THE GOULSTONIAN LECTURES ON THE LOCALISATION OF CEREBRAL DISEASE.

Delivered at the Royal College of Physicians of London.

By DAVID FERRIER, M.D., F.R.S., F.R.C.P.,

Professor of Forensic Medicine in King's College; Assistant-Physician to King's College Hospital; etc.

LECTURE III.—March 22nd.

II. *Irritative Lesions of the Motor Area.*—Hitherto I have directed your attention more particularly to destructive lesions of the motor area indicated by paralysis of voluntary motion, general or partial, theoretically separating them from irritative lesions indicated by unilateral convulsions or monospasms. Practically, however, they cannot be separated from each other by a hard and fast line; for many cases of limited destructive lesions are associated with unilateral convulsions or monospasms, and I have already referred to instances of this association. There are numerous cases, however, in which the predominant, and sometimes the only, feature has been a convulsive affection limited to a limb or combined in the way in which monoplegiæ are combined, or which, commencing in a constant and definite way, becomes generalised into an unilateral convulsion with or without loss of consciousness, or occasionally passes into bilateral convulsions. It is the great merit of Hughlings Jackson to have first clearly indicated the pathology of these affections.

I have already alluded to some of the observations and generalisations which had been made by Bravais, Bright, Wilks, etc.; and I ought to mention here the very close approximation to regional localisation of the lesions most commonly causing convulsive affections of the opposite side at which Mr. Callender* arrived. Callender concluded that convulsions occurred more particularly with superficial lesions of the cortex in the neighbourhood of the middle meningeal artery: an approximation amply borne out by subsequent clinical and experimental research.

From the mere occurrence of an unilateral convulsion with loss of consciousness, or of an epileptic attack in which the convulsions are exhibited mainly on one side, we can form no opinion as to the nature or seat of the lesion. We may suppose, and with reason, that the opposite cerebral hemisphere is more particularly at fault; but there need be no discoverable lesion; and, should a lesion exist, it need not be in any definite position. This is in accordance both with clinical facts and also with those of experiment, for I have found that long continued irritation applied to any part of the hemisphere other than the motor area may result in an attack of unilateral convulsions. If, however, the convulsion be of the character of a monospasm, or if, tending to become generalised, it begin invariably in the same way and do not cause loss of consciousness, and if it be followed by paresis or paralysis more or less permanent, we may diagnose an irritative lesion of the motor area of the opposite hemisphere.

The seat of the lesion may be approximately determined by the rules as to the localisation of destructive lesions, but only approximately, as the diagnosis of the seat of an irritative lesion is naturally more uncertain than that of a destructive lesion, owing to the difficulty of determining the extent of the zone or the special point in this zone in which vital irritation concentrates itself. Yet many cases are on record in which the phenomena of irritation have been such as to allow exact regional diagnosis in accordance with the principles of localisation of distinct centres.

The morbid anatomy of irritative lesions is various. They are all such as tend to induce irritability and hyperæmia of the cortical grey matter or subjacent medullary tracts: a condition which I have invariably observed in my own experiments. Magnan† has found the same condition of hyperæmia of the cortex in the epileptic convulsions induced by the introduction of absinthe into the system; and MM. Pitres and Franck‡ have shown that, in the partial or unilateral epileptiform attacks to which dogs are liable after injury to the cortex, the grey

matter surrounding the cicatrix is so hyperæmic, tumefied, and irritable, that even mechanical stimulation, which normally has no effect, is sufficient to cause motor discharge.

The irritative lesion may, therefore, be an acute inflammation, or a condition of irritability set up by some chronic morbid product. Most frequently the lesion is some form of meningo-encephalitis; and of these syphilitic disease is by far the most common, so that now syphilitic epilepsy is sometimes spoken of as synonymous with "Jacksonian epilepsy". Tubercular affections, tumours or cysts situated superficially, cicatrices of old wounds, spicula of bone, etc., are all capable of producing irritation.

Hughlings Jackson is of opinion that the lesion causes the centres to become charged to a state of high tension, so that, under certain vital conditions, they discharge themselves in a sudden and explosive manner, and become exhausted for the time being; hence the temporary epileptic hemi- or monoplegia. This would account for the occurrence of intermittent effects with a constant lesion; for, according to the law of discharge formulated by G. H. Lewes,* stimulations which fall short of actual discharge of a nerve-centre increase the tension; hence, after a certain accumulation of stimuli, sudden discharge is readily induced.

Frequently the disease, which begins as an irritative lesion, tends to invade and destroy the region on which it grows, and leads to permanent paralysis with secondary degeneration; irritative phenomena being liable to occur so long as the vitality of the grey matter and subjacent medullary fibres has not been absolutely annihilated.

"Jacksonian epilepsy", in the early stages at least, has frequently the character of a monospasm, which may be brachial, facial, or crural, or these combined, as in the manner of monoplegiæ. When the monospasm tends to become generalised into unilateral convulsion, the spasms seem to march usually in a certain definite order. In facial monospasm, the arm next becomes affected, and then the leg. If it commence in the hand, it goes up the arm, then to the face, and next attacks the leg. If it begin in the leg, it next invades the arm, and then the face. This order is very rarely inverted. Usually, when the convulsions have become unilateral, consciousness is lost, if not before. When the convulsion becomes bilateral, as is sometimes the case, Hughlings Jackson finds that the spasms march in the reverse order; e.g., if it have passed to the leg from the face, it ascends the opposite leg, and so to the arm and face. I have not confirmed this in experiments on animals, for I have frequently seen the same order followed on the other side as on that on which the spasms began. It would be in accordance with the bilateral association of motor nuclei (which Dr. Broadbent has applied so successfully to the explanation of the comparative escape of bilaterally associated movements in cerebral disease), that bilaterally associated movements should have most tendency to be discharged together. This fact has been well brought out by MM. Franck and Pitres, for they have shown that bilateral convulsions may still occur from excessive irritation of one hemisphere even when the motor centres of the other have been extirpated.

And many clinical cases might be cited which do not harmonise with the views of Hughlings Jackson as to the march of bilateral spasms.† It is not, however, my intention to discuss at length the pathology and symptomatology of irritative lesions: subjects which are well known to the profession in this country through the writings of Hughlings Jackson, and abroad more particularly through the writings of M. Charcot. I will content myself with calling attention to one or two of the more accurately recorded cases of circumscribed lesions with irritative symptoms, in order to show how closely the situation of irritative lesions (notwithstanding all the elements of uncertainty pertaining to them, as compared with destructive lesions) may be determined from the symptoms manifested; and, as I have already intimated, I allude only to those in which irritation or spasm was the predominant or only symptom, and only to those verified by *post mortem* examination, though I might justifiably cite others of the same kind, depending particularly on syphilis, which recover.

I. *Crural Monospasm or Protospasm.*—Of spasms limited to the leg, or invariably commencing in the leg, there are not many cases on record free from complication with paralysis, or in which the lesion remained circumscribed till death. I have already quoted two cases from Bourneville, in which crural monospasm complicated with paralysis was the chief symptom. A case is recorded by Broca‡ of crural monospasm cured by injury to the left side of the skull which was

* *Physical Basis of Mind*, p. 290.

† See Gowers's "Cases of Convulsion from Organic Brain-Disease" (BRIT. MED. JOURNAL, Sept. 26th, 1874); "Case of Intracranial Tumour", by Bramwell (BRIT. MED. JOURNAL, Sept. 1st, 1877).

‡ Soc. de Chirurgie, meeting of December 16th, 1866.

* *St. Bartholomew's Hospital Reports*, 1867; *Medico-Chirurgical Trans.*, 1871.

† *Recherches sur les Centres Nerveux*, 1876, p. 101.

‡ Soc. de Biologie, meeting of Dec. 29th, 1877; *Le Prog. Méd.*, Jan. 5th, 1878.

caused by trephining; but the exact position of the lesion I do not find recorded.

MM. Charcot and Pitres* quote a case from Griesinger of spasm of the leg frequently recurring, and also invading the arm, followed in the intervals by paralysis of the leg and arm. The lesion, however, was not strictly limited in this case. A hydatid cyst, 4 centimetres $\times$ 4.3 centimetres, was found on the surface of the opposite hemisphere, in such a position that its anterior border corresponded with a line drawn perpendicularly upwards from the external auditory meatus, *i. e.*, about the upper extremity of the fissure of Rolando. There were also several smaller cysts on the frontal and parietal surface of the hemisphere. If the spasm can be ascribed exclusively to the large cyst, then its position agrees with the motor centres of the leg. (Fig. 15, [1] [2].)

Hughlings Jackson† has described a case in which fits began almost invariably in the right leg, and were frequently limited to it. The leg began to become weak, and more so after each fit, the paresis deepening ultimately into a permanent paralysis. In the last stages, signs of general affection of the left hemisphere—aphasia, etc.—manifested themselves. A tumour was found at the upper posterior part of the left frontal lobe, about two inches in diameter, bounded posteriorly by the fissure of Rolando, and extending forward into the posterior part of the first and second frontal convolutions.

Another case is given by Hughlings Jackson‡ of convulsions beginning in the left great toe, often confined exclusively to the left leg, and followed ultimately by paresis of the left foot. This patient also had paralysis of the right third nerve. After death, a syphilitic lesion was found "at the upper part of the posterior ascending or ascending parietal convolution, extending over part of the upper end of the ascending frontal and over several of the adjacent convolutions of the parietal lobule" of the right hemisphere. On the right third nerve, a tumour of the size of a pea was found. This case is in exact correspondence with the situation which I have assigned to the motor centres of the foot and leg. (Fig. 15, [1] [2].)

2. *Brachial Monospasm or Protospasm.*—Of spasms limited to, or beginning in, the arm or hand, depending on localised cortical lesion, there are several cases on record. As a rule, the fits begin in the fingers, and more especially in the thumb and index finger—in the most volitional movements of the upper extremity, according to Hughlings Jackson; but this is not necessarily or invariably so. In the upper extremity, it must be remembered, there are several combinations of movements which have each a representative in the cortical motor area. These centres being all situated within a moderate compass, and all liable to be discharged by one irritative lesion, it is nevertheless possible that each may be the primary origin of the discharge, and so the mode in which the monospasm commences may vary accordingly. Hence the necessity of making minute investigation of the march of the spasm in any particular case.

Hughlings Jackson has recorded several cases of brachial monospasm. I will only mention those in which the lesion of the cortex was single and limited. A man had frequent convulsions limited to the right arm, which subsequently became partially paralysed. A nodule was found situated at the hinder extremity of the first frontal convolution of the left hemisphere. In this case, there was also a tumour in each lobe of the cerebellum, but there were no cerebellar symptoms. The march of the spasm was not recorded.§

In a second case of convulsions, nearly always limited to the right arm, and followed by temporary paralysis of that arm after each fit, the lesion, which was diagnosed by Hughlings Jackson during life, was a nodule, situated at the "posterior extremity of the first frontal convolution where it joins the ascending frontal". In this case, it was noted that the spasms always began in the shoulder and went down the arm, contrary to the usual order.|| This is an important case, as showing that the spasm began in muscles which, as the experiments on monkeys indicate, relate to the movement of the arm as a whole, and not to those of the fingers or wrist. (Fig. 15, [3] [4] [5].)

In a third case,¶ convulsions invariably began in the left thumb. After death, a tumour of the size of a hazel-nut was found under the grey matter at the posterior extremity of the third frontal convolution of the right hemisphere. Some granulations existed in the bed from which it was enucleated, or in the grey matter near it.

In a fourth case, the spasms began in the right hand, and occasionally in the right cheek. Before death, left hemiplegia came on,

which, however, soon passed off. Disease was found in both cerebral hemispheres, probably syphilitic. In the left hemisphere, *i. e.*, the side opposite the spasms, adhesion was found between the dura mater and the brain in a region including "the lower part of the ascending frontal and ascending parietal convolutions, to a trifling extent to the hinder part of the third frontal, and several of the convolutions of the upper wall of the fissure of Sylvius behind the ascending parietal". In the right hemisphere—the side opposite the paralysis—"on the surface, behind the fissure of Rolando, was a mass about the size of a chestnut. The dura mater was firmly adherent to it. There was very little softening about it."**

In a fifth,†† temporary right hemiplegia came on after an unilateral convulsion in which the patient did not lose consciousness. Convulsions occurred from time to time, beginning in the little finger of the right hand, occasionally in the right cheek, and followed always by slow and hesitating speech. After death, a syphilitic tumour of considerable size—as large as three small walnuts—was found growing into the cortex about the junction of the frontal and parietal lobes, surrounded by an area of softening in the posterior part of the frontals, ascending frontal and ascending parietal, and partly of the island of Reil. The sphenoidal lobe was also softened. This case, though of interest, is rather a complex one, and should, perhaps, scarcely be quoted as a limited lesion.

Dr. Dreschfeld‡‡ has recorded a very interesting case of brachial monospasm depending on syphilitic disease, the nature and position of which he accurately diagnosed during life. The patient suffered from repeated attacks of convulsion limited to the left arm, of which the phenomena were "sudden clenching of the fist, flexing of the wrist, and pronation of the forearm of the left side, the left angle of the mouth being at the same time strongly drawn downwards. This sudden tonic spasm lasted for several seconds, and was then followed by a few clonic spasms of the same extremity and a slight tremor of the arm; the patient at the same time was very agitated and looked very pale, but remained perfectly conscious. He stated that these paroxysms had always had the same character, varying only in degree". Death occurred from phthisis two years after the first onset of the disease. On *post mortem* examination, the dura mater was found adherent to the brain on the right side, over a space including the greater part of the ascending parietal convolution and the supramarginal lobule (fig. 21, *a* and *b*).

To these cases, in which irritative symptoms chiefly predominated, I may add one or two others, which may be considered as belonging

Fig. 21.

equally to irritative and destructive lesions. The cases are reported in full by MM. Charcot and Pitres §. A case is recorded by Lepine, of left hemiplegia, followed by convulsions, limited chiefly to the left arm. A small hæmorrhage of the size of a nut was found at the "posterior extremity of the first frontal convolution of the right hemisphere". In another, by the same observer, of left hemiplegia, convulsions occurred, either limited to the left arm, or invariably commencing there. A yellow patch was found at the base of the first and second convolutions of the right hemisphere. A third case is given by Mahot,|| of monoplegia and monospasm of the right arm. A glioma of the size of a pigeon's egg was found on the middle third of the ascending frontal convolution of the left hemisphere. In a fourth, recorded by Henrot, convulsions began in the fingers of the left hand, and were repeated at intervals, followed by left hemiparesis. A mass of tubercle

* *Op. cit.*

† *Medical Times and Gazette*, September 4th, 1875.

‡ *Medical Times and Gazette*, September 18th, 1876.

§ *Medical Mirror*, September 1st, 1869.

|| *Medical Times and Gazette*, June 5th, 1875.

¶ *Medical Times and Gazette*, November 20th, 1872.

* *Medical Times and Gazette*, December 28th, 1872.

† *Medical Times and Gazette*, March 1st, 1873.

‡ *Lancet*, February 24th, 1877.

§ *Op. cit.*, p. 365.

|| *Soc. Anat.*, December 15th, 1876.

was found embedded in the grey matter of the ascending frontal convolution of the right hemisphere, about the middle third. In this case, however, there was also a small tubercular mass on the right side of the pons.

It will be seen from these cases that the situation of the lesion causing brachial monospasm is not an accurately circumscribed one; and yet there is a greater agreement among them, when viewed in the light of experimental localisation of the brachial and manual centres, than may at first appear. As has already been said, there are several centres, each for a distinct movement. In four of these cases, in which the spasms were said simply to be in the arm, and in one of them in which it was noted that the spasms began in the proximal movements of the arm, the lesion was situated at the hinder extremity of the first frontal convolution, a position which, as will be seen by reference to the figures (figs. 15, 16 [5]), corresponds with the localisation of the centres of such movements in this region.

In some cases, there was no exact description of the march of the spasm; but in those in which it was more especially noted that the spasms began in the fingers or hand, the lesion was either in, or in close proximity to, the ascending parietal convolution, in which, in the monkey, these movements are especially centralised. Of these, Dreschfeld's case is the most striking, both as regards the precise character of the movements and the limited extent of the lesion, so precise as to have allowed of exact diagnosis, which was verified to the letter *post mortem*.

3. *Facial Monospasm or Protospasm*.—Though partial epileptiform convulsions beginning in the face are not uncommon as symptoms, and not unfrequently alternate with convulsions beginning in the hand, yet there are very few cases on record of facial monospasm, uncomplicated with brachial monospasm or other convulsive or paralytic symptoms, in which the position of the lesion has been verified by *post mortem* examination.

The cases of Hitzig and Wernher, already quoted, in which oral and facial monoplegia and facial monospasm alternated, are about the best examples of this on record. The lesion—the principal, at least—in these cases was the same in position, viz., the lower extremity of the ascending frontal.

A very interesting case, illustrating another fact of experimental localisation, has been recorded by Dr. Bramwell.* A woman, who had received a cranial injury some years previously, began to have right-sided convulsions, and numbness in the thumb and forefinger, followed by paralysis of the right arm and leg. She remained subject to frequently repeated convulsions, which always began in the right platysma, and frequently were almost entirely confined to this muscle. On *post mortem* examination, a spiculum of bone was found projecting from the inner table of the skull, and causing a very limited lesion of the inferior margin of the ascending parietal convolution. A reference to the figure of the brain of the monkey (fig. 15, [11]) will show that at the lower extremity of this convolution, and just posterior to the oral and lingual centres, there is an area marked off, irritation of which specially causes action of this muscle. Bramwell's case receives its explanation from this fact, and in Dreschfeld's case, in which spasm of the platysma was also a special feature, the lesion affected this point. Clinical and physiological experiment are here again in harmony.

B.—LESIONS OF THE SENSORY REGIONS.

I will now direct your attention to the subject of lesions of the sensory regions of the cerebral hemispheres. That the cerebral hemispheres are the seat of sensation or—in order to avoid disputes as to the meaning of sensation—of sensory perception, is abundantly evident both from the results of experimental physiology and from the facts of clinical medicine. And that the regions of the brain we have already considered, viz., the fronto-parietal regions, may be disorganised without causing loss of sensory perception, I consider to be demonstrated both by the facts of experiments on monkeys, and by the clinical evidence which I have laid before you. Some statements which have been made to the contrary, based on experiments on dogs and rabbits, are, as I hope to be able to show you further on, susceptible of a totally different interpretation, and one in harmony with what I have said respecting monkeys and men.

It is clear, therefore, that if the centres of sensory perception are localised in the hemispheres—and this, I think, no one will dispute—they are to be sought for in those regions yet remaining to be considered, viz., the occipital and parieto-temporal lobes. That this is so, we should be led to believe from anatomical as well as from other considerations. For, though I do not place much reliance on mere ana-

tomical investigation as a means of determining the exact course and destination and various connections of the cerebro-spinal tracts, yet, so far as main features are concerned, it furnishes us with substantial bases for other lines of research. It has been established, I think, beyond doubt, that the posterior strands of the crus, and their connections with the brain and the cord, are more especially the paths of centripetal or sensory impressions. The researches of Meynert and others would seem to show that these tracts are connected with those parts of the cortex which we are now considering. Beyond these general indications, however, I have considerable doubts as to the results of anatomical localisation. But in addition to general anatomical indications, we have experimental and pathological evidence as to the exact position of the paths which convey sensory impression to the cortex.

The experimental evidence has been furnished by the researches of Veyssière,* which have been repeated and verified by Carville and Duret, Raymond, and others. These experiments show that when section is made of the posterior part of the internal capsule, that part of the "projection system" which lies between the optic thalamus and lenticular nucleus of the corpus striatum (fig. 22 [x]), there ensues a

Fig. 22.

condition of hemianæsthesia of the opposite side of the body, frequently associated, temporarily however, with some degree of motor paralysis; whereas when the anterior part (two thirds) of the internal capsule (fig. 23)—that part lying between the caudate and lenticular

Fig. 23.

nuclei of the corpus striatum—is divided, motor paralysis, unaccompanied by sensory paralysis, or if so, functional and fleeting, is the constant result.

The facts of human pathology are no less precise. Motor hemiplegia is invariably the result of destructive lesion of the anterior two-thirds of the internal capsule, which may be accompanied temporarily by hemianæsthesia, if the lesion be such as to cause pressure, or functional disturbance of the posterior third. And we have now a tolerably large body of evidence to show that destructive lesions limited to the posterior third of the internal capsule cause cerebral hemianæsthesia.

The first observations relative to this localisation were made by Türck†

* BRITISH MEDICAL JOURNAL, August 28th, 1875.

* Sur l'Hémianesthésie de Cause Cérébrale, 1874.

† Sitzber. der kais. Acad. der Wissensch., Band xxxv; 1859.

in 1859, and since then, Charcot, Magnan, Bourneville, Rendu, Raymond, Pierret, Decaudin, Pitres, Boyer, etc., by their observations and researches, have established the pathology and symptomatology of this affection in a manner which leaves little to be desired.

Since the publication of Türck's four cases, in 1859, of the association of hemianæsthesia with lesion of the posterior part of the internal capsule, there have been recorded at least twenty others, all establishing the same fact.*

The symptoms of cerebral hemianæsthesia, which correspond with those of that strange and, as regards its therapeutics, mysterious affection termed hysterical hemianæsthesia, are such as to clearly differentiate it from anæsthesia of spinal or mesencephalic origin. The special diagnostic feature is, that all the forms of sensibility, general or special, are impaired or abolished; the organs of sense whose nerves take origin above the medulla oblongata being equally affected with those which arise here: whereas, in mesencephalic anæsthesia, in addition to the usual association of alternate paralysis, sight and smell remain unaffected.†

In cerebral hemianæsthesia, tactile sensation is affected unilaterally up to the middle line of the face and trunk; there being more or less complete insensibility to touch, pain, temperature, and also abolition of muscular sensibility, with complete retention of electro-motor contractility. The conjunctival, nasal, buccal, and cutaneo-mucous membranes are also anæsthetic. The viscera, however, remain sensitive, and deep pressure, as on the ovary, etc., is felt as before. In hysterical hemianæsthesia, there is usually hyperæsthesia in the ovarian region, and frequently also the condition termed by Charcot hysterio-epilepsy. Taste, smell, and hearing, are deficient or almost entirely abolished in a similar manner on the one side. As regards vision, the symptoms are especially noteworthy. The eye on the anæsthetic side is rarely rendered completely blind. There is rather a condition of amblyopia or diminution of the acuteness of sight, and a very remarkable contraction of the field of vision, more especially as regards the perception of colour. Landolt has found that the field of colour-perception becomes contracted in a manner corresponding to the relative extent of the colour-field in the normal state. Normally the blue field is the largest, next the yellow, orange, red, green, and last in order the violet, which is perceived only by the most central parts of the retina. In cerebral hemianæsthesia the sensibility for violet first disappears, then for the green, and later for the orange. Sensibility for yellow and blue may still persist; but in the higher degrees of anæsthesia, all colours merge into an uniform sepia tint. Landolt‡ has lately pointed out another important fact, viz., that the affection of vision is not altogether unilateral, but that the eye on the side of lesion participates, though to a less extent, in the anæsthesia.

It is further noteworthy that in this form of amblyopia, ophthalmoscopic examination reveals no organic lesion or degeneration of the optic nerve or retina, in the first instance at least; any atrophic changes which may show themselves subsequently being the consequence and not the cause of the blindness.

It is clear from these facts that the older theories respecting the decussation and distribution of the fibres of the optic tracts, and the representation in each hemisphere, only of the corresponding parts of both retinæ, are untenable. If this were the case, we should have, as the result of a central lesion, a hemiopia of both eyes; whereas we have not a hemiopia, but an amblyopia, which, though to some extent bilateral, is most marked on the side opposite the lesion.

Yet we know that hemiopia is not an uncommon symptom in connection with intracranial disease; but from the facts mentioned, we may conclude that in such cases the lesion must be situated below the cerebral cortex.

The scheme of the optic tracts and their relations, given by Charcot, enables us to give a satisfactory explanation of these facts. (Fig. 24.) Each optic tract contains two sets of fibres; the outer, passing to the eye on the same side; the inner, decussating with their fellows of the opposite side in the chiasma and passing to the corresponding part of the opposite eye. The fibres which do not decussate in the chiasma undergo decussation in the corpora quadrigemina, and pass on with the fibres which have done so to the opposite hemisphere, so that each hemisphere is brought into relation with the whole of the opposite eye. This scheme does not represent the bilateral relation of each hemisphere to both eyes, which is indicated by Landolt's researches; but we can account for this by the bilateral association in the lower ganglia. It is easy to see that a lesion of the one optic tract (K fig. 24)

will cause bilateral hemiopia; and that a lesion in the region of the corpora geniculata (CG), or posterior part of the optic thalamus, will have a similar effect, as in a case reported by Hughlings Jackson.* We

Fig. 24.

may also have cross amblyopia and bilateral hemiopia, as in a case reported by Gowers,† but as there was no necropsy, the position of the lesion is only a matter of speculation. I hope to be able to show you that these clinical observations are in harmony with the results of my experiments on monkeys.

It is evident that the lesion which causes hemianæsthesia, being in the medullary fasciculi, produces this effect, not by destroying the centres of sensory perception, but by causing solution of continuity of the paths of centripetal impressions; and the question is, whether the sensory fibres, like the motor fibres of the internal capsule, are distributed to localisable areas in the cortex. On this point, experimental physiology is, I think, in some respects at least definite enough; though it must be admitted that the same cannot as yet be said of clinical medicine and pathology. To these let us now turn.

Lesions of the Occipital Lobes.—I will first direct your attention to the occipital lobes, and compare the results of experimental investigation with the facts of disease of these regions. In reference to experimental investigation, we must rely mainly, if not exclusively, on experiments on monkeys; as in other animals these lobes are not specially differentiated and developed. I have found, as the result of numerous experiments, that electrical irritation may be applied to the occipital lobes without producing any objectively discoverable reaction. Neither does destruction or complete removal of these lobes singly, or on both sides, cause any appreciable sensory or motor disturbance. Animals so mutilated continue to see, hear, touch, taste, and smell, and retain all their powers of voluntary motion. The results of destruction, like those of irritation, are, therefore, mainly negative, and do not succeed in throwing very clear light on the functions of these regions. In one or two instances, I should mention that there seemed to be some affection of vision; but in these cases, I found that the lesion had extended beyond the occipital lobes, into the angular gyri; while in those in which the lesion did not extend beyond the occipital lobes, no such symptoms occurred. As a rule, the animals rapidly succumbed, with one exception; and I also observed that, contrary to the usual effects of destructive lesion of other parts of the hemispheres, the animals refused to eat: an occurrence which, from my numerous and close observations of the effects of destruction of every other part of the brain, I regard as in some way causally related to this lesion.

I admit that the inference as to causal relationship is considerably weakened by the fact that, in one of the animals in which I had removed both occipital lobes, the appetite for food returned after five days' abstinence. I have, however, attempted, whether successfully or not, to explain this, and propounded the hypothesis that the occipital lobes are specially related to the visceral sensibilities, and are the anatomical substrata of the correlated feelings which form a large portion of our personality and subjectivity. Whether the hypothesis is well founded or not, I leave to further investigation; but one thing is, to my mind, clearly established by these experiments, viz., that lesions of the occipital lobes, whether unilateral or bilateral, cannot be regarded as the direct cause either of motor or special sensory affections; and, therefore, I would at once discard the anatomical views of Meynert, Hugue-

* For references, etc., see Grasset, *Localisations dans les Maladies Cérébrales*. Montpellier, 1878.

† See Couty, "De l'Hémianesthésie Mésocéphalique," *Gaz. Hebdomadaire*, 1877, p. 30, et seq.

‡ *La France Médicale*, February 3rd, 1877.

* "A Physician's Notes on Ophthalmology" (*London Hospital Reports*, vol. viii, Part ii, 1875).

† "On Athetosis" (*Medico-Chirurgical Transactions*, vol. lix, Case 7).

nin, and others, which would place the central terminations of the optic tracts in the occipital lobes. They are not, I think, in harmony either with physiological experiment, or, as far as I can discover, with the facts of human pathology.

Though many speculations exist, to some of which I shall refer, as to the symptoms dependent on lesions of the occipital lobes, it does not as yet appear, as the following cases will show, that there are any symptoms, attaching to disease of these lobes, so definite and constant as to establish any direct causal relationship between them, or indicate the functions which these regions subserve. Lesions of the occipital lobes are, as a rule, *latent*.

M. Vauttier* records a case of yellow softening of the right occipital lobe, and, to a larger extent, of the internal aspect of the left (quadri-lateral lobule). No affection of motion or sensation existed; and, with the exception of considerable hebetude, there were no other symptoms of cerebral affection.

Pitres† relates a case, in which, in consequence of a fall on the head, an abscess, the size of a billiard-ball, formed in the postero-inferior aspect of the brain. There was no paralysis of motion or sensation, and mental obtuseness was the only indication of cerebral lesion.

Sir W. Gull‡ has recorded a case of abscess of the left posterior lobe, also without any objective symptoms.

A similar case, in which the abscess was situated in the right occipital lobe, is recorded by Rodocalat.§

Other cases, reported by Bleyne, Hébread, Martinet, Merriman, Ogle, Parrot, Ballinger, etc., are cited by M. Pitres in his before mentioned work,|| to which I would refer the reader.

It may be said, in reference to these cases, that the lesions being unilateral, and of slow growth, the absence of symptoms may be explained by functional compensation by the same or the opposite hemisphere. There are, however, some cases on record of traumatic lesions of the occipital lobes, also negative as regards objective symptoms.

Marcé¶ records a case of contusion, with effusion into the meninges, and softening of the cortex in the right occipital lobe, without any symptom as regards sensation or motion.

In addition to Vauttier's case already mentioned, a very important case of bilateral lesion of the occipital lobes has been put on record by Sestié.** In Sestié's case, there was an abscess in each occipital lobe, without any objective symptom; although the patient's memory was somewhat defective, there was nothing very remarkable in his mental condition.

Leger†† relates a case of tumour invading both occipital lobes, in which, beyond general mental obtuseness and headache, there were no objective symptoms. Sight was not impaired in this case, notwithstanding the existence of a cerebral tumour in this region. Except, therefore, as regards the fact of latency of lesions of the occipital lobes, these cases do not give us much positive material for generalisation.

Joffroy‡‡ attempts to establish a relation between lesions of the occipital lobes and the occurrence of acute sloughing of the sacrum; and thinks that the occipital lobes may be trophic centres. He quotes three cases in support of this hypothesis. One, a case of general paralysis, in which, though the cerebral lesions were not confined to the occipital lobes, yet the sacral sloughing was greater on the side opposite the lobe most affected. In the other two, there was only slight unilateral lesion: in the one case, a small focus of hæmorrhage; in the other, of softening, with sloughing on the opposite side of the sacrum.

But, in reference to this hypothesis, it needs only to be remarked that sacral sloughing occurs in connection with hemiplegia where no affection of the occipital lobes has been detected; and lesions of the occipital lobes are not even commonly associated with such results. Cases have occurred, and been carefully examined, in reference to this point since the publication of M. Joffroy's views, without confirming them. One such case is reported by Sazic,§§ and another by Dreyfus-Brisac.|||| These may be taken in conjunction with the other cases mentioned; for it is not likely that such a serious objective affection would have been overlooked had it actually existed.

In connection with softening of the occipital lobes, M. Charcot has occasionally noted, in addition to headache, etc., cutaneous formication and similar subjective sensations, but no true anæsthesia.

Hughlings Jackson and Bastian are of opinion that disease of the posterior lobes is more frequently associated with mental derangement than disease of the anterior lobes or other parts of the brain. Hughlings Jackson goes somewhat further, and thinks that such derangements, particularly "defective perception", are more commonly seen when the right side is affected, and that irritative lesions here give rise to coloured vision and other subjective ocular spectra. These are not put forward, however, as other than hypotheses, and I agree with Dr. Bastian when he says: "More extended observations, however, seem needed before we can safely arrive at a positive conclusion on this difficult subject."*

Even on my own hypothesis as to the relation between the occipital lobes and the organic sensations, I should regard it as highly probable that lesions of the occipital lobes should cause profound mental disturbances. For, if our feelings are our chief motors, it might readily be allowed that lesions of the anatomical substrata of such an important element of our feelings and emotions should lead to mental derangement.

But, not to indulge further in speculation, I think we have to admit that up to the present the facts do not yet enable us to generalise with certainty as to the positive effects of lesions of the occipital lobes. It is clear, however, from the negative effects of extirpation or disease that we cannot place in the occipital lobes the central terminations of the fibres of the internal capsule which convey impressions of special sense to the cortex.

[To be continued.]

CLAUDE BERNARD:

A LECTURE

Delivered to the Senior Class of Physiology at the Physiological Laboratory, New Museums, Cambridge.†

By MICHAEL FOSTER, M.D., F.R.S.,
Trinity Prælector in Physiology.

IN order, however, that you may understand the beginning of this matter, I must take you back a little.

Possibly it has never occurred to you that there was a time when the plain unstriated muscular fibres of the arteries were things unknown, or at least not believed in; and yet it is less than forty years ago (1840) that their presence was first demonstrated by Henle. Just imagine what lame and mutilated answers you would send up, in your examinations, if you were forbidden to employ any reasoning which involved the existence of muscular contractility in the arteries. You would be brought to a standstill, not in questions only belonging directly to the circulation, but in almost all kinds of physiological problems. How could you treat properly the secretion of the digestive juices, the excretion of urine, the origin and distribution of animal heat, or even the problems of the central nervous system itself, if you were debarred from all reference to vaso-motor action? Try to withdraw from the web of to-day's physiology the thread of vascular contractility, and the whole tissue becomes a tangled skein. How, then, you may naturally ask, were those phenomena explained which we now so readily interpret as the results of vaso-motor action? Happy are you that you can now learn physiology without having your minds burdened with the ideas of determination of blood, nutritive force, capillary attraction, and other *Dei ex machinâ* called in to explain the facts which you unravel by help of the simple protoplasmic property of contractility.

Let me read you what the great anatomist and physiologist Johannes Müller wrote, just before Henle's discovery. I read from Baly's translation, which was not published indeed till after Henle's paper had appeared. "The old writers, and many recent physiologists, have erroneously regarded the contraction of the arteries which follow their dilatation as a muscular act, and have looked upon the fibres of the middle arterial coat as muscular fibres. But the fibres of the elastic coat of arteries differ, as we have already mentioned, from muscular fibres, both by their physical and chemical properties, as Berzelius has shown, as well as by their form. The different arguments for the existence of the pretended muscular contractility of arteries, which have

* *Essai sur le Ramolissement Cérébral Latent*, 1868.

† *Lesions du Centre Ovale*.

‡ *Guy's Hospital Reports*, 1857.

§ *Bull. Soc. Anat.*, 1870, p. 289.

|| *Op. cit.*, p. 134.

¶ *Bull. Soc. Anat.*, 1854, p. 295.

** *Bull. Soc. Anat.*, 1833.

†† *Bull. Soc. Anat.*, November 1876.

‡‡ *Soc. de Biologie, Séance December 4th, 1875.*

§§ *Bull. Soc. Anat.*, December 15th, 1876.

|||| *Bull. Soc. Anat.*, March 23rd, 1877.

* *Paralysis from Brain-Disease*, p. 239.

† Concluded from page 521 of last number.