THE GOULSTONIAN LECTURES ON THE LOCALISATION OF CEREBRAL DISEASE.

Delivered at the Royal College of Physicians of London.

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LECTURE II.—March 20th.

A.—LESIONS OF THE MOTOR REGIONS.

THE motor area, as determined by experiments on monkeys, includes the bases of the three frontal convolutions, with those bounding the fissure of Rolando; viz., the *ascending frontal*, or *anterior central* (Ecker); the *ascending parietal*, or *posterior central* (Ecker), with its superior continuation, termed the *postero-parietal* or *superior parietal* (Ecker) lobule; together with the internal aspect of the same, which by our French brethren is generally called the *paracentral* lobule. (See fig. 1.) The homologous regions of the brain of the monkey and man are sufficiently evident not to require my taking up valuable time with any detailed anatomical description. In this region are situated certain definable areas stimulation of which by the electric current gives rise to certain definite movements on the opposite side—viz., of the leg and hand, facial, oral, and lingual muscles; and destruction of which causes paralysis of all those movements (*exc. exc.*) if the entire region be destroyed; limited or dissociated paralysis, if individual areas only be destroyed—the paralysis in this case being confined to those movements which are excited by irritation of the same. This region is supplied by the middle cerebral or Sylvian artery by four or five branches, each of which nourishes a special area. The artery may be occluded in its main trunk or in its individual branches; and the arrangement is such, according to the researches of Duret and Heubner, that the arteries of the basal ganglia may still remain pervious though the cortical arteries are occluded by emboli. Hence it is that softening limited to the cortical grey matter and subjacent medulla may and does frequently occur, while the basal ganglia remain absolutely intact.

The lesions which invade this region we may divide into *destructive* and *irritative*, in accordance with their main symptomatic characters. But a complete practical separation is not always possible, inasmuch as lesions which result ultimately in total disintegration are not unfrequently associated at times with irritative or convulsive phenomena limited or more or less generalised: a combination which may be likened to that in *anæsthesia dolorosa*.

We have now a very considerable body of evidence to show that destructive lesions of this region invariably cause paralysis of voluntary motion; and that there is a differentiation in this part of the cortex of distinct centres, corresponding in situation to those experimentally established in the brain of the monkey, destructive lesions of which cause limited or dissociated paralysis of their respective movements or muscular combinations. It is not maintained, however, that in all cases of purely cortical paralysis anatomical lesions are demonstrable in these centres; but I am unable, after much investigation, to find any satisfactory evidence of the occurrence of a destructive lesion here not associated with motor paralysis. I have met with occasional statements or hypotheses as to what may or must have been, but no proof of such a lesion having actually existed without paralysis.

Truly, Samt* has recorded a case in which a cyst was found on the cortex in the motor zone; but, as Charcot and Pitres remark,† the actual destruction of the grey matter was not proved, since we know that tumours may press aside, without destroying, the tissue on which they rest. Let the subject, however, be investigated anew, with all the most modern methods, and with the utmost possible scrutiny. When a clear case of destructive lesion of the cortex in this region without motor paralysis is forthcoming, it will be time to cast aside the immense body of positive experimental and clinical evidence which we possess in favour of the thesis enunciated.

The experimental evidence in the case of monkeys is of the following nature. The brain is exposed. The application of that which is universally recognised as an excitant of nerves and nerve-centres to a certain spot is followed by a definite movement. All other conditions remaining the same, this spot is destroyed by the cautery (universally recognised as a destroyer of animal tissues), and immediately we see paralysis of the muscles formerly thrown into action by the electrical stimulus. I am describing no hypothetical experiment, but one actually performed and frequently varied. If motor function is ascribed to a nerve because on irritation the muscle contracts, and on section the muscle is paralysed, I cannot see why motor function should not be predicated of the cortical centre, seeing that the phenomena are essentially the same.

Brown-Séquard, however, thinks that, in all cases of paralysis from cortical lesion, there is some intermediate link or *tertium quid* intervening between the antecedent and the consequent: a kind of inhibitory influence exerted by the lesion on some centre or centres which are credited with the functions which are lost. It would, I think, be easy by parity of reasoning—and I say it with all due respect to the distinguished author of this theory—to make a complete *reductio ad absurdum* of the whole of experimental physiology. We should never be entitled to establish direct relationship between organ and function, but be condemned to a perpetual search after some *tertium quid*, which, like an *ignis fatuus*, would for ever elude our grasp.

It is generally considered that explanation is satisfied when we assimilate one fact or set of facts to another familiar fact or set of facts; and, if we are entitled to call a nerve motor because motion ceases when it is cut, so, it seems to me, we may call a part of the brain motor if a similar result invariably follow on its destruction.

I. *Destructive Lesions of the Motor Area.*—I will first direct your attention to destructive lesions of the motor area, and under this head consider (1) general and (2) partial lesions.

1. *General or Extensive Lesions.*—The type of general lesion of the cortical area in the monkey is paralysis of voluntary motion without affection of sensation on the opposite side of the body, and possessing all those features which characterise ordinary cerebral paralysis (*hémiplegie centrale vulgaire*, Charcot); viz., hemiplegia which, though at first absolute, gradually subsides into a condition in which there is complete paralysis of all the most volitional movements, while associated, alternating, or bilateral movements are more or less spared. Hence the hand is more paralysed than the arm, the arm more than the leg, and the lower facial movements more than the upper; while the muscles of the trunk are scarcely, if at all, affected.

Fig. 7.

In fig. 7 is a representation of the extent of a lesion of the brain of the monkey causing complete hemiplegia of the opposite side. This was a case of encephalitis resulting from exposure, in which the phenomena in the early inflammatory stage were localised spasms on the opposite side, which gradually became general, followed by complete paralysis and flaccidity, without loss of sensation, when softening was complete.

Fig. 8 is a representation of a lesion causing* paralysis of the arm and leg of the opposite side; and fig. 9 (f) represents a lesion in a region stimulation of which caused supination and flexion of the forearm. The result was paralysis of voluntary motion limited to this movement. These will suffice to indicate the difference between general and partial lesions of the cortex under circumstances free from all com-

* *Archiv für Psychiatrie*, 1874.
† *Revue Mensuelle*, 1877.

* Also temporary blindness of opposite eye, which will be explained below in connection with lesions of the angular gyrus.

plication. The lesions of disease, however, are, as a rule, rarely so simple. I have already indicated the forms of disease which can scarcely be admitted as relevant in relation to the question of localisation, and I would further add that considerable caution requires to be

Fig. 8.

exercised in reference to traumatic lesions. These are most valuable in a negative point of view, as, for instance, in the case of the frontal lobes; but, when accompanied by positive symptoms, it is necessary that lesions of the base of the brain, which Duret shows may result from concussion and are apt to complicate the symptomatology of the local injury, should be shown to be absent.

Fig. 9.

Apart from traumatic lesions, the cases of cortical disease which I proceed to quote clearly establish the fact that lesions in that part of the human brain which corresponds to the area termed motor in the brain of the monkey produce paralysis of voluntary motion on the opposite side of the body: a hemiplegia like that resulting from destructive lesion of the corpus striatum, or more particularly of the anterior part of the internal capsule (*hémiplegie centrale vulgaire*). This paralysis is frequently associated with rigidity or convulsive spasms in the paralysed parts, particularly in the early stage; and, if destruction of the cortical substance be complete, the paralysis is of permanent duration, and sooner or later is followed by late rigidity and secondary sclerosis of the motor tracts. This degeneration is traceable in the medullary fibres, crus cerebri, pons, and pyramid of the medulla oblongata on the side of the lesion; and thence mainly on the opposite side of the spinal cord in the posterior part of the lateral column (*faisceaux encéph. externes ou croisés*, Bouchard; *faisceaux latéraux*, Charcot; *Pyramiden Seitenstrangbahnen*, Flechsig); while a corresponding band of secondary degeneration frequently exists on the internal aspect of the anterior column of the same side as the lesion (*faisceaux encéph. directes ou internes*, Bouchard; *faisceaux de Türck*, Charcot; *Pyramiden Vorderstrangbahnen*, Flechsig). These are the tracts which recent researches, to which I have before alluded, have shown to be the continuations of the pyramidal strands; and the fact of the direct continuity of this degeneration with that commencing under the cortex proves, in accordance with Waller's and Türck's researches, the direct motor significance of the cortical regions to which these fibres are traced.

I have already mentioned the experiments of Albertoni and Michieli, and those of Franck and Pitres, showing that secondary degeneration occurs in the medullary fibres of the centrum ovale after destruction of the cortex in dogs; and I may also quote the experiments of Vulpian,*

* *Archiv. de Physiologie*, 1876.

and those of Carville and Duret, establishing the occurrence of secondary degeneration in the spinal cord after removal of the limb-centres—sigmoid gyrus—in this animal.

A case is reported by Gliky* of unilateral convulsions of the left side followed by complete hemiplegia without loss of sensation, in which, after death, a caseous degeneration was found involving the ascending frontal and bases of the three frontal convolutions, the ascending parietal and postero parietal lobule, and the corresponding internal aspect of this region, or paracentral lobule. (See figs. 10 and 11.)

Fig. 10.

Lepinet† has recorded a case of right hemiplegia, without affection of sensation, of six years' duration, caused by yellow softening of the ascending parietal convolution in its whole extent, with partial affection of the ascending frontal, posterior digitations of the island of Reil, and

Fig. 11.

anterior part of the superior and inferior parietal lobule of the left hemisphere. The ganglia were intact. Secondary degeneration was traced in the left side of the pons Varolii and left pyramid.‡

In a case communicated by M. Brun to MM. Charcot and Pitres,§ left hemiplegia without aphasia had existed for four years, with late rigidity of the arm, and in a slighter degree of the leg. Yellow softening (*plaque jaune*) was found in the lower two-thirds of the ascending frontal, lower half of the ascending parietal, the posterior three-fourths

* *Deutsches Archiv für Klin. Medizin*, December 1875.

† *Localisations dans les Maladies Cérébrales*. Thèse d'Agrégation, 1875.

‡ A mistake occurs in the account given of this case, which has been copied by MM. Charcot and Pitres, (*op. cit.*, p. 123), the atrophy being described as being on the right of the pons instead of the left. This is corrected by M. Lepine's own hand in the copy which I possess.

§ *Op. cit.*, p. 121.

of the *second and third frontal*, and the whole of the convolutions of the island of Reil in the right hemisphere. (Fig. 12.) The ganglia

Fig. 12

were intact. No examination, however, was made in reference to secondary sclerosis in this case.

Another case is given by the same authors* of right hemiplegia, with aphasia, of one year's duration, and accompanied by late rigidity of both limbs, more particularly of the arm. A patch of yellow softening was found involving the whole of the *ascending frontal* and base of the *third frontal* convolution, the whole of the *ascending parietal*, together with the inferior parietal lobule and two posterior digitations of the island of Reil in the left hemisphere. The ganglia were normal. Secondary degeneration was evident in the crus, pons, and pyramid on the same side; but the cord was not examined.

Trousseau quotes a case which occurred in the *clinique* of M. Charcot,† in which secondary degeneration was traced in the crus, pons, and pyramid of the same side as the lesion, and in the opposite side of the spinal cord; the lesion being, softening of the *ascending frontal*, the *three frontal convolutions*, and of the island of Reil in the right hemisphere. The case was one of left hemiplegia of three months' duration; death occurring from pneumonia.

These cases, out of many on record,‡ are sufficient to show that, from purely cortical lesions, permanent paralysis may result; that, in consequence of such lesions, secondary degeneration takes place in the motor tracts, with its accompaniment late rigidity.

In the cases I have quoted, there has been more or less complete hemiplegia. In some of these, the lesion has invaded the whole of the motor area, and it only; in others, the lesion of the motor area, though extensive, has not been anatomically coextensive with this area; and in most there has also been affection of parts not considered as belonging to the motor area. Clinical cases in which a lesion should be extensive enough to involve the whole motor area, and it alone, must naturally be considered as quite exceptional. But it is not at all difficult, by a process of exclusion, to eliminate those regions, a lesion of which does not cause paralysis or secondary degeneration; and it may be stated provisionally (of which proofs will be adduced subsequently) that it is only in cases of lesion of the motor area, as above defined, that secondary degeneration occurs, and only in these that paralysis is invariably the result.

I will return to the fact of complete hemiplegia, with comparatively restricted lesions of the motor area.

But before doing so, I would call your attention to the researches of M. Pitres, who, in his recent work,§ has collected a large body of evidence to show that what is true of the lesions of the cortical substance holds also in respect to lesions in corresponding parts of the centrum ovale; a term which he extends to the whole of the medullary substance intervening between the cortex and the basal ganglia. In all future observations and pathological records, it would be advisable to follow M. Pitres' system of nomenclature of the divisions of the centrum ovale. (See plate I in M. Pitres' work.) A vertical section of the hemisphere at right angles to the long axis in the prefrontal

region gives the *prefrontal section*. The next section, carried two centimètres in advance of the fissure of Rolando, passes through the bases of the three frontal convolutions and forms the *pediculo-frontal section*, divided into a *superior*, *middle*, and *inferior pediculo-frontal fasciculus*, corresponding with the respective frontal convolutions. The next section is the *frontal*, formed by dividing the hemisphere parallel to the fissure of Rolando through the ascending frontal convolution. Here also there are three divisions—the *superior*, *middle*, and *inferior frontal fasciculi*. (In this section, the sphenoidal region is partly shown.) Next is the *parietal section*, carried in a similar manner through the ascending parietal convolutions. Three segments are also seen here; viz., the *superior*, *middle*, and *inferior parietal fasciculi*. Next is the *pediculo-parietal section*, made by dividing the hemisphere three centimètres (1.18 inches) posterior to the fissure of Rolando, and cutting the superior and inferior parietal lobules. Here we distinguish a corresponding *superior* and *inferior pediculo-parietal fasciculus*. Below is the *sphenoidal fasciculus*. Section of the occipital lobe gives the *occipital section*, in which no separate fasciculi are differentiated.

Now, it is only in certain of the medullary fasciculi so marked out, that lesions cause paralysis of motion and degeneration of the motor tracts. These regions are included in the *pediculo frontal*, *frontal*, and *parietal sections*, or, generally, in the *fronto-parietal area*. Lesions here have exactly the same effect as lesions of the corresponding cortical region, according as they are destructive or irritative, or according as they are limited or general. And M. Pitres has brought forward evidence of a very satisfactory kind to show (p. 100 *et seq.*) that the early rigidity and muscular spasms, which so frequently accompany hemiplegia with effusion into the lateral ventricles, are essentially due to irritation of the fronto-parietal fasciculi of the centrum ovale.

As to the occurrence of hemiplegia with recent hæmorrhage into the fronto-parietal region of the centrum ovale, without direct affection of the cortex or basal ganglia, I might quote many cases; but, though the explanation is, through other facts, rendered simple enough, they may be objected to as evidence of paralysis from direct lesion of this area. For it might be said, and not without reason, that the paralysis was due to indirect affection, by pressure, etc., of the motor ganglia and tracts.

It will be better, therefore, to restrict ourselves to cases in which this element of suddenness or of pressure is entirely absent, *i.e.*, cases in which there was mere softening or solution of continuity of the medullary fibres.

Hodgson* has related a case of right hemiplegia with aphasia, followed several months after the seizure with late rigidity of the right arm. Death occurred from chronic bronchitis more than a year after the attack. In the centrum ovale of the left hemisphere, was a cavity nearly empty, one inch and a quarter long, situated external to the lateral ventricle and between its anterior horn and the island of Reil. The rest of the brain, except the part immediately around the cavity (which was yellowish), was normal.

A similar case of right hemiplegia, but without aphasia, was recently brought before the Société Anatomique† by Landouzy. In this case, there was late rigidity and atrophy of the paralysed limbs. A slough formed in the sacrum, and death resulted six months after the attack. The lesion was a lacuna or cicatrix resulting from a hæmorrhagic effusion, situated in the centrum ovale, and extending from the base of the first frontal to the postero-parietal lobule. The form was irregularly triangular, the widest part situated posteriorly. Secondary degeneration was visible in the left crus and right side of the spinal cord, but the exact extent of the degeneration had not been minutely examined at the time the record was made.

For other similar instances, I would refer to M. Pitres' above-mentioned work.

2. *Partial Lesions of the Motor Area.*—a. *With Hemiplegia.* It would seem that, in order to cause more or less complete hemiplegia of the opposite side, it is not absolutely necessary that the anatomical lesions should be co-extensive with the whole motor area. I have already given one or two instances in which the lesion, though extensive, did not involve the whole of this region; to these I add one or two instances of hemiplegia in which the lesion was still more limited anatomically than in those already mentioned. But the area of anatomically demonstrated lesion is not necessarily the area of functional disturbance; and it is this element of uncertainty which, as I have before indicated, renders conclusions as to exact localisation, from a purely clinical point of view, always more or less doubtful.

There is, of course, no difficulty in accounting for complete hemiplegia in connection with very limited lesions of a sudden character in

* *Op. cit.*, p. 121, *et seq.*

† Charcot and Pitres, *op. cit.*, p. 123.

‡ The reader is referred to the works, among others, of Cotard (*Atrophie Partielle du Cerveau*, 1868; and Landouzy (*Convulsions et Paralyxies liées aux lésions des parties Fronto-Pariétales*, 1876) for many similar cases.

§ *Lésions du Centre Ovale*. Paris, 1877.

* *Lancet*, 1866, vol. i, p. 397.

† October 1877. *Progress Médical*, December 29th, 1877.

the motor area, such as hæmorrhagic extravasation or a traumatic lesion. In time, however, the symptoms generally disappear, with the exception of those attributable to the part immediately destroyed or irritated. But there are on record a few cases in which, though the lesions were of a more chronic nature and apparently anatomically circumscribed, a more extensive paralysis has existed than can be accounted for by the amount of cortex visibly destroyed.

MM. Charcot and Pitres* give a case of right hemiplegia, without aphasia, but with facial paresis and late rigidity of the limbs. The lesion was situated in the lower two-thirds of the ascending parietal

Fig. 13.

convolution of the left hemisphere (fig. 13). Secondary sclerosis existed in the motor tracts.

In another,† of right hemiplegia, without aphasia, and rigidity of the limbs (the paralysis alternating with unilateral convulsions), the chief lesion occupied the left paracentral lobule (fig. 14), which was

Fig. 14.

softened and atrophied. The lesion also involved the anterior third of the quadrilateral lobule and the upper extremity of the ascending frontal convolutions. In this case there were also other indications of degeneration in different parts of the hemispheres. Secondary degeneration of the motor tracts existed.

A similar case has been also reported by Pitres.‡

The first of these cases is the most difficult to account for, if the lesion were actually confined to the region indicated. The other two are more in accordance with the usual results of lesion situated at the upper extremity of the fissure of Rolando.

b. *Partial Lesions and Monoplegia.*—I will now proceed to lay before you some of the more carefully recorded clinical cases of limited lesions of the cortex with correspondingly limited paralysis or monoplegia of the movements governed by these parts respectively; and I hope to be able to show you that the situation of the various motor centres in the human brain closely corresponds with the position I have assigned to them on grounds of experiment and anatomical homology. But I would repeat that the observations on record are not yet sufficiently

numerous to establish by themselves, apart from the precise facts of physiological experiment, the exact situation and limits of these centres. With these facts, however, the case is altogether altered.

Unilateral Oculo-motor Monoplegia.—At the base of the first frontal, and extending partly into the second frontal convolution in the brain of the monkey, there is an area ([12] figs. 15 and 16) irritation of

Fig. 15.

which causes elevation of the eyelids, dilatation of the pupil, conjugate deviation of the eyes, and turning of the head to the opposite side. I have placed these various reactions in the order in which they occur with slight and longer continued stimulation respectively. In the faintest form of stimulation, elevation of the eyelids is the only effect observable. Whether individual centres, incapable of sharp demarcation from each other for each of the movements indicated, exist here, has not been determined experimentally.

Fig. 16.

There are, however, clinical cases on record tending to show that there must be a distinct centre for the levator palpebræ superioris, inasmuch as paralysis may occur, limited to this muscle without affecting the other muscles supplied by the third nerve; an occurrence difficult to explain by peripheral affection of this nerve. Some such cases have been observed in connection with disease of the cortex, and attempts have been made, but not, I think, successful or in accordance with experimental lesions, to localise this centre in the angular gyrus.* If such a centre really exist, I should be inclined to look for it in the

* *Revue Mensuelle*, 1877, p. 191.

† *Op. cit.*, 1877, p. 193.

‡ *Le Progrès Médical*, September 10th, 1876.

* Landouzy, "Blépharoptose Cérébrale"; *Archiv. Gén. de Méd.*, August 1877.

region of which I am speaking. Still there are difficulties in the way; for affections of this region are not uncommon, and paralysis of the levator palpebræ superioris without other oculo-motor paralysis is not so, or at least has not been often observed. Hence, if the centre of this movement exist here, its escape from paralysis needs explanation. Whether this can be found in the intimate bilateral association of the oculo-motor nuclei or not, is a question on which, as we have so little to go on, I forbear at present to speculate further.

In the case, however, of the bilateral and antagonistic movements of the head and eyes, it would be natural to look for lateral distortion should the centre in one hemisphere be suddenly removed; and the distortion would be towards the side of lesion, owing to the unantagonised action of the centre which remains. This affords a sufficiently satisfactory explanation of the conjugate deviation of the head and eyes observed in the early stages of hemiplegia, whether of cortical or ganglionic origin.

Certain facts, however, exist tending to show that this lateral movement of the eyes may be paralysed singly, or thrown into spasm singly, constituting what may be termed unilateral oculo-motor monoplegia or monospasm. This, even in the absence of a necropsy, would strongly point to cortical lesion, or lesion of the medullary fibres between the centre and the corpus striatum. It is only in such conditions that dissociated paralysis or spasm finds a satisfactory explanation.

This form of paralysis or spasm is by no means common—I find only three cases on record—and the actual *post mortem* evidence is still more scanty. Two such cases have been recorded by Priestley Smith.* In one of these cases, the eyes were directed to the right, and the patient, though he possessed the convergent movements and power of accommodation, could not voluntarily move his eyes to the left. In the second, the eyes were also turned to the right, and inability to look to the left was also observed, as in the other.

Priestley Smith thinks that these cases are to be explained by either irritation of the opposite or destructive lesion on the same side of the centre I am describing. In this he is followed by Hughlings Jackson,† and with these views, I have to express myself as being in complete accordance. So far, they are perhaps speculative, as recovery took place in both cases.

The following case, however, related by Chouppe,‡ which is quoted by Landouzy,§ supports this view by *post mortem* evidence. This was a case of a lad, aged 19, who showed symptoms of tubercular meningitis, in which, in addition to pain, vomiting, etc., the most remarkable symptom was a rotation of the head and eyes to the right. This could be overcome with moderate effort, but the head and eyes returned to their position when left to themselves. No other paralysis or contracture existed elsewhere. After death, a patch of disease, free from granulations and quite superficial, of the size of a franc piece, was found on the "superior part of the middle frontal convolution" in the left hemisphere. Other lesions were found in the superior and lateral part of the sphenoidal lobe of the right hemisphere, but, as will be subsequently shown, these cannot be regarded as much complicating the case. There was no other cerebral lesion. The exact situation of the lesion in the left hemisphere is not indicated more precisely than in the words quoted; but they may, I think, be taken in support of the theory that these special symptoms were due to irritative lesion of that which corresponds with the oculo-motor centre in the brain of the monkey.

Crural Monoplegia.—As there seems to be some misapprehension abroad in reference to the centres of movement of the hinder extremity, let me first call your attention to the facts of experiment. Irritation of the *postero-parietal* or *superior parietal lobule*—area 1—(fig. 15) causes flexion of the foot in the ankle, occasionally combined with flexion of the thigh on the pelvis, and extension forward of the leg as in the act of walking.

On the other hand, in my experiments on monkeys, stimulation of area 2 (fig. 15), a region which includes the upper extremity of the ascending frontal, as well as part of the ascending parietal, gave rise to more complex movements, the result of which was to bring the foot towards the middle line of the trunk, as if the animal were to scratch its abdomen or lay hold of something in this position. The area 3, near it, gave rise to movements of the tail. Below it (4), certain movements of the arm—viz., adduction and retraction—were excited.

It is a question by no means easy to answer *à priori*, how far the movements of the human leg can be compared with those of the leg-arm and foot-hand of the monkey; or what is the representative in man of the centre for the tail which, in the New World monkeys, plays the part of a hand. We must, therefore, be cautious in drawing

conclusions as to the exact position of the arm and leg centres in man from considerations merely of anatomical homology. And there is reason for exact and careful analysis of the movements which are affected, or more particularly affected, in any given case of crural monoplegia of cortical origin, for on this may depend the exact regional diagnosis.

Clinical evidence in favour of a distinct centre or centres for the leg, clearly differentiated from those of the arm, is not as yet very extensive. We have many cases on record in which the leg and arm have been paralysed together—brachio-crural monoplegia—an association easily accounted for by the close relation of the leg and arm centres to each other. Still, there are some cases in which the leg only has been paralysed; and in others, in which leg and arm have been ultimately conjointly affected, the paralysis has shown itself first in the leg. This latter fact has an important bearing on the question of a distinct leg-centre, and its exact situation. A few cases are on record of paralysis occurring in one or both legs from injury to the vertex in the parietal region; but we cannot exercise too much caution in the inferences we draw as to the seat of lesion in such cases. The researches of Duret* have shown that local and general spinal paralyses may result from bulbar and spinal lesions owing to sudden displacement downwards through the aqueduct of Sylvius of the cerebro-spinal fluid in consequence of blows on the head. A case in which, from the symptoms, this was evidently the mode of causation, is related by Guthrie.† A soldier at Waterloo was struck by a shot which caused depressed fracture of both parietal bones. On his recovering consciousness, he was found paralysed in both legs, and benumbed from the loins downwards. He recovered on being trephined ten days afterwards. It cannot be doubted that the paralysis in this case was spinal paraplegia, and easily accounted for in the manner demonstrated by Duret.

In some other cases, however, the lesion may have been cerebral; the fact of the paralysis being crossed would support this view. Hitzig‡ quotes from Löffler the following among others. A Danish corporal was struck by a shot at the *superior and posterior extremity of the left parietal bone*, close to the sagittal suture (overlying the postero-parietal lobule, see fig. 3). The right leg was immediately paralysed. The right arm became affected on the seventh day. On trephining, recovery took place—the arm first, and then the leg. This may fairly be taken as a cortical lesion, as the subsequent affection of the arm is in accordance with extension of softening to neighbouring centres; a feature so characteristic of cortical lesions. Another case of fracture of the "summit of the right parietal bone" by a shot was followed by paralysis of the left leg.

As cases of disease of the cortex with crural monoplegia, the following two (though they are not altogether precise) may be taken, quoted from Landouzy.§ One is reported by Becquerel, of paralysis limited to the left leg. The lesion in this case is described as being situated "at the upper part of the right hemisphere", consisting of granulations and adhesions (p. 211). Slight affections of the pia mater and subjacent cortex were, however, also observed in the left hemisphere. The second is recorded by Rendu. This was a case of paralysis of the right leg followed by paresis of the right arm, which gradually increased. In the *parietal convolutions* of the left hemisphere, *close to the longitudinal fissure*, was a zone of exudation and interstitial hæmorrhages, affecting both the cortex and medullary substance to a considerable depth, but not extending to the basal ganglia. In the parietal convolution (exact position not stated) was found a caseous nodule of the size of a nut. It is, however, to be noted that the cerebral membranes generally in both hemispheres indicated signs of tubercular meningitis. Hence, the case is not quite free from complication.

I may here mention a case which, though one of tumour, has a considerable value in reference to the question before us. The particulars were privately communicated to me by Dr. Haddon of Manchester, and the question arose during life as to the exact locality of the tumour and the advisability of trephining. Though our diagnosis proved absolutely correct, the operation, for other reasons, was not performed. The patient began first to have tingling in the left leg, followed by paresis of this limb, gradually increasing, and continuing restricted to the left leg for five months. Then the left arm became weak. After occasional attacks of rigidity and twitching, the arm and leg became both completely paralysed. Shortly before death, signs of weakness showed themselves also in the right leg. After death, a tumour, three inches in diameter, was found growing from the dura mater, and pressing perpendicularly downwards on the region included in the *upper extremity*

* "Bilateral Deviations of the Eyes"; *Birmingham Medical Review*, 1875.

† "Ophthalmology in Relation to General Medicine"; *Lancet*, May 12th, p. 676.

‡ *Bulletin de la Soc. Anat.*, 1871, p. 380.

§ *Op. cit.*, p. 160.

* *Traumatismes Cérébraux*. Thèse (unpublished), 1878.

† Quoted by Wilks, *Guy's Hospital Reports*, 1866, p. 57.

‡ *Untersuchungen über das Gehirn*, 1874.

§ *Op. cit.*, pp. 211-212.

of the *ascending frontal*, *ascending parietal*, and *postero-parietal* convolutions. The tumour had grown downwards as far as the floor of the lateral ventricle, compressing the brain-substance in its course. It also impinged on the other hemisphere. The point of origin of this tumour, and the mode of growth, warrant the conclusion that the paresis of the left leg, which was the first and for a long time the only motor symptom, was due to the affection of the cortex in the region where experiments in the monkey would lead us to localise the leg-centres.

I will mention, lastly, two cases reported by Bourneville, which, though not uncomplicated cases of crural monoplegia, yet deserve to be taken in consideration with the others. They might also be referred to as instances of partial hemiplegic epilepsy. The first of these* is quoted by Charcot and Pitres. This was the case of a girl, aged 18, who had been affected with infantile hemiplegia of the left side at the age of four. She was subject to epileptic attacks, beginning in the left leg, which was paralysed. There was no facial paralysis. After death, a patch of degeneration was found in the right hemisphere, occupying the upper half of the ascending frontal, the bases of the first and second frontal, the anterior part of the postero-parietal lobule, and the whole of the internal aspect of these regions (fig. 17). Secondary

Fig. 17.

degeneration of the motor tracts of the spinal cord existed. The second case was a similar one of infantile hemiplegia, followed by partial epilepsy; the spasms being limited to the right leg, but gradually invading the right arm and the right side of the face. The position of the lesion, diagnosed during life, was the *upper extremity* of the (one *cm.* in extent) *ascending frontal and parietal convolutions*, and the internal surface corresponding, or paracentral lobule. This region was the seat of softening. No other lesion existed. In this case also, secondary degeneration was found in the right half of the spinal cord, in the postero-lateral column.

These cases, I think, suffice to prove that the lesions which have been described in connection with crural monoplegia very closely agree with the position of the centres for the movements of the leg in monkeys. In the absence of other cases of lesion serving to determine the exact position and extent of the leg-centre, M. Bourdon† has endeavoured to arrive at this by another method. The method he adopts is to examine the brain in cases of amputation or arrested development of the lower extremity, with the expectation of finding atrophy of the corresponding centre, in accordance with Luys' views. Whether these expectations are altogether justifiable may be questioned.

But, theoretical considerations apart, the practical difficulties in the way of a satisfactory solution of this problem are very great, and I do not think that, up to the present at least, they have been disposed of successfully. An important communication on this subject was recently presented to the Société de Biologie (January 5th, 1878) by M. Féré, well known for his valuable researches in cerebral topography. M. Féré shows that neither the method of ascertaining the existence of atrophy, by comparing the relative size of homologous convolutions in each hemisphere, nor that founded on the advance or recession of the fissure of Rolando (round which the motor centres are grouped) is reliable; inasmuch as in the perfectly normal brain there are great variations and want of symmetry in the convolutions and fissures in the two hemispheres, and there is no absolutely constant relation between the position of the fissure of Rolando and a certain cranial region, or in its position in the one hemisphere as compared with the other. Hence

the necessity of receiving all statements with regard to atrophy with extreme caution, unless substantiated by a thorough microscopical investigation of the parts said to be so affected.

With these considerations, I will now give the results of M. Bourdon's analysis of cases of amputation. In one of amputation of the thigh, thirty-five years before death, atrophy was said to exist at the upper extremity of the ascending frontal, at its junction with first frontal convolution of the opposite hemisphere. In a second, of amputation of the thigh, fifty-two years before death, atrophy was said to exist in exactly the same situation. In a third, of amputation of the thigh, twenty-one years before death, a linear depression was found interrupting the continuity of the second frontal convolution in the opposite hemisphere.

To these cases, which M. Bourdon quotes from M. Luys, a fourth is added, observed by M. Landouzy. This was a case of arrested development of the right leg at the age of eighteen months, death occurring at the age of forty-five. The left hemisphere was smaller than the right, and the fissures of Rolando were unsymmetrical. The left ascending parietal convolution appeared somewhat smaller than the same convolution in the right hemisphere. There was also asymmetry of the pons and medulla, the left side being the smaller.

None of these cases appear to have been investigated microscopically. If we regard them as satisfactory, which I think we are scarcely entitled to do, we do not find any more exact correspondence as regards the situation of the leg-centre, than in the cases of lesion already mentioned. I think, however, that, though the exact limits are not defined, the evidence points to a position closely in harmony with that defined in the brain of the monkey; and that the conclusion formed by M. Lucas-Championnière is well justified, viz., that, to expose the leg-centre, it is necessary to apply the crown of the trephine over the upper extremity of the fissure of Rolando.

[To be continued.]

CLINICAL OBSERVATIONS

ON A

FAILURE IN THE ANTISEPTIC MANAGEMENT OF A CASE OF EMPYEMA.

By JOHN DUNCAN,

Surgeon to the Royal Infirmary, Edinburgh.

GENTLEMEN,—On November 20th, 1877, Dr. Balfour sent to us for operation a patient who had been admitted to the medical house, suffering from empyema. The vicissitudes in her history since she came under our observation deserve your attentive consideration.

Four years ago, she had a severe attack of pleurisy, from which she never completely recovered; but she dates her present illness from exposure to cold six months before admission. Pus was three times drawn from the chest by means of a Bowditch's syringe while she was under Dr. Balfour's care; but the relief was only of temporary duration. On November 23rd, I made a free incision between the sixth and seventh ribs in the axillary line, and evacuated a quantity of pus, which my house-surgeon (Mr. Black), partly from measurement, estimated at more than three hundred ounces. Those of you who were present will remember the extraordinary force with which the matter was projected the moment the cavity was opened. For the next month, the progress of the case was all that could be desired. The temperature, which, before the operation, had varied between 99 deg. and 101 deg., fell to normal, and the highest reading recorded is 98.8 deg. The discharge rapidly diminished until only about a tablespoonful daily (probably less) of sero-purulent fluid came away by the drainage-tube.

On December 23rd, we dressed the wound under the spray from a new steam spray-producer, which we afterwards found had been discharging a carbolic solution of about one to a thousand, on account of the imperfect boring of the nozzle. You may recollect that I then speculated on the probable consequences, and it is certain that my fears were speedily realised. Next evening, the temperature stood at 100.6 deg.; and perhaps I cannot give you a more graphic description of the condition of affairs than by reading to you the morning and evening temperatures of the next few days.

				M.				E.
December	24th	...	...	98.4	...	...	...	100.6
"	25th	...	...	100.6	...	...	...	102.2
"	26th	...	...	100.8	...	...	...	103.2
"	27th	...	...	100.9	...	...	...	104.3
"	28th	...	...	102.4	...	...	...	102.4

* Gazette Médicale de Paris, December 1876.

† Recherches Cliniques sur les Centres Moteurs des Membres. Paris, 1877.