Clinical lectures on intra-cranial tumours. Lecture 2 / by Byrom Bramwell.

cyst were of considerable thickness (from one to two lines), but varying slightly in different parts. (Wishing to preserve the tumour entire, I did not examine the lining membrane.)

31*st* (*vespere*).—The child has cried less since the operation than it has done for some days, and has slept. Skin around the pedicle on the under side has given way, and the surface becoming purulent; the exposed part encroaching upon the canal of communication in the pedicle.

1*st January* 1881.—The child has remained in a torpid condition since last night, refusing the breast. The eyes are heavy, dim, and vacant; pupils slightly and equally contracted. Brow smooth; no crying. During the rest of the day the torpor deepened into coma, and after slight convulsions the infant died.

Post mortem examination not allowed, but I managed to pass a probe along the canal, now quite exposed below the base of the pedicle where the ligature had eaten through it, and found it to take an oblique course upwards and forwards, through the position which should have been occupied by the arch of the atlas, right into the cranial cavity, no force being used to make it take this direction.

I have communicated these notes, 1st, on account of the rarity of such cases; 2d, because of the treatment and its result. Repeated tapping of the cyst is a method which holds out no hope of cure, the result being to drain away the cerebro-spinal fluid, and merely prolong the lingering death. The only chance we can give the child is to remove the tumour early, taking precautions to keep the section strictly antiseptic, and to avoid that exposure of the canal of the pedicle which, in this case, was probably the cause of death by, *first*, allowing the cerebro-spinal fluid to drain away, and, *secondly*, permitting the absorption of septic or irritating matter into the cranial cavity. The operation has succeeded, as in the case operated on by Professor Annandale, quoted in Erichsen's *Surgery*.

ARTICLE X.—*Clinical Lectures on Intra-Cranial Tumours.* By BYROM BRAMWELL, M.D., F.R.C.P.E., Lecturer on the Principles and Practice of Medicine and on Practical Medicine and Medical Diagnosis in the Extra-Academical School of Medicine, Edinburgh; late Physician and Pathologist to the Newcastle-on-Tyne Infirmary; formerly Lecturer on Clinical Medicine and on Pathology in the University of Durham College of Medicine, Newcastle-on-Tyne, etc., etc.

LECTURE II.

IN my last lecture, gentlemen, I described to you the symptoms which are characteristic of cerebral tumours irrespective of their

position. To-day I propose to direct your attention to some symptoms which result from the presence of tumours in particular parts of the brain, or cranial cavity. These symptoms are commonly termed "localizing," because they serve to indicate the position of the growth.

Let us, then, in the first place, consider the *alterations in the motor nerve apparatus* which may be caused by the presence of an intra-cranial tumour.

Spasms and *paralyses*, the symptoms which result from alterations in the motor nerve supply of muscles, are of frequent occurrence, and, from an anatomical and physiological point of view, are the most interesting of all the symptoms.

An intra-cranial tumour may be regarded as a foreign body which, as soon as it attains to sufficient size, presses upon and injures nerve tissue.

The symptoms which result from the pressure of a tumour on *motor* nerve tissue depend upon—

1. The function of the particular portion of motor nerve tissue which is injured, *i.e.*, upon the position of the tumour.

2. The manner in which it is injured, viz., whether it is irritated, or destroyed.

3. (In the case of destroying lesions) the *rapidity* of the destruction.

1. *The Position of the Tumour.*—In order that you may thoroughly understand the important influence which the position of the tumour exercises upon the symptoms (the extent and distribution of the resulting spasm or paralysis), allow me very briefly to direct your attention to the arrangement of the motor nerve tract within the encephalon.

Recent researches, more particularly the clinical researches of Dr Hughlings Jackson on the human subject, and the experimental researches of Hitzig, Ferrier, and others on the lower animals, go to show, that the convolutions in the neighbourhood of the Fissure of Rolando (more especially the ascending frontal and ascending parietal convolutions) are concerned in the production of voluntary movements, and that individual movements and groups of movements are, *specially*, (observe, I do not say entirely) represented in particular portions or centres of this motor area. In this diagram, which is copied from Professor Ferrier's splendid work, *The Functions of the Brain*, you will see the exact position of these motor centres (see Fig. 1).

These centres functionate—*i.e.*, discharge or liberate motor nerve force—in obedience to the commands of the will. (The centre for the will has not as yet, and probably never will be exactly, localized. It is probably scattered over an extensive area of the cerebral cortex.)

From the motor cerebral cortex conducting fibres pass downwards, and convey the motor nerve force, which has been liberated by the generating centres, to the muscles on *the opposite side of the body.*

In their passage the conducting fibres form connexions with masses of gray matter in the cerebrum, pons, and medulla; the distribution and arrangement of which it is, for our present purpose, unnecessary to describe.

The conducting fibres first converge towards the internal capsule and lenticular nucleus of the corpus striatum (forming the corona radiata); pass through those structures; and form the descending fibres of the crus cerebri. This structure contains, therefore, all the motor fibres passing from the hemisphere on the same side, *i.e.*, all the motor fibres for the opposite side of the body. (Some authorities think that the movements of both sides of the body may be governed by either hemisphere. This is certainly true for the more automatic movements, and for the movements of the two sides which are in the habit of being performed in concert, such combined movements, for example, as those of the external rectus of one eye with the internal rectus of the other. The more highly specialized movements, *i.e.*, those of the upper extremity, are in all probability, governed by one, *i.e.*, the opposite, hemisphere.)

Below the crus cerebri the motor tract successively passes through the pons and medulla; and at the lower end of the medulla decussates in order to reach the opposite side of the spinal cord. (The whole of the pyramidal motor fibres do not decussate in the medulla. A small part passes down on the same side and forms the inner portion of the anterior column (see Fig. 2). The proportion of direct fibres passing down in the anterior column is usually, according to Flechsig, from 3 to 9 per cent. of the whole pyramidal tract.[1])

From the motor tract, as it passes through the pons and medulla, bundles of nerve fibres are successively given off, which cross the middle line, become connected with masses of gray matter (the trophic nerve nuclei), and then leave the nerve centres as the cranial motor nerves (see Fig. 3).

In the diagrams to which I now direct your attention the course of the motor tract and the position of the various nerve nuclei in the pons and medulla are shown (see Figs. 3 and 4). You will, by inspecting them, more readily understand that the position of the tumour must materially influence the character of the symptoms, *i.e.*, the extent and distribution of the spasms and paralysis. Where, for example, the tumour presses upon the crus cerebri (*cc* in Fig. 3) the paralysis will involve all the more highly specialized muscles (face, arm, and leg) of the opposite side. Where it presses upon a limited portion of the cerebral cortex—say the facial centre FC in figure 4—the resulting paralysis or spasm will be limited to the facial muscles. Where the lower part of the medulla (M in Fig. 3) is pressed upon, the facial muscles will escape, for the

[1] Abstract by Dr W. J. Dodds of Flechsig's article in the *Journal of Anatomy and Physiology*, vol. xiv.

facial nerve has passed out at a point above the level of the lesion.[1]

2. *The Manner in which Motor Nerve Tissue is injured, whether irritated, or destroyed, or both irritated and destroyed.*—It may be laid down as a general proposition that all nerve lesions produce one or other of two results, viz., either diminished or increased function (it would perhaps be better to say *perverted* function, for the increased function which results from pathological causes is never quite normal). We will, in the first place, consider the derangements which are manifested externally as increased (perverted) function.

Spasms (Convulsions).—The increased and perverted function which results from irritation (or loss of control) of motor nerve tissue by the pressure of a tumour is manifested externally as spasms and convulsions, and the lesion in such a case is said to be a "*discharging*" one. But in making use of this term I must beg of you to remember that it is not the tumour itself which discharges, but the motor nerve tissue which is irritated by it.[2]

The *character* of the convulsions, whether clonic or tonic, depends in part at least upon the nature of the nerve tissue which is irritated, whether gray matter or conducting fibres.

Clonic spasms (epileptiform convulsions) might theoretically be produced by irritation of any motor gray matter in the cerebrum, pons, or medulla. As a matter of fact, I believe they (clonic spasms) are generally due to discharge of motor centres in the *cerebral cortex*.

The extent and distribution of the spasms varies with the extent (and amount) of gray matter which is discharged. All degrees of clonic spasms (epileptiform convulsions), from the twitching of a single muscle or portion of a muscle to general (bilateral) epileptiform convulsions, are met with. Limited epileptiform convulsions are very characteristic of a "coarse" cortical lesion; and it is important to remember that, in such cases, the irritation has a great tendency to extend to and to involve adjacent and more distant centres, until, in some cases, the convulsions become general, and affect the muscles on both sides of the body.

The following case is a striking example of this fact:—

Case IV.—Mary C., æt. 37, a hawker, was admitted to this Infirmary, under my care, on 23d May 1875, suffering from right-sided convulsions and right-sided hemiplegia.

Some years previously she had received a violent blow from a

[1] The *exact* effects which a tumour causes in each of these situations will be afterwards described.

[2] An intra-cranial tumour can theoretically induce discharge of a motor centre in other ways than by direct irritation (pressure), viz., by (1) causing arrest or interference with the blood supply of that centre by (*a*) direct pressure on its nutrient vessels, (*b*) irritation of the vaso-motor nerves distributed to its nutrient vessels; or (2) by reflex irritation of the convulsive centre in the medulla.

poker on the left side of the head, which had fractured the skull. She was in consequence confined to bed for some time, but ultimately recovered, and, with the exception of "numbness" in the fingers and thumb of the right hand, and occasionally "twitchings" in the same parts, she had enjoyed good health until 10th May 1875, when the convulsions from which she was suffering at the date of her admission to hospital, commenced. The exciting cause of the attack seemed to have been a violent drinking-bout.

The *convulsions* were of three kinds—*slight, moderate,* and *severe.* In all the "march of the spasms" was the same.

In the *first* or *slight* form the muscles of the face and neck were alone affected.

In the *second* or *moderate* form the spasm commenced as before; first involved the muscles of the face and neck; then extended to the right hand, forearm, and arm; and finally to those of the right leg.

In the *third* or *severe* form the spasm commenced in the muscles of the face and neck (as in the first variety of fit); next involved the muscles of the right arm and leg (as in the second form), and finally extended to the muscles on the left side of the body.[1]

The patient died a week after admission.

On post-mortem examination I found that a lancet-shaped exostosis, about a quarter of an inch in length, which projected from the inner table of the skull, had caused a well-marked depression on the surface of the cerebrum. The exact position of the depression is shown in the diagram (see Fig. 5). It corresponds, you will perceive, to a point an inch and a half above the Fissure of Sylvius, and involves a part of the ascending parietal convolution.

[1] The exact sequence of the spasms was so important that I quote from my previous paper in detail :—

The right corner of the mouth was drawn down in tonic spasm, the platysma being rigid. The eyes were then partly opened, and the head and eyeballs rotated slowly to the right. Clonic spasms next occurred in both eyelids, the right being convulsed much more powerfully than the left; in the muscles of the tongue, right side of the face and neck, the platysma being chiefly affected. After a short interval the spasms became less frequent, the head and eyeballs were slowly turned back to the middle line; the eyelids were widely dilated, and the patient presented an animated appearance. The eyeballs were finally rotated upwards and to the left; the eyelids closed, and the patient apparently fell asleep.

In the *second* or *moderate* form the convulsions commenced as before. After the head had been rotated to the right, and as the clonic spasms were commencing, the fingers of the right hand were drawn in to the palm; the hand was then flexed at the wrist, and the forearm, bent to a right angle, placed across the chest. The muscles of the right leg at the same time became rigid, and the foot strongly inverted. Clonic spasms then occurred in the muscles of the arm and forearm, the flexors being more powerfully affected than the extensors. A few spasmodic twitchings were seen in the extensors of the leg and thigh. There was never any flexion of the hip or knee.

In the *third* variety the convulsions became general. The fit commenced as before, and passed through the various stages enumerated above. After flexion of the right forearm, the arm was slowly raised at the shoulder until

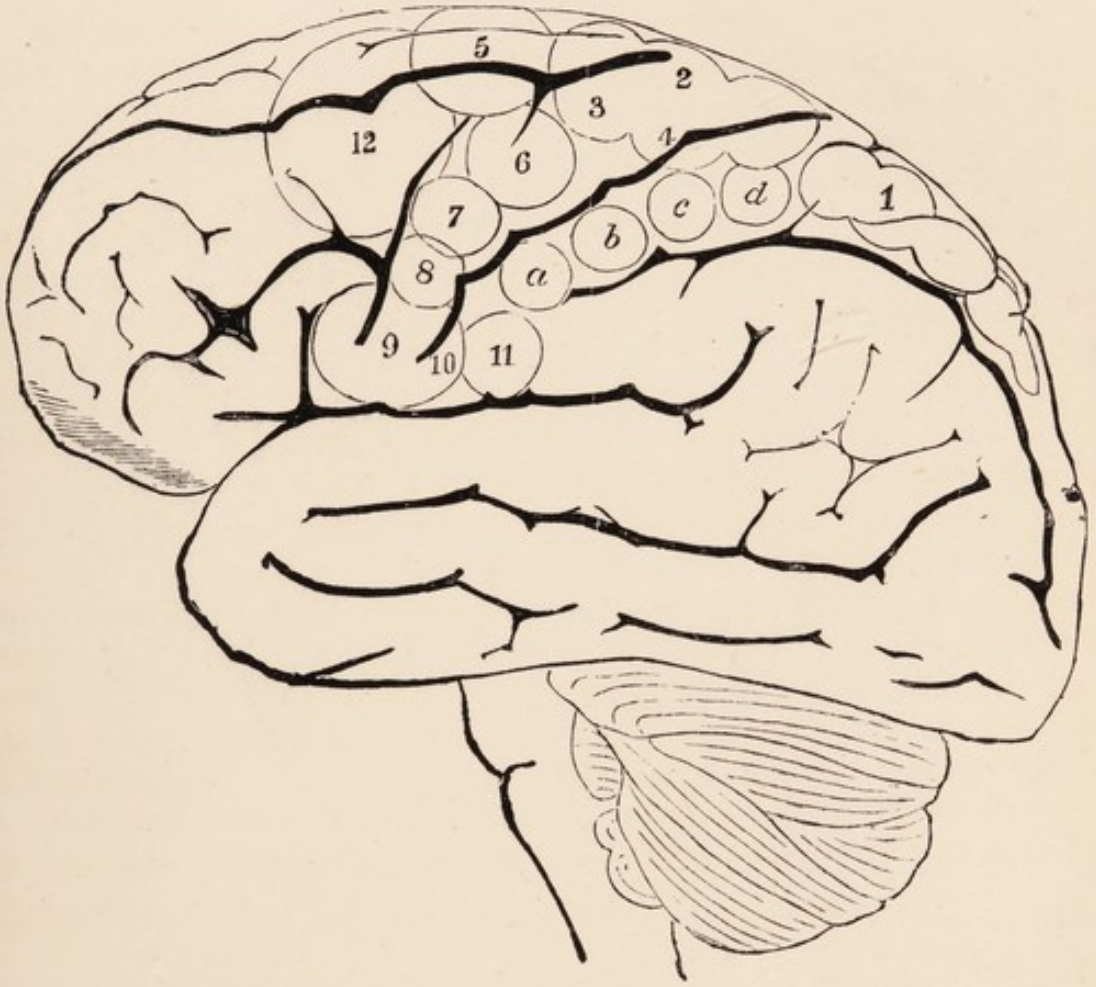

Fig. 1.—Lateral view of the human brain, showing the position of the motor centres. (After Ferrier.)

(1.) Centre for opposite leg and foot.

(2, 3, 4.) Centres for movements of arms and legs such as are concerned in climbing, swimming, etc.

(5.) Centre for extension forwards of the arm and hand.

(6.) Centre for the supination of the hand and flexion of the forearm.

(7 and 8.) Centres for elevators and depressors of mouth respectively.

(9 and 10.) Centre for the movements of the lips and tongue in articulation.

(11.) Centre for the platysma; retraction of the angle of the mouth.

(12) Centre for lateral movements of the head and eyes, with elevation of the eyelids and dilatation of the pupil.

(*a*, *b*, *c*, *d*.) Centre for the movements of the hand and wrist.

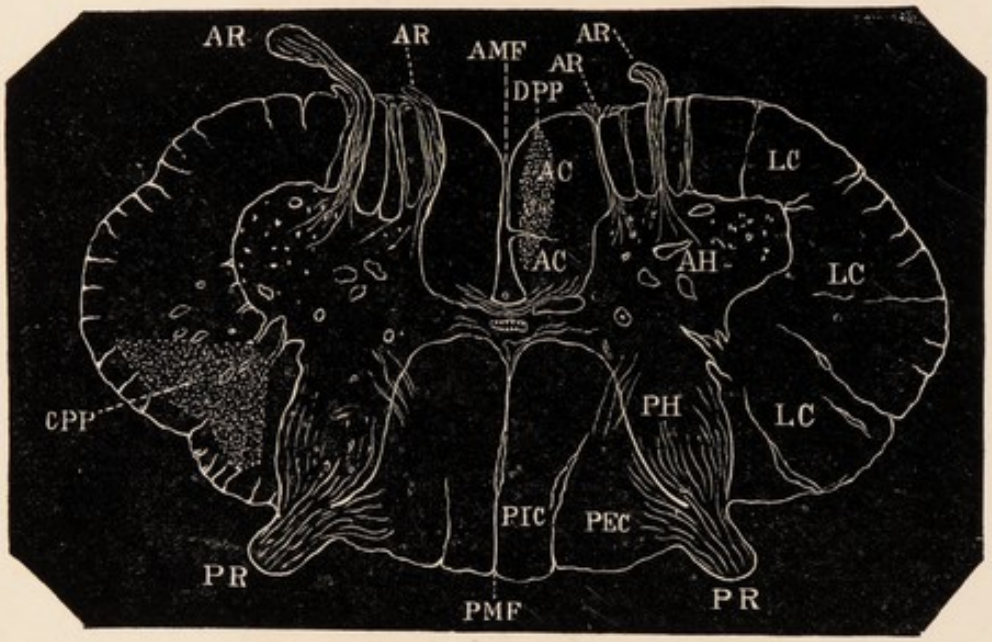

Fig. 2.—Transverse section of the spinal cord in the cervical region, showing the position of the degenerated fibres of the direct and crossed pyramidal tracts in a case of destruction of the right motor pyramidal tract above its decussation in the medulla. The greater part of the pyramidal fibres (crossed pyramidal tract), CPP, decussate in the medulla, and occupy the posterior part of the lateral column of the cord on the opposite side to the lesion. A small portion of the pyramidal fibres (direct pyramidal tract), DPP, do not decussate in the medulla, but pass down in the inner part of the anterior column of the cord on the same side as the brain lesion. CPP = Crossed pyramidal tract. DPP = Direct pyramidal tract. AC = Anterior column. LC = Lateral column. PEC = Postero-external column, or posterior root-zone of Charcot. PIC = Postero-internal column, or postero-median column, or column of Goll. AH = Anterior horn of gray matter. PH = Posterior horn of gray matter. AR = Anterior nerve roots emerging in separate bundles. PR = Posterior nerve roots entering in a single bundle. AMF = Anterior median fissure. PMF = Posterior median fissure.

Note.—The direct pyramidal tract (DPP) should come quite to the edge of the anterior median fissure, AMF.

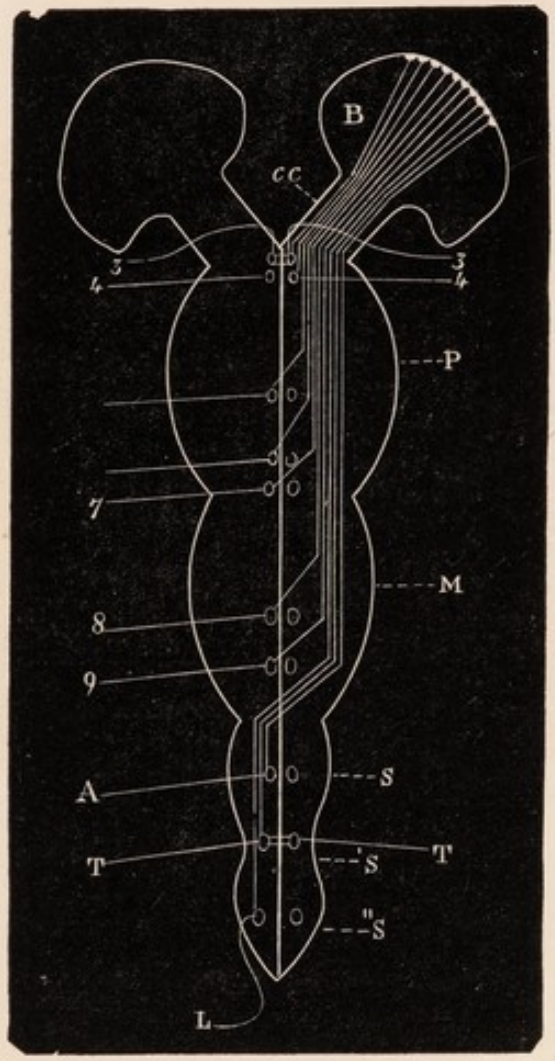

Fig 3.—Diagrammatic representation of the course of the chief motor fibres from the right hemisphere of the brain to the muscles on the left side of the body. The position of the nerve nuclei in the pons, medulla, and cord is shown. The trophic nerve nuclei of the third, fourth, and trunk muscles on the opposite sides of the body are connected, and can be thrown into action from either hemisphere. 3 = Third nerve (motor oculi). 4 = Fourth nerve (trochlear). 5 (number omitted by mistake in cut) = Motor division of the fifth nerve. 6 (number omitted by mistake in cut) = Sixth nerve (abducens). 7 = Portio dura or facial. 8 = Spinal accessory. 9 = Hypoglossal. B = Right hemisphere of brain. *cc* = Right crus cerebri. P = Pons. M = Medulla. S = Cervical portion of spinal cord. 'S = Dorsal portion of spinal cord. "S = Lumbar portion of spinal cord. A = Arm muscles. T = Trunk muscles. LM = Leg muscles.

Note.—The fibres passing from the brain, B, to the third and fourth nerves should decussate and join the nuclei on the opposite side of the middle line.

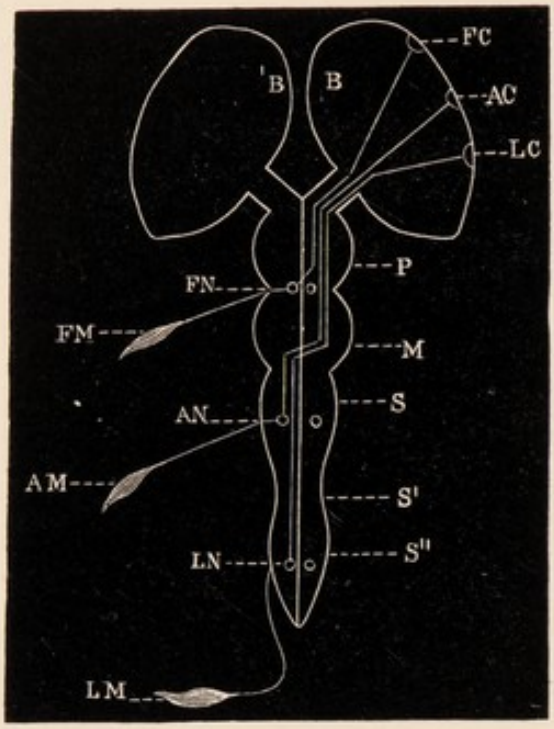

Fig. 4.—Diagrammatic representation of the course of the motor fibres to the right side of the face, right arm, and right leg. B = Right hemisphere of brain. FC = Facial centre in cortex. FN = Facial nerve nucleus. FM = Facial muscles. AC = Arm centre in cortex. AN = Trophic nerve nucleus for motor nerves supplying arm. LC = Leg centre in cortex. LN = Trophic nerve nucleus for motor nerves supplying leg. LM = Leg muscles. P = Pons varolii. M = Medulla oblongata. S = Cervical portion of spinal cord. S′ = Dorsal portion of spinal cord. S″ = Lumbar portion of spinal cord.

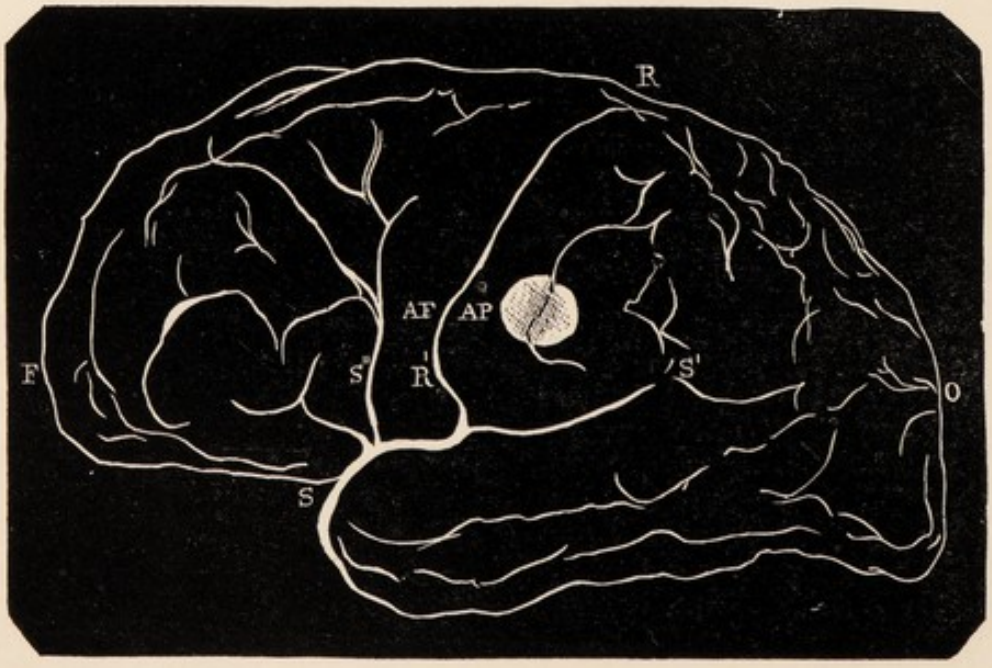

Fig. 5.—Outline of the left hemisphere of the brain in the case of M. C. The position of the lesion is shaded white. SS′ = Fissure of Sylvius. SS″ = Fissure of Sylvius (ascending limb). RR′ = Fissure of Rolando. AF = Ascending frontal convolution. AP = Ascending parietal convolution.

One striking feature of the limited or unilateral epileptiform convulsion which follows the irritation produced by a "coarse" cortical lesion, such as a tumour, and one which is an important distinction between spasms depending on this cause and the spasms which occur in so-called "idiopathic" or genuine epilepsy, is the fact that there is usually no loss of consciousness. When the spasms become bilateral, consciousness is generally lost, just as in an ordinary epileptic fit; but that this does not always occur, we have recently had an opportunity of proving.

Case V.—The patient, whom I now introduce to you, is a foreman bricklayer, æt. 35. He was first admitted to this Infirmary, under my care, on 4th November 1875, suffering from left hemiplegia. The paralysis was of five weeks' duration, and had followed a convulsion, in which the muscles of the left side of the body were alone affected, and in which consciousness was retained. There was a history of syphilis and of headache. Sight was normal, but, on ophthalmoscopic examination, both discs were seen to present the characteristic appearances of well-marked neuritis. These positive facts, together with the (youth) age of the patient, and the fact that there was neither cardiac nor renal disease, led me to conclude that the symptoms were due to a syphilitic tumour of the right cerebral hemisphere; while the fact that the paralysis had followed a unilateral convulsion, induced me to think that it (the paralysis) was functional epileptic in character. Under antisyphilitic treatment (full doses of iodide of potassium) rapid improvement took place; and on 31st July he discharged himself, saying that he was quite well. He continued to show himself occasionally, as an out-patient, until 22d April, when he returned to thank me, and to say that he was working regularly. I heard nothing more of him until the 23d of February last, when he was carried into the ward, at the time of my visit, in a fit; and I will now read you brief extracts from the notes which were taken at that time:—"His friends stated that he had continued perfectly well until 23d September 1876, when a scaffold, on which he and others were working, fell to the ground.

it was nearly at a right angle with the body. The tonic spasm then passed to the muscles of the left arm and of the left leg in the following order: the fingers of the left hand were first drawn in to the palm; the arm was then raised upwards and brought over to the right side, so that the hand approached the forehead; the left leg was at the same time flexed upon the abdomen, the knee being slightly bent, the toes spread out, and the foot flexed at the ankle-joint. The tonic spasms soon passed off. Clonic spasms of the muscles generally occurred: the patient foamed at the mouth, and made a cackling noise.

In the bilateral convulsions the muscles on the right side were always more powerfully affected than those on the left.

It is important to observe that the general (bilateral) convulsions only occurred occasionally, six or eight in the twenty-four hours, whereas the *slight* spasms occurred every few minutes, and the *moderate* ones frequently.

The case is reported in full in the *British Medical Journal*, 1st September 1877, page 290.

His right collar-bone was broken, and he received a severe cut on the back of the right side of the head. He was laid up from the effects of the accident for a month, and during the whole of that time suffered from intense headache. He then returned to work, and, notwithstanding the fact that he did not feel well, he continued to follow his employment until 9th February, when he took a fit. The convulsion commenced about 8 P.M., and lasted for five minutes. He had hardly come out of the first when he took a second; a few minutes after, a third; and he has continued to 'work in the fits' ever since. They have never left him, for more than five minutes at a time, during the whole fifteen days. He has been quite sensible during the whole time, and has been able to take nourishment and medicine. The left side, which has been much more powerfully convulsed than the right, became paralyzed after the first few fits."

Those of you who were present at the time will remember that the spasms, which commenced locally, soon became very violent; that both sides of the body, but especially the left, were convulsed; that the patient foamed at the mouth, and made a cackling noise; that, in short, the attack exactly resembled the convulsions which occur in ordinary (idiopathic) epilepsy; with the important exception that the patient was quite conscious throughout the fit. You will remember that we were at first disposed to doubt that such was the fact, but that after a long examination, and after having carefully tested the patient in various ways, we thoroughly satisfied ourselves that it was the case.[1]

Another characteristic of the limited epileptiform convulsions which result from the presence of a "coarse" lesion, such as a tumour, in the motor area of the cerebral cortex, is that the spasms are very often followed by temporary (so-called epileptiform) paralysis.

The case to which I have just referred (Case V.) is a good example of this fact. On both occasions, on which this patient came under my care, the left side of the body was paralyzed, and I beg you to observe that the paralysis *followed* the fit; and *affected those muscles which were convulsed.*

The cause of the paralysis in such cases was supposed by the late Dr Todd—and in this he is supported by the high authority of Dr Robertson of Glasgow and Dr Hughlings Jackson—to be the exhaustion of gray matter (motor nerve cells of the cortex) which follows the excessive and violent discharge which is manifested externally as spasms.

Supposing this explanation to be true, it is a remarkable fact that paralysis does not occur after the convulsions of the *idiopathic*

[1] The patient died on 31st December 1879, and on post-mortem examination a syphilitic growth was found in the posterior third of the first and second frontal convolutions on the right side. The case is reported in full in this Journal, January 1879, page 599.

disease (genuine epilepsy), in which the discharge, as measured by the severity of the spasms, is very violent. The duration of the discharge is perhaps greater in most cases of coarse lesion, the fits, as a rule, recurring more frequently than in the idiopathic form of the disease. This explanation will not, however, do for all cases, for I have seen temporary paralysis follow a fit (a *single* fit in a case of coarse lesion), which was by no means of longer duration nor of a greater severity than an average convulsion of the *idiopathic* disease. We are driven, therefore, to conclude that there must be some difference in the process of discharge in the two cases. Possibly the explanation is to be found in the seat of the gray matter involved. I must, however, confess that I am disposed to think that the motor cells of the cerebral cortex are affected in both cases.

Such cases as the above (in which epileptiform paralysis followed a single "fit") are exceptional, and may possibly be explained by supposing that the lesion causing the discharge produced, at the same time, some local structural change in addition to irritation of nerve cells; and that the additional local change—say œdema, for example—was the cause of the paralysis. The question naturally arises, whether such a structural change is not the cause of all cases of temporary epileptiform paralysis.[1]

[1] In the last number of "*Brain*," Dr Hughlings Jackson takes up the consideration of this question. He submits, page 445, that there is paralysis after epileptic (*i.e.*, after genuine epileptic) seizures, and that this paralysis is universally spread, and that it is owing to exhaustion of some nervous arrangements of the highest centres, and perhaps of lower ones too. He says, page 448, "I think, as a matter of observation, that, other things being equal, the more deliberately spasm sets in, and the more strongly it spreads, the more local it is, and also the longer it continues; and further, that the paralysis after such spasm is correspondingly more local, is greater in degree, and more persistent. If so, we may, on Todd and Robertson's hypothesis, say of the central process that the more deliberately the discharge begins, and the slower it is, the less widespread are the nerve currents developed, and the longer they continue; and that, consequently, the post-epileptiform exhaustion of centres is more local, more complete, and more persisting. On the other hand, the more suddenly spasm sets in, and the faster it begins to spread, the greater, I think, is the range it attains; and further, that the paralysis after it is more widespread, less in degree, and more transient. . . . As a matter of fact, I submit that the paralysis is really less in degree in the part first convulsed when the spreading of spasm is rapid and goes beyond the part, than when it is slow and is confined to that part. There is less spasm of the part first convulsed, that is, of the parts most specially represented by the cells discharging" (page 450). If the above reasoning be valid, we should expect, after sudden, rapid, short discharges, no decided local persisting paralysis, but slight, widespread, temporary paralysis, often so slight, so widespread, and so temporary, that it is ignored as paralysis and called prostration. If, to speak roughly, as much paralysis as makes one arm useless for four hours could be spread out thin all over the body for perhaps a quarter of an hour, it would not be recognised as paralysis at all, but as weakness, etc."—*Brain*, January 1881, p. 433. This explanation fails, I think, to account for the occurrence of paralysis in exceptional cases such as that to which I have referred. The duration of the discharge in that case was not longer than the

The practical outcome of all this is, that whenever you meet with a case of limited epileptiform convulsions which is unattended by loss of consciousness,—or whenever you meet with a case of epilepsy in which the spasms, which were at first limited and unattended with loss of consciousness, tend to spread to and affect other muscles, until finally the attack assumes the type of *true* epilepsy (bilateral epileptiform convulsions with loss of consciousness),—you should suspect the presence of an intra-cranial tumour, for you have to do with a discharging lesion of the motor cerebral cortex, and intra-cranial tumours are common causes of that condition.

Epileptiform convulsions of the ordinary type (ushered in with loss of consciousness and commencing as bilateral spasms) are also met with in some cases of intra-cranial tumour. They are of no importance in a localizing point of view, for they may result from the presence of a tumour in any part of the cranial cavity. General epileptiform convulsions of this kind are, however, by no means unimportant, for it not infrequently happens that a patient dies during such an attack. The following is a case in point:—

CASE VI.—An old woman, æt. 64, was admitted to this Infirmary, under my care, on the 28th September 1875, suffering from mitral stenosis and resulting dropsy. She was stupid, and unable to give any account of herself or her complaints. The people who brought her to the hospital stated that she had had "a stroke" three weeks previously, and had in consequence fallen down stairs and injured her left side. On examination I found that she was unable to stand, but that there was no definite paralysis. The chief nervous symptoms were headache, vomiting, deafness in the left ear, and occasional trembling fits, in which she complained of feeling

duration of the discharge in an average case of genuine epilepsy; and in genuine epilepsy the severity of the discharge of any given centre,—say the arm centre, for example,—as measured by the severity of the spasms, is, while it lasts, as great or even greater than the discharge in cases of local spasms. Granting, then, that the same nerve cells, *i.e.*, the motor nerve cells of the cortex, are discharged in the two cases (and we must, I think, grant that, wherever the discharge begins, it passes out through these motor nerve cells), it is difficult, I think, to explain the occurrence of paralysis in one case and its absence in the other, unless we suppose that there is some fundamental difference in the process of the discharge, or that some additional physical condition (such as œdema) capable of producing the paralysis is present in the one case and absent in the other. A more plausible explanation than that which the œdema theory affords is perhaps the following:—In the case of a coarse lesion the discharge is probably induced by direct irritation, leading to temporary increased production of nerve force, which, before it can escape, will have to overcome the (inhibitory) resistance, which, in such cases, is, we will presume, normal. In the case of genuine epilepsy, in which the discharge probably begins in the highest centre, the cause of the discharge is probably, as Dr Gowers suggests in his admirable *Gulstonian Lectures*, not excessive production of nerve force (in the motor cortical centre, *i.e.*, lower centres to those first discharged in genuine epilepsy), but diminished resistance; a greater amount of nerve force will therefore be required (to produce the same amount of spasm) in the case of a coarse lesion; hence the exhaustion of the nerve cells will be greater, and paralysis will result.

cold. The right optic disc was very much redder than the left, but there was no œdema. There was evidently some intra-cranial lesion, but an exact diagnosis was not ventured upon, as it did not seem justified by the facts. A month after admission she took an epileptic fit, and before the House-surgeon, who was immediately summoned, could reach the ward, she died. On post-mortem examination this beautiful little tumour, which I now show you, was found beneath the tentorium on the left side. It is, you will see, quite round, and about the size of a greengage plum. It springs from the dura mater, and has, you will perceive, made a deep indentation in the left lateral lobe of the cerebellum. In addition to this tumour, a small hæmorrhage, evidently of some weeks' duration, was found in the extra-ventricular portion of the left corpus striatum. The brain was otherwise normal.

The exact cause of death in this case was probably the sudden arrest of the functions of the "vital centres" (for the heart and respiration) in the medulla. In some other fatal cases of this description a profuse hæmorrhage takes place into or around the tumour.[1]

(*To be continued.*)

Part Second.

REVIEWS.

The Utricular Glands of the Uterus, and the Glandular Organ of New Formation which is developed during Pregnancy in the Uterus of the Mammalia, including the Human Species, etc., etc., etc. With a Quarto Atlas of fifteen Plates. Translated from the Italian under the direction of HENRY O. MAREY, A.M., M.D. Boston: Houghton, Osgood, & Company: 1880.

THE work before us contains the various papers hitherto published by Professor Ercolani on the anatomy of the placenta, and places before the English reader the peculiar but interesting opinions of that author in an exceedingly fascinating and agreeable form.

The fundamental ideas, which the author works out with much original research and argumentation, but also with no little repetition, may be formulated as two: 1st, That in all forms of placentation we have the co-aptation of two surfaces, the one of which, the maternal, is in its essence glandular, and secretes the

[1] A tumour which is not situated in the motor area may theoretically produce convulsions of this description, either by causing reflex irritation of the convulsive centre in the medulla, or by producing profound alterations of the intra-cranial circulation.

nutritive fluid; and the other the fœtal, which is absorbent, and, being always bathed in the nutritive fluid secreted by the maternal surface, constantly absorbs it for the benefit of the fœtus. The simplest placenta is thus formed on the type of the simple juxtaposition of an absorbent villus with a secreting villus. The secreting surface, however, by its arrangements usually encloses the absorbent within its folds, so as to form follicles, which include and invest the absorbent villi. 2d, The secreting surface, like the absorbent surface, is not formed out of any pre-existent tissues, but is a structure of new formation, which in the different classes of animals bears a varying relation to the original mucous membrane. This new structure is formed of elements possessing only a temporary vitality, and ready to be expelled or absorbed at the termination of the period of utero-gestation. In the formation of this new structure, or, as Ercolani calls it, glandular organ, the utricular glands take no share. The author proceeds, by the process of comparative anatomy, to demonstrate the truth of his views, beginning with the simplest forms of diffused and multiple placentæ, and passing on to the more complex single placenta in the dog, the quadrumana, and in man. The patience, care, and transparent honesty with which the author conducts this part of his work is beyond praise. It would lead us too far to go into details; but we may state that, in regard to the human placenta, Professor Ercolani holds that the new formation, at first distributed over the entire mucous membrane of the body of the uterus, becomes rapidly arrested in growth over the rest of the mucous membrane other than that of the serotinal area. There it continues to grow, and forms the maternal part of the placenta. This maternal part of the placenta or glandular organ, or, as it is usually called, the decidua serotina, in the woman, is formed, according to Ercolani, from the rapid proliferation of the cells of the submucous connective tissue. These cells, which on the uterine surface of the placenta are well known to anatomists as the large cells of the placenta, according to our author are the subjects of rapid transformation. By their vascularization they form the bloodvessels of the glandular organ; by becoming condensed they form into fibrous bands to attach it to the chorion and for the support of the villi. They also provide the bloodvessels with connective tissue investments, and, in truth, appear to make themselves generally useful where needed for any particular purpose, such as fixation, separation, or secretion.

The author traces in the human placenta development by successive stages that frequently correspond with the complete forms in other animals. The type of the absorbent villus is a loop of bloodvessels lying in a matrix of connective tissue, and covered with an epithelial lining of its own. In those placentæ in which the connexion between the absorbent surface and the secreting surface is not specially close, this epithelial layer remains on the