owing to distortion of left side of brain. The difference in the two thalami is very easily seen, the *left* pulvinar being very poorly developed, the external and posterior part falling away externally to a sharp point. Note here the dilatation of the *left* lateral ventricle, its anterior and descending horns being markedly distended. The internal capsule can only be seen on the right side.

FIG. VIII.

Fourth section : posterior aspect.

The dilatation of the posterior horn of the *left* lateral ventricle is more marked than before, the choroid plexus distended and cystic ; inferior to the ventricle on the right side can be seen the full and well rounded end of the optic thalamus or pulvinar which cannot be seen on the *left* side ; when dissected out, the left pulvinar was found to be fully a third less. Below the splenium of the corpus callosum are the two superior quadrigeminal bodies, the right larger than the *left*. The centrum ovale is markedly less on the *left* side.
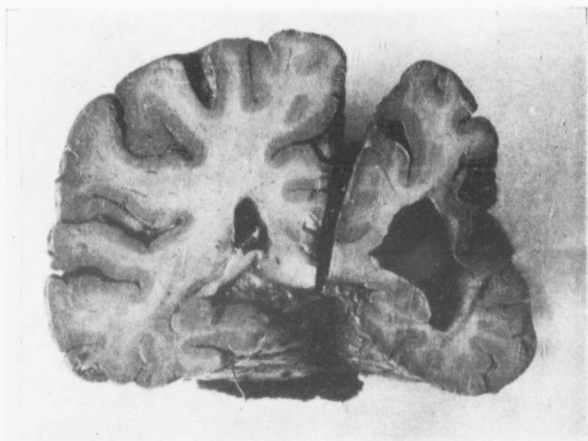
Fig. ix.

Hindmost section: anterior aspect.

This plate shows an enormous dilatation of the posterior horn of the *left* lateral ventricle; externally is to be seen the thickened ventricular ependyma and membrane containing shreds of nervous tissue. This figure is from a photograph of the *anterior* aspect of the hindermost section of the brain. The previous figures have been of the *posterior* aspects of the sections.

MINUTE ANATOMY.

The various parts of the central nervous system were preserved in formalin, alcohol and Müller's fluid; the methods employed for staining the sections were those of Van Gieson and Pal-Weigert, hæmatoxylin and eosin and a modification of Nissl's method which I have developed for showing the cellular and connective tissue elements.

In describing the results of the examinations and determining thus the effects of the porencephalus, it has seemed to me that the clearest understanding of the case will be gained by beginning from below, from the spinal cord, and working upwards; while throughout I shall as far as

possible divide my description of the various sections into the appearances of the *white fasciculi*, the *grey matter* and the *cellular elements*, respectively.

1. White Fasciculi : In sections stained by Pal-Weigert the difference in size and appearance of the two halves of the spinal cord is most pronounced, the *right* side is markedly the smaller throughout the cord. This is brought about by a nearly complete absence of the crossed pyramidal tract on that side ; only a few healthy fibres can be seen in the area, and these possibly are the so-called homolateral fibres, which

FIG. X.

Inferior aspect of section from region of 4th cervical.

have their origin from the right motor hemisphere and do not cross over at the decussation with the rest of the crossing efferent motor fibres, but continue down the right side of the cord.

Sherrington,[8] Muratoff and others have pointed out this bilateral condition. Those fibres occupying the above said position are surrounded by sclerosed tissue which is very abundant and spreads out in a fan-shape, the expanding edge of the fan being external. The line of demarcation between the atrophied crossed pyramidal tract and the direct cerebellar tract is very sharp and distinct, this last being quite free

from any excess of connective tissue between its fibres.
The demarcation is well shown in Fig. 10.

Anteriorly the fan-shaped sclerotic area, remains of the
crossed pyramidal tract, runs well forward; particularly is this
the case in the cervical region where it nearly approaches the
antero-lateral tracts of the cord. The appearances here are not
in harmony with those noted by Löwenthal.[9] He states that it
is where there has been injury to the cord that the degenerated
pyramidal tract has a fairly sharp lateral boundary, whereas
in injury to the cerebral cortex the edge of the degenerated
pyramidal tract is diffuse towards the cerebellar tract.
Throughout the cord there is no sign of diffuse sclerosis in
the area above indicated ; while further, the extension of the
sclerosis forward anteriorly would seem to indicate that the
pyramidal fibres themselves extend farther forward than is
usually considered to be the case. This anterior extension
appeared to trespass beyond the area which Flechsig gives
as representing the pyramidal tract. I may mention that
Schiefferdecker[10] has called attention to the fact that sclerosis
frequently extends beyond this area.

Turning now to the pyramidal tract on the *left* side, there
is here a diffuse sclerosis, and by the Pal-Weigert stain a
large number of white pin-points can be made out scattered
among the healthy fibres of the tract, while with Van Gieson's
stain there is seen to be an increased development of the
connective tissue framework of the part. Just as in the de-
generated right pyramidal area there is present a certain
number of healthy fibres, so in the relatively unaffected left
pyramidal tract we come across these scattered small areas of
sclerosis. And as the presence of the healthy fibres in the
former case is best explained by assuming them to be fibres
of the original right pyramidal tract which had not decussated,
so we may assume this scattered sclerosis to indicate areas
corresponding to degenerated non-decussating fibres of the
left pyramidal tract. There is a fair correspondence between
the amount of sclerosis on the healthy side and the number
of intact fibres on the sclerosed side. The fibres of this tract
coming from the unaffected right hemisphere are, I should
add, well developed.

As to the white fasciculi in the anterior columns between
the ventral fissure and the issuing fibres of the anterior root
bundles, the left is but half the size of the right. This differ-

ence is to be seen well to the end of the dorsal cord, and is due to the nearly complete absence of the *direct pyramidal tract* (Türck's Column) on the *left* side. It is to be noted that along the margins of the ventral fissure on the left side a few healthy fibres can be seen imbedded in scleroid tissue.

Along the whole length of the spinal cord, down to the lumbar region, *i.e.*, so far down as I possess material, this condition of asymmetry of the two sides continues, the right half being greatly larger than the left.

The posterior fasciculi of Goll and Burdach are through-out normal. At one time I was inclined to think that I recognised a certain amount of sclerosis in these two columns, but after having made a large number of sections and stained them by various methods, I feel assured that this is not the case.

Of peculiar interest, as indicating the existence of a tract not often differentiated or apparently noticed, is the fact that in the region of the fasciculus ventro-lateralis of Gowers, there is on both sides to be recognised a distinct area of degenera-tion beginning about the level of the 7th cervical segment and extending upwards until it reaches the medulla. The tract on the *right* can be traced upwards postero-laterally to the olivary nucleus of the same side. This is called by some the *triangular path of Helweg*,[11] or the *olivary tract of Bechterew*. The degenerated tract on the *left* is possibly the descending spino-thalamic. [12]

At the 7th cervical region these areas of degeneration lie on either side as thin bands on the outer surface of the ventro-lateral tract, and as we trace them upwards on both sides they assume the shape of a triangle, the apex pointing backwards ; at the 4th segment the apex on the right side is in close contact with the anterior edge of the fan-shaped sclerotic area of the crossed pyramidal tract. I shall discuss these tracts later in connection with degeneration of the fillet.

2. *The Grey Matter.*

The presence or absence of cell clusters or columns has much to do with the alterations in shape of the horns of grey matter, and here, as in the white matter, we have a marked asymmetry. The anterior horn on the right side is smaller than on the left, and the smallness is especially noticeable in

the cervical region, at the 5th, 6th and 7th cervical segments, on account of the absence, or atrophy, of the ventro-lateral and postero-external groups of cell bodies of the upper extremity group. If we cut off the ventro-lateral and

FIG. XI.

Inferior aspect of section through region of the 7th cervical segment.

postero-external groups of cell bodies of the left anterior horn, which are absent in the right anterior horn, the surface area of the right anterior horn becomes greater than that of the left. It may well be that this alteration in shape of the right anterior horn is in part at least accounted for by the absence or destruction of the crossed pyramidal tract, the consequent sclerosis and shrinkage of that side being followed by displacement of the grey matter outwards and backwards.

The anterior and posterior commissures are well developed, being exceedingly rich in commissural nerve fibres, both medullated and non-medullated.

The right posterior horn is short and stout compared with its fellow on the opposite side. On both sides the gelatinous substance of Rolando at the extremity of the posterior horns is well developed both as regards shape and amount.

3. *Cellular Elements.*

In the different segments of the spinal cord, the number of cell bodies and their arrangement vary greatly; in the grey matter of the *left* side, from the first cervical segment downwards, the different cell groups and individual cell-bodies are present and are in their usual position.

Examining first the section of the cord just below the termination of the medulla, where the crossing of the pyramidal tract is being completed, we notice in both anterior

horns two distinct groups of cell-bodies, internal and external. Of these the internal, if followed upwards, is seen to become the nucleus of the twelfth nerve, the external the nucleus of the eleventh nerve. In the grey matter of the second segments ventral and dorsal groups of cell-bodies are well developed on both sides of the cord, and as we proceed downwards through the third, fourth and fifth segments we can clearly demonstrate the different nuclei normally present—the accessory, the lateral horn cells of Waldeyer, Stilling's nucleus, the nucleus of the phrenic (well seen in the lower part of the third cervical and occupying a ventro-medial position between the cell bodies for the back muscles and the accessorius group)—all these are equally developed on the two sides.

In the lower part of the fourth cervical segment, extending through the fifth, sixth, seventh and eighth and beginning to disappear in the first dorsal region, is a group of cell-bodies called the upper extremity group. These cells are arranged in three sets—the anterior, the posterior and the internal—increasing and decreasing as they pass through the different segments. This group corresponds to the brachial plexus. The cells of the upper part of this group supply the muscles of the shoulder and arm, those of the lower part innervate the muscles of the forearm and hand. The different sets of cells forming this upper extremity group are all easily demonstrable on the *left* side, but in the *right* anterior horn the middle part of the ventro-lateral group, lying posterior to the accessorius nucleus, is absent. The absence is most noticeable in the sixth, seventh and eighth cervical segments. And also a few cells are absent in the dorso-lateral group, especially in the eighth cervical and first dorsal segments. Other cells of this group are normal

A group of cells, the *mesial anterior*, in the anterior horns, which shows a peculiar variation from the normal, is the nucleus supposed to innervate the back muscles, (Kaiser ([13]) and others), and also called by some writers (Lenhossek ([14])), the *commissural nucleus*, their axones never having been seen to enter the anterior nerve roots along with the other fibres. As a matter of fact, I have in several of my specimens clearly made out that the axones of these cells pass backwards and are to be seen entering the anterior commissure. Situated ventrally and towards the median aspect of the

anterior horn, this group is remarkable in the normal state for the persistence of its relative position in each succeeding segment, so that passing downwards it appears to form a well differentiated and distinct column through the different segments. This group or nucleus is especially noticeable in the lower animals.

Studying this group in the present case, I make out upon examining the various cervical sections from the fourth segment down to the first dorsal, that it is at one time to be seen distinctly in a normal position in the right horn, but to be absent in the left, or if not absent, showing marked signs of atrophy, many of the cells being completely absent, or of a low form, the rest markedly shrivelled.

Upon examination of a series of sections, made caudal wards, I found the condition reversed, the cell group being well formed in the left horn, absent or atrophied in the right; a little lower and there is a reversal of the picture, this notwithstanding the fact that the sections have been carefully cut in a transverse and not an oblique plane. I mention this without attempting to give any explanation at this point. Fig. 18, p. 45, shows the position occupied by these cells in the anterior horn.

It is in the cervical enlargement that the condition above described is best marked. As we pass down to the thoracic segments this commissural nucleus is not so isolated but is to a certain extent fused with the ventral horn cells, and here no noticeable difference can be detected between the cell groups of the two horns save that the cells appear, as in the cervical region of the cord, to be numerically more numerous in the right horn than the left, both in the cervical and the thoracic regions. Here, however, they are not so highly developed, nor so rich in dendrites, nor so markedly arranged into clusters, but they seem to be of a lower grade and more uniformly scattered.

Clark's cells and the *posterior horns* are normal.

THE CEREBRAL PEDUNCLES.

In viewing the different transverse sections through the brain stem the asymmetry of the parts is as strongly marked as was the case in the spinal cord, but here it is on the left side

and not on the right that the absence of many of the impor-
tant structures is to be made out.

Posteriorly the *left superior quadrigeminal body* is about
one-third smaller than the right ; it contains fewer nerve cells,
both large and small, and throughout its extent fewer medul-
lated fibres.   Anteriorly the zonal fibres on the left are fewer
than normal, and the superficial white fibres are almost wholly
wanting.   The *inferior quadrigeminal body* on the *left* is also
distinctly smaller than on the right and its brachium contains
hardly any healthy nerve fibres, while its body presents a
marked diminution of the myelinic network and of cellular
elements.   The right is normal.   The *left lemniscus* is
deficient in size compared with its fellow on the opposite side,
and the diminution affects both the fibres of the lemniscus
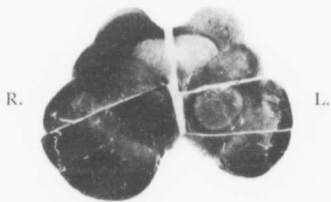lateralis, which enter the quadrigeminum inferior, and those

R.   L.

FIG. XII
Superior aspect of transverse section of the Peduncles.

of the lemniscus medialis originating in the nucleus funiculi
cuneati, as well as the bundles from the lemniscus to the pes.

The *corpus geniculatum mediale* on the *left* side is
absent ; the *left superior cerebellar peduncle* is markedly
smaller than its pair above the decussation, where the tracts
lie close together on either side of the middle line ; indeed the
left is less than half the size of the right peduncle in this
region and lower down, where the decussation takes place, it
it also very unequal.

The *left red nucleus* is markedly diminished in size.

The substantia nigra seems to be equally healthy and
well developed on both sides ; on the left side the posterior
longitudinal bundle is slightly the larger ; the fibres of the
fasciculus retroflexus Meynerti are reduced in number on
the left.   While the *pedal fibres* of the right side are well
developed forming a well rounded and bulging pedal system,

on the *left* side this track is very much diminished, being
present in the form of a narrow band of fibres running on the
ventral surface of the crus internally from the points of exit
of the roots of the third nerve outwards and backwards to
the groove situated at the most external end of the substantia
nigra.

The four different tracts which go to form the bundle of
fibres in the basis pedunculi and lie ventrally to the sub-
stantia nigra, consist of the pyramidalis (fasciculus longitudi-
nalis), the median bundle (the fronto-cerebro-cortico-pontal
path), the lateral bundle (the occipito-temporo-cerebro-cortico-
pontal path), and the fibres of the accessory bundle of the
lemniscus.   All the fibres that are present and which repre-
sent these tracts are most markedly diminished in number.
The nuclei and roots of the third nerve are both normal.

<center>THE PONS.</center>

In an oblique cut section of the pons and crus the follow-
ing is to be made out :—

Posteriorly, the decussation of the 4th nerve, more
anteriorly, the posterior longitudinal bundles, of which the left
is slightly larger than the right ; laterally, the *left superior
cerebellar peduncle*, which is nearly twice the size of the right,
the peduncle from the right hemisphere having crossed over to
this side at the decussation.   External to either peduncle we
have the lemniscus lateralis, that on the left side being
distinctly more slender than that on the right.

The *nucleus lemniscus lateralis* is well formed on the
right side, absent on the left.   The *nucleus reticularis
tegmenti* is not so healthily developed on the left side as on
the right.   Further a marked difference is to be made out
between the two *lemnisci mediales* ; the right is at least from
a third to a half broader ventro-dorsally than its fellow.
The fibres of the medial accessory bundle in the lemniscus
medialis, passing to the motor nuclei of the cerebral nerves,
is wanting on the left side, as are also the fibres of the
lemniscus which originate in the nucleus funiculi gracilis.
The fibres of the lemniscus pertaining to the nucleus funiculi
cuneati are well developed on the right side, not so well
developed on the left ; the fibres from the region of the

inferior quadrigeminal body to the nucleus reticularis teg-
menti and to the pons, are not so abundant on the left side
as on the right.

More anteriorly on the right side of the pontal region we
have stretching from near the outer angle of the median
lemiscus, forwards and inwards to the superficial fibres of
the pons, three well-formed longitudinal bunches of nerve
fibres separated from each other by pontal fibres and by the
mass of grey matter which make up the nuclei pontis. Of
these on the left only a few small longitudinal bundles of
medullated fibres can be seen, and these gradually decrease
in size as they go downwards and backwards. Only a few
fibres of the fronto-cerebro-cortico-pontal part and the
fasciculi longitudinales (pyramidales) are to be made out.
Neither at the cerebral nor caudal end of the pons are any
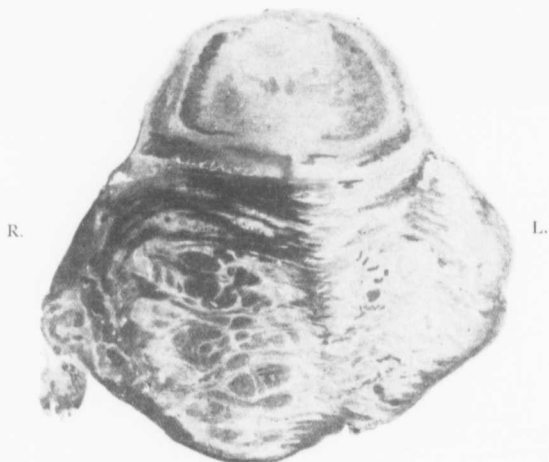fibres of the temporo-occipito-cerebro-cortico-pontal path to

R.   L.

FIG. XIII.

Superior aspect of section from superior pontal region.

be recognized. All these tracts are well developed on the
right side.

In the tegmental part of the pons many of the nuclei on the left side have disappeared and their places are filled up by fibrous tissue.

In a section taken below the level of the upper half of the 4th ventricle we see a marked asymmetry in the two *superior cerebellar peduncles*; as seen in previous sections, that on the *left* is about twice the size of that on the right ; so also the *middle peduncle* of the cerebellum (brachium pontis) is larger on the *left* side.

The *valve of Vieussens* shows marked signs of atrophy ; the granular layer on the right half has almost entirely disappeared, and both the fine plexus of nerve fibres and the large fibres are markedly diminished in amount. This degeneration or atrophy is connected with the degenerated tract of the right cord, in the region of the direct cerebellar tract known as Gower's tract.
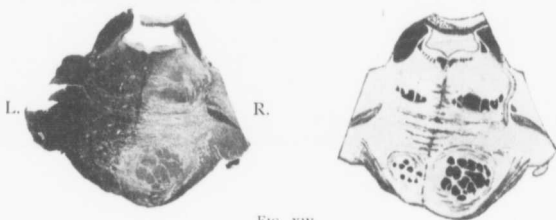
FIG. XIV.

Inferior aspect of inferior pontal region, with tracing made from the original photograph in order to indicate more diagrammatically the relative sizes of the more important tracts as against certain areas of degeneration.

MEDULLA OBLONGATA.

Having thus given the condition of the parts below in the cord and above in the crural and pontal regions, I would now describe the connecting medulla oblongata. Sections from the lower extremity of this made immediately above the plane of the decussation of the anterior pyramids show that the two sides differ greatly, the difference being due largely to the shrinkage of the degenerated *left anterior pyramid.* The internal arcuate fibres of the left side arising from the nucleus gracilis and nucleus cuneatus are well marked as they proceed forwards on the outside of the roots of the glosso-pharyngeal nerve and afterwards turn inwards towards the

raphe to form the decussation of the fillet, and then place themselves between the antero-lateral ground bundles and the anterior pyramid.

On the *right* side the nuclei of the *posterior funiculi* are seemingly atrophied ; the fine plexus of nerve fibres and the cellular elements are greatly diminished, more especially in the nucleus funiculi gracilis. The internal arciform fibres are much less in number, and are not easily to be made out in their passage from these nuclei to the raphe in front to form the decussation. The calibre of these fibres seems to be reduced, the fibres themselves to be more scattered, nor can their entrance into the nucleus funiculi gracilis be made out. Ventrally, on the right side, the large bundle of healthy pyramidal fibres is well in evidence ; on the *left* side this tract is markedly shrunken, consisting largely of fibrous tissue in which very few healthy longitudinal fibres remain. Running transversely through these tracts can be seen some of the

R.   L.
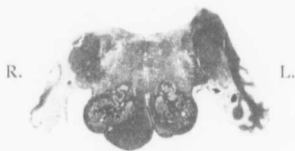
FIG. XV.

Superior aspect of section from Medulla.

anterior external arcuate fibres. In general, on both sides these external arcuate fibres are well developed.

On the most ventral aspect of this region, the left nucleus for the arcuate fibres is very highly developed. This nucleus can be made out in the various sections extending upwards as high as the upper end of the olivary body.

On studying sections through the mid-olivary region the asymmetry of the parts is more marked than ever. The lateral internal arcuate fibres (fibræ olivo-cerebellares) which pass from the restiform body, partly through, partly behind, and partly in front of the olive, and end in the opposite olive, are present in about equal numbers on the right as on the left side. But the inter-olivary stratum of the *lemniscus* here is less than half the size of its fellow on the opposite side ; the difference between the posterior longitudinal bundles at this

level is not so marked.   It is, however, to be noted that the
median internal arcuate fibres are less abundant on the *right*
side than on the left.

The left *restiform body* is larger than the *right.*   The
formatio reticularis on the right side is larger than the *left.*
With regard to the olivary bodies both appear to be healthy,
and their finer structure does not seem to depart from the
normal, but the left olive is certainly much more contorted
and irregular, the hilus in some sections looking forwards and
upwards.   The pyramidal bundles at this level are much the
same as has already been described above.

OPTIC NERVES AND OPTIC TRACT.

The   left   nerve   is   distinctly   smaller   than   the   right,
both macroscopically as well as microscopically.   In sections

FIG. XVI.
Sections through optic nerve.

made one-third of an inch forward from the chiasm it is seen
that along the inferior border a large number of nerve bundles
in the left nerve are markedly atrophied and surrounded by an
overgrowth of glia tissue.   This tissue is present in greater ex-
tent than normal all through the nerve, though in the centre and

superior and lateral borders the bundles of nerve fibres appear
well developed. A section of the right optic nerve shows at
the same level a marked atrophy of the central bundle of
fibres, with proliferation of the interstitial glia tissue ground-
work between all the bundles.

Optic Tract.—The *left* optic tract arises from one root,
for, as already mentioned, the *internal geniculate body* with
its brachium and the *inferior corpus quadrigeminum* are
absent or very greatly atrophied, and thus the inner root is
wanting. A section of the tract shows that it is much
diminished in circumference as compared with that on the
right side, though what bundles are present are equal in size
throughout, and little if any sclerotic tissue is to be found ;
indeed, on both sides there is an absence of sclerosis.

Pulvinar and Optic Thalamus.—Vertical sections made
through this show that on the *left* side there is marked
atrophy both in the cellular constituents and fibrillar network.
Figure 17 demonstrates the presence of a large number of

FIG. XVII.

To left, sections through left Thalamus ; to right, sections through right.

medullated nerve fibres in the pulvinar in the right side, while
on the left very few can be made out, and those mostly at the
circumference.

CEREBELLUM.

The *right* side is found to be considerably atrophied in
all directions, proving the existence of a crossed cerebro-
cerebellar connection.

The right dentate nucleus is much smaller than that on
the left side and contains fewer cells. The cells of Purkinje are

markedly lessened in number, as is also the granular layer. The large fibres of the medullary rays are greatly atrophied. Those of the finer association system are distinctly to be seen. To the cause of this atrophy I will refer later (pp. 52 *et seq.*).

CEREBRUM.

Sections from different parts of the left cerebral cortex show that this is uniformly atrophied throughout the hemisphere. The convolutions are small, the cortex is thinned, occasional *plaques jaunes* and small cysts are to be made out. There is great increase of glial tissue in the grey matter, with thickening round the blood vessels and atrophy of the nerve elements, in particular of the large pyramidal and polymorphous cells. The smaller pyramidal cells appear fairly abundant. The white matter is greatly diminished, there being a marked lessening of the projection system and of the superficial tangential fibres, while the long association fibres are fairly numerous. The atrophy above mentioned is particularly marked in what remains of the central convolutions, while in the frontal and occipital regions there is not such a marked absence of the large pyramidal cells, or of the projection fibres.

ETIOLOGY.

Porencephaly in general may be produced by the following causes :

1. Prenatal conditions.
2. Those related to birth accidents.
3. Postnatal, those dependent upon disease or tumour.

The processes leading to this condition may further be classified as follows :

(*a*) Agenesis, or lack of development of the brain elements *ab initio.*

(*b*) Constitutional lack of capacity for growth, the tissue elements being in development so weak as to succumb easily to intercurrent disease and toxic influence, forming thus a *locus minoris resistentiæ.* These diseases and influences are : (i) Prenatal meningoencephalitis, (ii) congenital syphilis and other toxic conditions, (iii) intra-uterine disease of the cerebral vessels (thrombosis, embolism, meningeal and intracerebral hemorrhages).

(*c*) Direct injury to the brain before, during and after birth. With reference to the processes occurring during birth, the condition may follow protracted labour, precipitate labour or forceps accidents : of these the second is especially liable to develop the condition.

(*d*) Disease processes affecting the fully developed brain substance, (hemorrhage, embolism, endo-and peri-arterial changes, cerebral venous thrombosis and encephalitis). Strumpell claims cortical polio-encephalitis as one cause.

From the description given of this case there can be little or no question that its causation comes under the first of the above headings, that it was of prenatal origin, and further that the process was set up by vascular disease in the fœtus ; for we find the middle cerebral occluded and arrested at a certain point, the absence of tissue corresponding with peculiar sharpness to the usual distribution of the branches of the middle cerebral artery beyond the point of obstruction. That the condition is not due to agenesis is, I take it, proven by the condition of the cavity. According to Kundrat where there is developmental defect there is a characteristic radiate arrangement of the convolutions around the cyst with thinning

or absence of the walls of the lateral ventricles.   Here there
is no such arrangement.*   As a further support of this view
we have the fact that the right hemisphere is well developed,
which would scarce be the case were we dealing with a con-
genital lack of growth.   In all likelihood the lesion occurred
some time late in intra-uterine life, possibly about the eighth
month of development.

It is deserving of note that though such extensive de-
struction had occurred there was little evidence of asymmetry
of the skull (this has frequently been noted in other cases),
even though the uninjured convolutions of the left side were
poorly developed, so also that there was no very great arrest
of development of the paralysed parts although there was
such marked absence of motor fibres in the one pyramidal
tract.

ON THE DIAGNOSIS OF PORENCEPHALUS.

The history of our case shows very clearly how difficult, if
not impossible, it is to give during life a diagnosis of the
extent of the lesions.   In most cases of porencephalus with
lesions as extensive as those found in this case, the patients
are more or less idiotic, presenting marked spastic con-
ditions and suffering from epilepsy or convulsions.   Here,
although so large an area was affected, the mental
power was good, the paralysis was of little account,
spasticity also slight.   Nor during the whole course of the
patient's seventy-six years was there a single convulsion
or epileptic fit.   It is to be noted that here the oblitera-
tion of the middle cerebral apparently occurred at a point
beyond the region of the artery from which is given off the
blood supply to the basal ganglia, so that the blood supply to
these was intact—another fact which would seem to account for
the normal appearance of the caudate and lenticular nuclei.
It is difficult to account for the atrophy of the optic thalamus
save on the assumption that the neurones of the destroyed
area were more closely related with that body, its general

* I am not of the opinion that the radiate arrangement of the sulci is alone
sufficient to diagnose a condition as congenital.   For on the one hand, as pointed
out by Collier and Russel (15), undoubted congenital cases have been reported
by Anton, Schultz and von Lumbeck, where the arrangement of the convolu-
tions was found normal, and on the other hand the radiate arrangements have been
found in cases certainly post-natal by von Monakow, von Kahlden and others.
Porencephaly has been produced experimentally in animals by Bikeles and
D'Alundo.

integrity being incompatible with the idea that its blood supply had been cut off.

I would now, passing from the more immediate description and study of the case, proceed to discuss certain matters of wider neurological interest upon which my findings here appear to throw light.

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ON PRIMARY AND SECONDARY DEGENERATION.

As above stated, there was a profuse atrophy of the grey matter of that portion of the left hemisphere not directly involved in the porencephaly, and this atrophy had associated with it a lessening of the large pyramidal cells and projection system of fibres and to a less degree of the tangential fibres, those present consisting mostly of the long association fibres. We must regard this atrophy as due in part to the partial destruction of the associate and projection system of fibres in their passage through the destroyed area, by anæmic necrosis following upon thrombosis, as being, in short, a Gudden's atrophy. Remembering (*a*) that each axone is part of a neurone, an injury to any part of which will affect the rest to a greater or less extent, (*b*) that no considerable portion of the neurone is capable of existence for any great length of time after severance of its connection with the rest of the nerve unit, and (*c*) that this is true, according to Barker, for both motor and sensory neurones, we thus gain an explanation for the great diminution in the number of nerve cell bodies and fibres, and so, in consequence, for the diminution in the size of the remaining portion of the left hemisphere.

The view here enunciated that destruction or severance of part of the nerve unit leads sooner or later to degeneration of the rest of that unit is in contradiction to the doctrine of trophic centres as formulated by Waller, and in connection with this case, in which we have such extensive degeneration not only of those tracts which are formed of axones in direct connection with cell-bodies in the affected portion of the left cerebral hemisphere, but also of other fibres and centres which form tracts not directly connected, but associated, with the cortical system of neurones, it may be well to discuss this matter of the nature and extent of degenerative and atrophic processes in the neurones.

From the appearances presented in this case, to what extent are we justified in adhering or departing from Wallerian doctrine? In the first place we have to recognize that it is not necessarily true that that part of the neurone remaining in connection with the nucleus and cell body is unaffected. Marinesco indeed has shown that after amputation of the limb or after section of a peripheral nerve, definite pathological changes occur in the central or proximal portions of the nerve, the intensity of which depends upon the species and more especially upon the age of the animal and upon the length of time intervening between the injury and death. While appearing at a much later date than in the distal portion, he points out that the degeneration in the central portion of a divided nerve presents similar morphological appearances, and is apparently analogous to the process occurring in the distal portion, and this degeneration occurs although there has been no disturbance of continuity between the proximal portion and its trophic centre.

Whether this latter " proximal degeneration " is in all essential characters identical with the Wallerian " distal " degeneration is still in question. We must admit that the nerve cell is liable to conform to the general law applicable to all other active cells of the organism, the law that atrophy follows disuse. Admitting this, Mott[14] doubts whether this simple atrophy is of the same order as the degeneration which follows upon severance of portions of a neurone from its trophic centre, and he points out that in it there is an absence of any chemical change, save in those cases in which the injury is so severe as to cause death of the neurone as a whole.

In studying a mixed nerve, not only do the sensory fibres in it—those distal to the spinal ganglion – degenerate, but after a time there is recognized degeneration and disappearance of fibres in the dorsal root proximal to the ganglion and in the corresponding fibres, their collaterals and terminals in the dorsal funiculi of the cord, these eventually undergoing total disappearance. As regards the motor fibres in the central stem of the amputated limb, these gradually diminish in number ; in some instances they appear to vanish almost entirely ; and, corresponding to this, a large number of the motor cells of the ventral horns dwindle in size, and may after a time completely disappear.

The bodies as distinct from the processes of the sensory neurones above mentioned, namely, spinal ganglion cells, do not show gross alterations for some considerable time after the peripheral and central fibres have degenerated (Friedlænder,[15] Krause[16] and Marinesco[17]), a finding which denotes that the trophic mechanism here differs in some way from that connected with the nutrition of the cells of the ventral horns. Barker thinks this may depend upon possesion by the ganglion cells of a cellular capsule.* Nevertheless this degeneration eventually occurs.

While thus the nutrition of the cell body determines to some extent the presence or absence of immediate degeneration, we have to recognize that nutrition is but a secondary factor in the matter. Nay more, we have to recognize that in the nervous system, as in other parts of the body, nutrition is correlated to function : increased function brings about increased nutrition of the part, decreased function diminished nutrition. And if from any cause the function of a tissue is arrested—we can go further and say, if the function of a cell is arrested—that arrest brings about diminished nutrition and atrophy.

Upon general pathological principles, therefore, we must be prepared to find that if a nerve cell is prevented from

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*Another suggestion given (I have mislaid the reference), is that the peculiar relationship of the spinal ganglion cell to its axone, is capable of explaining this difference in the behaviour of the cell. It is suggested that as a consequence of the T-shaped connection of the axone with the body, stimuli normally pass up the dendrites and distal cell processes from the periphery and pass by the cell body without traversing the vertical limb of the T, that thus the spinal ganglion cell body leads usually a less active existence, if I may so express it, is more passive and less liable to be influenced by severance of the peripheral fibrils and cutting off the stimuli which normally traverse its axone.

exercising its function, that nerve cell must surely present eventual evidences of a greater or less degree of atrophy from disuse. And I would go beyond this, and would state that if in the efferent cortico-cerebro-spinal path, after destruction of certain areas of the cortex, we find that certain centres along the path with their associated fibres and tracts, undergo atrophy, whereas other centres do not show such atrophy, the inference to be made is, that in the first case the centres receive their predominant impulses from the neurones of the destroyed area and, in the second, that while there may be connection between the neurones of the destroyed cortical area and the centres, nevertheless, there must be equally or more important associations between the centres and other areas that are unaffected. Or, in other words, in the first case destruction of the neurones of the destroyed cortical area has been followed by deprivation of function and consequent atrophy; in the second case, destruction of the cortical neurones has not had this result, impulses reaching the cells of the centres by other paths.

ON AREAS AND TRACTS OF SECONDARY "DEGENERATION" AND ATROPHY.

Passing from these more general considerations, I would now say a few words with reference to the presence of sclerosis and disappearance of fibres in the *fillet* system, the thalamus, the *inferior quadrigeminal body*, and again to the absence of the *mesial geniculate body*. Von Monakow has pointed out that the majority of the nuclei and cell clusters in the thalamus, the geniculate bodies, the substantia nigra, and the pons, are made up of projection neurones whose axones are mostly cortico-petal, terminating in the cerebral cortex. If this be so, then the atrophy of these nuclei is to be explained by the fact that a destruction of a large area of the cortex has led to the injury, not to say destruction, of the peripheral endings of a large number of neurones, and we must regard the degeneration or atrophy of the nuclei as due to progressive degeneration of these infra-cortical neurones or cells-bodies, secondary to injury of their peripheral portions. In either case, whether all the nuclei above mentioned possess cortico-petal axones or whether

the cells-bodies in these nuclei are associated with cortico-fugal axones, the degeneration and atrophy that occurs must, it seems to me, be ascribed primarily to disuse and loss of function. In this view I find that I am in agreement with a large number of authorities, though Marinesco and others are inclined to lay stress upon the fact that a true degeneration takes place in the tracts above mentioned.

In recent experiments carried out by Mellus in which he extirpated small areas of the motor cortex and killed the animals (monkeys) from two to five weeks after the operation; there, studying the brain by Marchi's method, he found degeneration affecting many of the associated fibres, as also degeneration of the projection fibres, which he could follow through the centrum ovale to the internal capsule. From this region he noticed that many fine, degenerated fibres passed into the thalamus and into the substantia nigra, apparently terminating there. Von Monakow also has shewn that, where there is destruction of the frontal convolutions, degeneration extends back through the internal capsule and downwards through the base to the cerebral peduncle, and in some instances the radiations of the nucleus medialis thalami and a portion of the nucleus lateralis thalami, atrophy and, after a time, disappear. Whether all these tracts undergo an immediate or a secondary degeneration, or no, we can at least recognize, more especially from these observations of von Monakow, why in our case the thalamus was so markedly atrophied.

ON THE EXTENT OF SCLEROSIS.

The large amount of sclerotic tissue present in the right pyramidal tract and other tracts which have undergone degeneration in our case, is very remarkable. As a rule one does not expect to find sclerosis well-marked in cases of secondary degeneration following a lesion occurring before birth and, probably, before myelination of the efferent system had taken place, and especially at such a late date (seventy-six years) after the primary lesion; one would have expected absorption to have occurred to a much greater extent. Freud, quoted by Collier,[46] says that, in secondary degeneration, the fibres of the pyramidal system may after a long time disappear so completely as to leave no

trace of their existence ; this, he says, may account for the absence of the tracts in some cases.

Sherrington and Langley,[47] in speaking of degeneration of nerve fibres followed by sclerosis, state that the first five or six weeks of degeneration are marked by the gradual disappearance of the fatty constituents of the medulla, and the swelling up of the axis cylinders of the nerves. In the later stages the medulla and axis cylinders entirely disappear, and after a time the only sign of degeneration is a slightly increased amount of connective tissue laid down between the remaining normal nerve fibres. According to Singer, this hypertrophy of connective tissue also in turn disappears, so that in from two to six months no signs of degeneration may be visible.

But François Franck and Pitres found degeneration recognizable ten months after cortical lesions. I am inclined to think that one explanation for this apparent absence of sclerosis so soon after lesions may be the use of carmine as a stain ; for at first, soon after its formation, sclerosed tissue stains deeply with carmine, but as time goes on the fibres stain less and less, the red patches become less and less noticeable, so that at length no satisfactory indication is obtained by this means of the presence of sclerosis. By the Pal-Weigert stain however, the presence of sclerosis can be determined for very long periods.

Another point worth noticing in this case is that, despite the large amount of sclerosis, the nerve fibres do not appear to be pressed upon. We find healthy looking fibres right in the centre of the sclerotic scar, apparently wholly unaffected. It is often stated in connection with locomotor ataxia, for instance, that in the sclerosed areas the nerve fibres are pressed upon, thereby being caused to atrophy and disappear. It is, I think, conceivable that where there is acute or sub-acute inflammatory disturbance in the cord, there the excessive development of new connective tissue may be followed by marked shrinkage, leading to atrophy of the incarcerated nerve fibres ; but where there is a slow process of " replacement " fibrosis, the new connective tissue strictly replacing and filling up the gap left by the atrophy of the nerve tissue, it may well be that such shrinkage plays a very minor part.

In this connection a word or two may be said with regard to the existence already noted of the presence of a diffuse sclerosis in the crossed pyramidal area on the left side, i.e., in the relatively healthy side. According to Sherrington, this is due to recrossed fibres, i.e., to the presence in the tract of one side of fibres belonging to the opposite side. Sherrington's first idea was that in their path downwards they twice crossed the median line, the proximal crossing being at the pyramidal decussation. Mellus is of the opinion that these have not twice crossed but have never crossed, i.e., that instead of decussating in the pyramidal region they have continued downwards on the same side. I remember seeing the specimens at Johns Hopkins Hospital, Baltimore, in which Dr. Mellus was able to demonstrate this fact. Sherrington, in more recent articles, appears to agree with him and with Muratoff, that the bilateral degeneration of the lateral columns following upon lesions of one hemisphere is due to the pyramidal tract dividing at the decussation into (1), the major portion, which in the well known manner crosses to form the opposite lateral funiculus of the cord, and (2), a minor portion which enters the lateral funiculus of its own side, the ratio of the uncrossed to the crossed tract being roughly one-fourth. Hamilton again states that he has followed degenerated fibres from one cerebral cortex through the corpus callosum down through the internal capsule on the opposite side and so through the pyramidal tract of the cord of the opposite side.[48]

The above observations are of high significance as showing how one hemisphere by means of the pyramidal tracts has connection with both sides of the cord and with the lower motor neurones of both sides of the body. To this we add the later researches of Hoche, [49] in which he has shewn that, from the lateral pyramidal tracts, fibres pass, not only to the ventral horns of the same side, but also through the ventral or anterior white commissure to enter the ventral horns of the opposite side; so that the fibres from the direct pyramidal tracts on the one side terminate in the grey matter of the ventral horns of both sides. We thus gain material help in explaining how in a case like the present, in which we have nearly total destruction of the pyramidal motor fibres of the one cerebral cortex, the other hemisphere, through its con-

nection with both sides of the cord, can control the motor
function of both sides.

ON THE ABSENCE OF THE DIRECT PYRAMIDAL TRACT AND ITS EFFECTS.

Attention may at this point be called to the complete, or
almost complete, absence of the direct pyramidal tract on the
left side. I have already noted (p. 25) the atrophic condition of
what certain authorities speak of as the commisural nucleus, a
group of cells situated towards the medial anterior aspect of
the anterior horns. According to certain authorities (Kaiser
and others) this nucleus is the motor centre for, more especi-
ally, the back muscles and the pectorals. Certainly in this case,
as observed at the autopsy, the pectorals on the right side were
distinctly small and weak, and the kyphosis and inclination to
one side would, I think, indicate a weakness in the back
muscles, points which at least favour and do not contradict this
view as to the function of the nucleus in question.

I must here recall the curious alternation of groups of
healthy and atrophied neurones of these nuclei on either side
in the cervical region. For this alternation I can afford no
adequate explanation ; I can at most suggest that the fibres
of the direct pyramidal tract are in part directly associated
with the cells of the commisural nucleus of the same side,
others decussate and become associated with the cells of the
nucleus of the opposite side, in the manner indicated by Hoche
in connection with the lateral pyramidal fibres. If this be
so, the accompanying figure indicates how the alternation
may be brought about.

In connection with the absence of cell bodies in the
ventro-medial and dorso-lateral nuclei of the upper extremity
nucleus, the disappearance is most marked from the seventh
cervical to the first dorsal segment ; the group which lies in
the most lateral part of the ventro-medial group and the
most dorsal and inferior part of the dorso-lateral group, are
more particularly involved. The absence of these groups
coincide very closely to the localization given by Ham-
mond [50] for the supply of the muscles of the forearm in the
ventro-medial group and of the hand in the dorso-lateral
group. The patient showed greater wasting and atrophy of

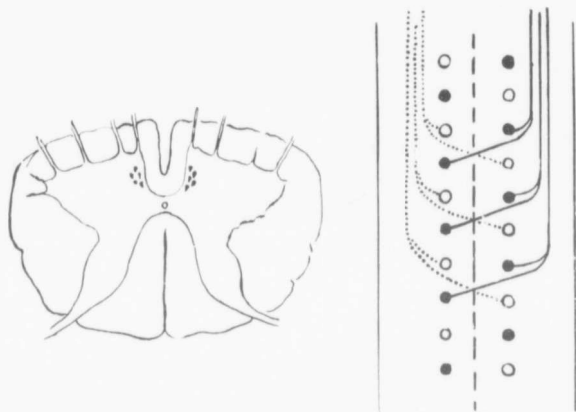the right forearm and hand than she did of any other part, and the relationship seems to be very close.

To illustrate the position of the " commisural nucleus" and the relationship between the direct pyramidal tract and the nuclei of the two sides.

ON THE FILLET.

There is great difference of opinion regarding the relationship between fillet fibres and the cortex cerebri and as to whether they consist of efferent or afferent fibres. Many state that they end in the thalamus and consist only of afferent fibres. Gee in his article in Brain,[16] points to ascending and descending degeneration of the posterior longitudinal bundles in a pontine hemorrhage, but in his case there was no degeneration of the fillet below the lesion, while above it was distinctly degenerated. His conclusions are that the lemniscus consists of fibres which ascend only, that the lateral

lemniscus appears to end in the lateral corpora quadrige-
mina, the mesial proceeding brainwards towards the optic
thalamus and there dividing into two bundles, one going to
the thalamus, the other mass of fibres appearing to take a
lateral direction dorsal to the corpus subthalamicum and that
these fibres may enter the optic thalamus higher up or may
pursue an uninterrupted course to the cortex.

Kingdon and Russell[19] describe a descending degenera-
tion of the fillet apparently secondary to extensive cortical
degenerations in children. Bruce[20] shows that in consequence
of a lesion destroying the basal ganglia and internal capsule
in the cerebrum, the fillet does degenerate in a descending
direction through the inner field of Flechsig on the same side
of the medulla, through the median internal arcuate fibres to
the opposite side as far as the nuclei of the two divisions of
the posterior columns of the cord, and that the nuclei there
undergo a secondary atrophy proportional to the degeneration
of the fillet.

Flechsig and Hossel[21] maintain that there is a direct
connection between the nuclei of the posterior funiculi and the
central convolutions, i.e., that some of the axones of the cells
of the nuclei of Burdach and Goll pass uninterruptedly to
the sensory motor cortex of the Rolandic region, forming
what is termed the cortical fillet. Mott,[22] Dejerine[23] and
von Monakow[24] maintain that there is not any *uninterrupted*
connection between the nuclei of the posterior funiculi and
the cortex. Mott has noted that in monkeys, in which he
destroyed the nuclei, he obtained extensive degeneration of
the fillet, but found no evidence of degeneration affecting the
internal capsule or the cortex.

Langley and Grünbaum,[25] Jacob[26] and others, after
producing large cortical lesions in animals, found the fillet did
not show immediate degeneration but that it might be atrophied
as a consequence of these cortical lesions.

The presence of sclerosis in the median lemniscus in
our case might indicate the descending motor tract which
Hoche[27] has shewn in his recent researches to be situated in
that region, a region which we have been accustomed to
consider as almost purely sensory. These motor fibres are
entirely different from the majority of the fibres of the
lemniscus medialis, and this is proven not only by the fact
that they degenerate in a descending direction, but also by

the fact that higher up in the nervous system they become entirely separate from the rest of the fibres of the median lemniscus. In the uppermost planes of the cerebral peduncle these fibres do not lie in the region of the lemniscus at all, they are situated at the base of the peduncle in the immediate neighbourhood of the fibres of the pyramidal tract and lateral to them. But long before these observations of Hoche, von Bechterew[28] and Flechsig[29] made out that a certain number of fibres from the median lemniscus become medullated at a later period than do the majority, and they gave to this special bundle the name of *median accessory bundle*.

In our case as before described, we have a marked atrophy and degeneration and disappearance of fibres and the presence of sclerosis in both the median and lateral fillet of the left side ; this is most noticeable in the pontal region. We can trace this tract of atrophy and degeneration down to the atrophied nuclei of Goll and Burdach on the opposite side. Above we find that the fillet fibres above the pons on the left side become much thinned out and appear to terminate at different stations. As the corpora quadrigemina are approached we notice that the lemniscus gives off a dorsal branch, the lateral lemniscus, and of this branch the large majority of fibres seem to end in the corpora. Yet, confirming what has been recently stated by Gee,[30] a few fibres, apparently belonging to this part of the fillet, cross to the opposite side by way of the lingula (vide Fig. 14). The main or median lemniscus travels upwards towards the optic thalamus, but whether many of the fibres pass on to the cortex, is, as already stated, a disputed point and cannot be proven in this case.

ON TRACTS IN THE VENTRO-LATERAL REGION OF THE CORD.

Of peculiar interest, as indicating the existence of tracts not often differentiated or apparently noticed, is the fact that in the region of the fasciculus ventro-lateralis of Gowers, there is to be recognized on either side a distinct area of degeneration which begins about the level of the 7th cervical

segment and extends upwards until it reaches the medulla.
The track on the right side I can trace a segment lower than
that on the left. They both begin as a thin band of sclerosis
on the outer surface of the ventro-lateral tract and as they are
traced upwards on both sides they assume the shape of a
triangle, especially well noticed at the 4th cervical segment.
The apex of the one on the right side comes in close contact
with the anterior edge of the fan-shaped sclerotic area of the
degenerated crossed pyramidal tract. For several reasons, to
be mentioned later, I am inclined to think that this
degenerated tract on the *right* side must be a Gudden's atrophy
of Gower's tract, the *fasciculus spino-cerebellaris ventralis,* and
not the descending cerebellar tract of Marchi or Biedl,
although there are some points that would favour this latter.
Although I cannot trace this sclerotic area below the 8th
cervical segment, yet I can trace it up in the medulla, pons
and mid-brain in the position usually given by the majority of
observers for the position of Gower's tract, namely : passing
up from the ventro-lateral portion of the continuation in the
medulla of the ventro-lateral fasciculus of the cord, it first
lies lateral to the nucleus of the olivaris inferior, and
higher up it becomes situated ventro-laterally to the nucleus
olivaris superioris. In the pons the fibres leave their ventral
situation to form a loop over the 5th nerve ; they are then
directed obliquely upwards and backwards to the surface
of the superior peduncle, forming a layer of fibres continuous
with the valve of Vieussens. They then run downwards on
the posterior surface of the peduncle to stream inwards to the
superior vermis (Mott,[30] Schaefer) and to the lateral lobes
(Patrick [32]), in exactly the position described by the above,
and they can be made out in the specimens as well as in the
photographs in the medulla and in the pons. One can see
the sclerotic area looping over the 5th nerve and can follow
it obliquely upwards and backwards to the surface of the
superior peduncle (vide Fig. 14). This superior peduncle is
noticeably smaller, apparently on account of absence of fibres
from the right cerebellum and also from the absence of fibres
making up this tract. The valve of Vieussens is distinctly
atrophied on the right side, so also is the superior vermis and
the lateral lobe.

Recent writers state that the tract that is called Gower's
tract consists of at least several distinct neurone systems and

takes a course for at least a long distance in the spinal cord and rhombencephalon. Mott [31] distinguishes, in addition to the (largely crossed) conjunctival spino-cerebellar system, a *ventro-lateral superior spino-quadrigeminal system*, and a *ventro-lateral-spino-thalamic system*. Here I think this degeneration belongs to the first named — the crossed conjunctival spino-cerebellar system; we have no atrophy or sclerosis in the right quadrigeminal body or in the lemniscus lateralis on that side, which would be present if the spino-quadrigeminal tract were affected, nor in the last mentioned spino-thalamic tract do we have any alteration in the thalamus.

On the other hand Marchi [33] has shown that there is a cerebellar system in the antero-lateral columns of the cord, originating mainly from the vermis, descending into the ventro-lateral fasciculi of the cord. Ferrier,[34] Russell[35] and Turner[26] say that this system is not direct, as Marchi states, but proceeds by way of Deiter's nucleus, which was normal in this case. In our case we have an atrophy of the right inferior restiform body which might point to descending fibres from the cerebellum going *via* that body into the spinal cord. Now, as pointed out by Biedl as the result of an experimental section of the corpus restiforme, two descending degenerated tracts are to be found, one in the ventro-lateral fasciculus, in the area corresponding to that described by Marchi and Lœwenthal; the other tract descends in the funiculus lateralis in the exact position occupied by the pyramidal tract.

Admitting, therefore—as I think we have to admit—that in the ventro-lateral area of the cord there are several different tracts, is it possible that here in our case in this right-sided ventro-lateral area of degeneration and atrophy we have present both a Gudden's atrophy of Gower's tract and a descending atrophy of the tract depicted by Biedl?

The degenerated track on the *left* side I took at first as being probably the *triangular path* of Helweg, or the *olivary fasciculus* (Olivenstrang) of Bechterew,[37] which extends between the olivary nucleus and the spinal cord. The inferior olive on the left side is distorted and out of its position, yet with careful examination I could make out no alteration in its structure from health. Bechterew himself says that there is no proof that these fibres are directly related to the nerve

cells of the olive. (This being so I would urge that the term
"Olivenstrang" is misleading and faulty.)

Now in our case I find a tract of degeneration exactly
corresponding to that described by Bechterew. I, like Bech-
terew, found no involvment of the olive; unlike him I was
able to trace the degenerated tract in the ventro-lateral region
of the cord up into the medulla in the region occupied by the
antero-lateral ground bundles and from that through the inter-
olivary and median fillet.

In a recent article Mott and Tredgold[1] describe a well
marked atrophy at the periphery of the cord in the situation
of the antero-lateral tract, extending as low down as the first
lumbar segment; it is situated in the region of the descending
cerebellar path, but they did not think it due to involve-
ment of that path. The view they take is that the atrophy
in the descending path has its origin in the thalamic region,
the fibres passing through the middle fillet of the pons and
the interolivary lemniscus in the medulla, to reach the peri-
pheral part of the antero-lateral column of the cord. They
suggest, in view of their findings, the possibility of cerebellar
fibres to the cord passing by way of the basal ganglia,
possibly the optic thalamus. In connection with my case it is
interesting to note that there was undoubtedly distinct
atrophy of the right cerebellum, of the left thalamus and of
the middle fillet and interolivary lemniscus. I originally
accounted for this atrophy in the fillet as being associated
with the destruction of the cells in the nucleus gracilis and
cuneatus and consequent scarcity of the internal arcuate fibres
that go to form the interolivary lemniscus and middle fillet.
But I can see that it is possible, on account of the marked
atrophy of the above mentioned tract that part of the atrophy
may be due to the absence of fibres that go to form the spino-
thalamic tract mentioned by Mott and Tredgold.

In my case certainly there was not the marked atrophy
in the spinal cord as in Mott's case, nor could I trace it so far
down the cord. I think that in their case whatever atrophy or
alteration was present in the shape and size of the left half of
the cord might be accounted for by the unequal crossing of the
pyramidal tracts which was a marked feature in the case.
Dr. Mott mentions that it was not an area of sclerosis
which marked out the antero-lateral tract, but an absence
of tissue. I would urge that one has to be careful in pro-

nouncing this or that alteration in the shape of a cord which is abnormal in the crossing of its motor fibres, as being due to the absence of a tract not before pointed out. In the same paper they mention another case in which there was thrombosis at the commencement of the middle cerebral artery ; here definite degeneration was shown in the antero-lateral area of the cervical region. This case more nearly conforms to the findings under discussion. Why there should be atrophy of the *fasciculus cerebellaris ventralis* in mine and not in Mott's case, is, I think, in part due to the age attained by my patient (seventy-six), consequently a Gudden's atrophy (retrograde degeneration of different relays or series of stations) would have had time to take place ; or, in part, to the possibility that the atrophy was not apparent in Mott's case as he had excess of fibres in the ventral part of his cord due to the unequal crossing of the pyramidal tract.

It is worthy of note that in Wigglesworth's case (Brain, 1897, Plate III., Fig. ii.), on examination of the photographs of sections through the spinal cord in the upper cervical region, one can clearly see degenerated areas on both sides in the region of Gower's tract. No mention is made of this in the letterpress.

ON ATROPHY OF THE CEREBELLUM AND CEREBRUM.

Cerebellum : As noted, we found that the right half and vermis were markedly atrophied, the atrophy not being confined to any particular lobe. The dentate nucleus was smaller than the one in the opposite side and contained fewer cells. On microscopical examination the cells of Purkinje were lessened, the molecular layer greatly narrowed, the granular layer and larger fibres making up the medullary centre also greatly lessened. This atrophy of the right cerebellum is no doubt due to the connection between the cerebrum and the cerebellum having been interrupted, there being marked atrophy of the left thalamus, left red nucleus and superior cerebellar peduncle and cerebellum, and the continu-

ation of that from the cerebellum to cord and pons is shown by atrophy of the inferior restiform body and of the middle peduncle of the pons.

<div align="center">ON THE PEDUNCULAR TRACTS.</div>

Opinions are varied as to the origin of the different tracts which, along with the pyramidal, go to form the bundles in the *basis pedunculi.* As in this case we have marked destruction of the cortical tissue (the supposed origin of those fibres), and as further the fibres remaining in the basis pedunculi are very few, to that extent our case is in harmony with the view that the bundles of fibres in question are connected with the cortical area which here has undergone destruction.

In this connection it is perhaps worth while to note the various opinions brought forward as to the origin of the groups of fibres and their relationship to the cerebral cortex :

<div align="center">(A.) THE FRONTO-CEREBRO-CORTICO-PONTAL PATH.</div>

This as described by Flechsig is assumed by him to arise from the feet of the three frontal gyri, possibly also from the middle portion of the gyrus fornicatus. These fibres coming together pass through the pars frontalis of the internal capsule near the genu, pass thence from the base of the cerebral peduncle medially to the fibres of the pyramidal tract. In the pars basilaris pontis they occupy a dorso-medial position relative to the other longitudinal fibres, but in more caudal planes they turn ventralwards, and so come to lie ventrally and somewhat medially to the fibres of the pyramidal tract. Flechsig holds that this tract is concerned with the movements of bilaterally innervated muscles, such as those of the eye, the neck and the trunk.

Barker[39] has pointed out that this tract may degenerate after lesions of the middle and inferior frontal gyri. Von Monakow[40] also states that where we have a degeneration

backwards from this portion of the cortex down through the internal capsule, we obtain an atrophy of the radiation of the nucleus medialis thalami and of a portion of radiation of the nucleus lateralis thalami with subsequent disappearance of both of these nuclei. On the other hand Zacher[41] denies any connection between the frontal lobe and the median segment of the base of the cerebral peduncle; he concludes that the fibres, in part at least, have their origin in the Island of Reil; while Dejerine attributed the origin of the fibres to the Rolandic operculum.

In our case we have, it is true, the grey matter still remaining of the superior frontal convolutions, and of the gyrus fornicatus, as, also there is present the frontal portion of the internal capsule and that in a better developed condition and containing more fibres than does the occipital portion of the same. We have further fibres present in the most medial portion of the basis pedunculi and have a few bundles of longitudinal fibres in the pons, situated mesially and ventrally to the degenerated or atrophied bundles of the pyramidal tract; and these correspond fairly in number to the amount of the cortical area given for the origin of this tract. Thus our case, so far as it goes lends support to Flechsig's view, for, as already mentioned, Flechsig attributes the origin of these fibres to the ends of the frontal gyri and possibly also to the middle portion of the gyrus fornicatus, and this persistence in our case of at least a portion of the fronto-cerebro-cortico-pontal path is in harmony with such an origin.

(B.) THE TEMPORO-CEREBRO-CORTICO-PONTAL PATH.

Flechsig believes that the fibres of this tract arise from cells situated in the auditory sense area of the cerebral cortex (the superior and transverse temporal gyri), that these pass down the posterior portion of the pars occipitalis of the internal capsule to the lateral region of the base of the cerebral peduncle, and so to the pons where they lie dorso-laterally to the fasciculus cerebro-spinalis. The fibres of this and the preceding tract are medullated at a later period than the fibres of the pyramidal tract. Like the preceding tract this degenerates in a descending direction (Zacher,[42] von Monakow[43] and Dejerine.[44]

Dejerine states that the fibres mainly arise in the middle and inferior temporal convolutions and that they do not pass through the internal capsule but proceed below the lenticulate nucleus to the cerebral peduncle. Ferrier and Turner[49] have extirpated the superior temporal gyrus and have obtained degeneration in the lateral bundles of the pes. This observation supports the view of Flechsig, but like Dejerine they found that the fibres pass laterally and ventrally to the lenticulate nucleus and not through the internal capsule. Flechsig further believes that a certain number of lateral fibres in the lateral area of the crus (of the cerebral peduncle) may arise in the visual sense area of the occipital lobe and his views are supported by the morphological studies of Bechterew.

Zacher also holds this view and believes that the fibres from the occipital lobe are more laterally placed than those from the temporal lobe. In our case the superior and transverse temporal gyri were absent, and we were unable to find any fibres in this tract either in the internal capsule or at the cerebral or the caudal end of the pons. To this extent our case supports Flechsig. If, as Dejerine asserted, the bundles of this path arise from the whole temporal lobe, we should certainly have expected to find some of its fibres in the pons, for there still remained in our case portions of the second and third temporal convolutions.

AS TO THE CAUSE OF SPASM AND RIGIDITY.

The medical doctrine that was held, and is held to-day, by many, and I think erroneously, is that when we have complete transverse lesion of the cord above the lumbar enlargement, we have a condition of increased reflexes due to the inhibitory influence of the upper motor neurones being removed, or again, that in a case of hemorrhage in the internal capsule, followed by a descending degeneration in the motor tract, the presence of increased reflexes is due to the above named cause.

Dr. Charlton Bastian[51] was perhaps the first to cast doubt on this generally received opinion. In an article pub-

lished in 1890 he states that, with a total transverse lesion of
the cord of the lower cervical region, nearly all reflexes
dependent upon lower portions of the cord are abolished
rather than increased. He has also again shown more
recently, that, with transverse lesion of the cord above the
lumbar enlargement, there is a flaccidity of the legs and a
loss of the knee jerks.

Bowlby,[52] Thorburn[53] and Bruns[34] of Hanover have
confirmed Bastian's conclusions, so also has Ernest S.
Reynolds.[55]

Charcot held the theory that in ordinary hemiplegia
occurring in the adult the cause of the late rigidity is an
irritation of the motor tracts by the cicatrisation that occurs
at the seat of the lesion and extending along them. In our
case the sclerosis was quite in evidence, but in a large number
of reported cases the degree of sclerosis was very slight or
completely absent.

So far as it goes a study of our case would suggest that
the cerebellar hemisphere is involved in the production of the
spastic state. Here it may be noted that Dr. Hughlings
Jackson in 1877 formulated a theory and published an article
entitled, "On Certain Relations of the Cerebrum and Cere-
bellum, on Rigidity of Hemplegia and on Paralysis Agitans."
To quote what Dr. Jackson says :—He is "of the opinion
that the rigidity in hemiplegia is not owing to the cerebral
lesion nor to the lateral sclerosis ; there is evidently a duplex
symptomatic condition, negative and positive, with loss of
power over the muscles ; there is a tonic action in them with
loss of power. Whilst the primary cerebral lesion can account
for the paralytic element—negative condition—it cannot (nor
can the sclerosis in the lateral columns) account for the tonic
condition of the muscles—positive element. Negative states
of the nerve centres cannot cause positive states of muscles:
they may permit them."

His speculation was that rigidity is owing to an un-
antagonized influence of the cerebellum. He believed that,
in health, the whole of the muscles of the body are innervated
—innervated both by the cerebrum and cerebellum—there
being an antagonizing co-operation betwixt the two great
centres.

Some time after publishing those statements he went
half way towards abandoning the doctrine of cerebellar influx

because his critics pointed out that upon complete transverse lesion of the cord above the lumbar enlargement (whereby both cerebral and cerebellar influences are cut off from the motor centres below the transverse lesion), the knee jerks are accentuated, and that the legs are, or soon become, rigid. In consequence of Dr. Charlton Bastian's recent important researches on the effects of total transverse lesion of the supra lumbar cord, confirmed as they are by Bowlby, Thorburn and Bruns and others, demonstrating that a state occurs of flaccidity of the legs and loss of the knee jerks, and now admitted by nearly all medical neurologists to be correct. Dr. Hughlings Jackson has now reiterated his early theory.

In my patient there was extremely little spasm or rigidity unless in the hand, and to a slight extent in the foot. To account for this absence of spasticity, I am of the opinion that it was due to the atrophy of the right cerebellar hemisphere, in consequence of which the influx of stimuli from that organ must have been markedly lessened; hence the condition.

In some cases of porencephaly spasticity is a marked feature, in others the reverse is the case. On looking over the literature to observe whether it was possible to trace any relationship between involvement of the cerebellum or its absence, and absence or presence of spasticity, I found that so far it is not possible to arrive at any definite conclusion—so few of the case reports are complete. This, however, I may say, that I encountered no case in which there was both extensive atrophy of the cerebellum and presence of well marked spasticity. I would point out to future workers in this field that careful notes be taken of the extent of spasm during life and state of involvement of the cerebellum found post mortem.*

On the whole, therefore, I am inclined to believe that probably the reason why we have marked spasm in some cases and not in others, is due to the non-involvement or involvement, respectively, of the cerebellum.

I might here mention that in cases of cerebral diplegias, where pathological changes are confined to the cerebral cortex, the cerebellum being intact, a condition of generalized rigidity

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* Mott and Tredgold have pointed out that where the lesion affected primarily the optic thalamus or internal capsule with secondary atrophy of the cortical grey matter, they found that atrophy of the cerebellum of the opposite side occurred, but where the lesion was just confined to the cortex, and the basal ganglia intact, there was no atrophy of the cerebellum.

is a marked feature. So also in conditions of combined sclerosis, the presence of spasticity is an early sign in the majority of cases, later on a flaccid state and absence of knee jerks are characteristic. Two cases that were recently under my care which had the above clinical signs showed microscopically that the cerebellar tracts and cerebellar hemispheres were under certain late conditions of degeneration which might account for the flaccid condition. No evidence of degeneration of the lower motor neurones was to be seen.

SUMMARY.

Inasmuch as of necessity a very large number of points have been touched upon in the preceding study, it would be well if I attempted here to summarise my main findings :—

CLINICAL HISTORY :

History of a fright to mother of patient at about eighth month, with history of attempted abortion. Birth of patient normal in every way.

Rigidity and paresis first noted about second week. General backwardness of child in walking and talking. At age of twenty she was to all purposes a normal woman. Married twice, no family.

At or about age of sixty deterioration of mental faculties began to show themselves. Never had any history of fits of any kind. Had kyphotic curvature of dorsal vertebrae with slight dragging of right foot. Spasticity of little account save in arm and hand of right side.

PATHOLOGICAL :

A. *The Porencephalic Cavity and Hemispheres.* 1. A probable primary lesion of middle cerebral artery (thrombosis), the occlusion taking place immediately after the perforating arteries from the basal ganglia are given off. This lesion had apparently occurred in the later months of fœtal existence, and was due to rupture of fœtal vessels in the placenta. The lesion was followed by atrophy and destruction of a large section of the cerebral cortex on the left side in an

area supplied by the middle cerebral artery. The destruction was more extensive than was apparent at first sight, the cavity being in part filled in by the puckering and collapse of the surrounding tissue.

I am not inclined .ɔ believe that the atrophy of the vessels was secondary to the destruction of the cortex.

2. The porencephalic cavity thus found did not communicate with the lateral ventricles, but was bounded externally by the arachnoid, while the pia lined the sides and bottom of the cavity.

3. The existence of a general diffuse atrophy of the cerebral convolutions of the left side (but these were not simplified), with a marked lessening of the large pyramidal cells and of the projection system of fibres, especially so in what remained of the motor region; the association fibres apparently normal.

4. Right hemisphere normal in shape and structure with an increase in the large motor cells.

5. Symmetrical condition of the bones of the skull.

6. Dura not thickened or adherent.

7. The corpus callosum apparently normal in size.

B. *Basal Nuclei.* 8. The left caudate and lenticular nuclei normal in size and fully equal to their fellows of the opposite side.

9. General atrophy of the left optic thalamus, there being marked lessening in the number of cell bodies and of nerve fibres of the different nuclei present in that body.

10. Marked atrophy and absence of the middle and posterior portion of the internal capsule.

C. *Second Nerve.* 11. The left superior quadrigeminal body less in size than the one on the right.

12. Left inferior quadrigeminal body presented marked atrophy.

13. The brachium of the left inferior quadrigeminal body showed an almost complete absence of fibres.

14. The left corpus geniculatum mediale absent.

15. The left optic tract markedly atrophied.

16. The left optic nerve was found smaller in calibre than the right, certain bundles in the inferior portion being very markedly atrophied, and there was present an increased amount of glial tissue between all the bundles. The right

optic nerve showed atrophy of its central bundles with a general increase of glial tissue.

17. The red nucleus on the left side was markedly smaller than that on the right.

D. *Cerebellum.* 18. The left superior cerebellar peduncle above the decussation was smaller than the right, and the right was smaller in the pontal region.

19. Atrophy of the right dentate nucleus and right cerebellar hemisphere with lessening in the number of Purkinje cells, projection fibres and molecular layer; association fibres normal.

20. Marked atrophy of the lingula on the right side.

E. *Pes.* 21. In the basis pedunculi there was well marked atrophy of the pyramidal motor fibres of the left side.

22. Atrophy of fibres of the fronto-cerebro cortico-pontal path in the medial portion of the basis pedunculi. The fibres of the temporo-cortico-cerebro-pontal path atrophied in the lateral portion of the crus, and though not quite wanting here, wholly wanting in the upper part of the pons.

23. Locus niger on both sides seemed normal.

F. *Pons.* 24. Left middle and inferior cerebellar peduncles were much larger than those on the right.

25. A large number of pontal nuclei on the left side were absent, their place being taken by glial tissue.

26. The posterior longitudinal fasciculus is smaller on the left side.

27. The left olive was much contorted, but otherwise appeared normal.

28. The nucleus of origin of all cranial nerves on the floor of the fourth ventricle appeared normal.

29. The external arcuate fibres of both sides were well developed; marked hypertrophy of the arcuate nucleus on the left side.

G. *Fillet.* 30. Slight atrophy of right nucleus cuneatus, but more so of its internal nucleus; nucleus gracilis distinctly atrophied.

31. Marked lessening of internal arcuate fibres of the right side.

32. Marked lessening in size of the interolivary stratum of left side.

33. The lateral internal arcuate fibres were normal on both sides.

34. Marked contraction with sclerosis of the inner field of Flechsig on the left side seen throughout the medulla.

35. The left lateral fillet was markedly sclerosed and smaller than the right both in size and in number of nerve fibres present.

36. The nucleus of the lateral fillet on the left side was absent.

37. There was a sclerosis and absence of the nerve fibres forming the most medial portion of the left median fillet (motor). There was further a general sclerosis affecting the lateral part of the left mesial fillet, with associated lessening in size and number of the component fibres.

H. *Medulla.* 38. As under normal conditions, the lemniscus increased in size as it progressed upwards, owing to an accession of fibres from the sensory nuclei of the medulla.

39. In the left pyramidal tract in the medulla only a trace of fibres were to be seen.

40. In the right side of the pons and medulla there was present a sclerosed tract in the area usually occupied by Gowers' ascending fasciculus.

CORD:

I. *White Matter.* 41. The crossed pyramidal tract situated on the right side of the spinal cord was absent. Notwithstanding the long continuance of the condition there were still recognizable a most marked sclerosis in the region of the absent fibres of this tract.

42. The crossed pyramidal tract on the left side, while showing abundant well formed fibres, showed also some diffuse sclerosis.

43. The direct pyramidal tract on the left side was found almost completely absent.

44. Goll and Burdach's columns normal.

J. *Grey Matter.* 45. Clark's vesicular column normal.

46. The anterior horn (grey matter) of the right side was altered both in size and shape; the cell bodies were more numerous than in the left horns, but were of a lower type.

47. One group of cell bodies, the lateral and posterior external, were absent.

48. There was an abnormal arrangement of the nuclei of the back muscles (commissural group).

49. Degenerate tracts in the antero-lateral region of the cord on both sides, most marked in upper cervical region.

50. In this case we have a large number of superimposed neurones affected, numerically greater than in other cases described. This may be due to the age the patient attained (seventy-six).

I desire, in conclusion, to express my deep obligation to Dr. Adami for the valuable suggestions and advice freely tendered during the course of my study of the case, and during the preparation of this paper. I would also express my sincere thanks to Dr. C. F. Martin for relinquishing to me the full study of the case; to Dr. Patrick for his care in producing the photographs employed as illustrations; and again to Dr. Adami for aid in connection with the diagrams here given.

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