THE GOULSTONIAN LECTURES ON THE LOCALISATION OF CEREBRAL DISEASE.

Delivered at the Royal College of Physicians of London.

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LECTURE II (concluded).—March 20th.

Brachio-Crural Monoplegia.*—Paralysis of the leg occurs more frequently in association with paralysis of the arm than singly as the result of cortical lesions. I have already given several cases of this kind, to which the following may be added.

M.M. Charcot and Pitres† give a case of paralysis with rigidity of the left limbs of three years' duration, sensibility being unimpaired. A patch of softening 5×2.5 centimetres was found at the upper extremity of the fissure of Rolando, on the convex surface of the right hemisphere. (Fig. 18.)

M. Moutard-Martin‡ reports a case of fracture of the skull and injury of the brain, causing paralysis of motion of the right arm and

Fig. 18.

leg, for which trephining was practised without success. The cerebral lesion occupied the upper extremity of the ascending frontal, ascending parietal, and anterior two-thirds of the postero-parietal lobule, and slightly to the inner aspect of this region, or paracentral lobule.

M. Pitres§ gives a case in which the lesion affected, not the cortex, but the medullary fasciculi of the superior frontal and parietal region.

Brachial Monoplegia.—The centres for the movements of the arm and hand occupy a considerable space, as might be expected from their importance as organs of intelligence. Area [5] (figs. 15 and 16) is the centre for the forward extension of the arm and hand; area [4], for the adduction and retraction; area [6], for the supination and flexion; while the various letters (*a, b, c, d*) placed in the ascending parietal convolution indicate the portion of the various centres for the movements of the wrist and fingers. With these latter, retraction of the angle of the mouth is apt to be associated, owing to the proximity of the facial and oral centres (7, 8, 9, 10, 11, fig. 15): a proximity which serves to explain, among other things, the retraction of the angle of the mouth so commonly seen associated with powerful action of the hand. Hence also the fact that brachial monoplegia occurs less often than brachio-facial paralysis.

It is obviously highly important, in any given case of brachial monoplegia of cortical origin, to determine, if possible, which of the movements of the upper extremity are particularly affected. This has not been done with any great degree of accuracy in many of the recorded cases.

M. Maurice Raynaud|| reports a case of paralysis limited to the left

arm, and more particularly to the extensor muscles, though the flexors were also paretic. There was no diminution in sensation, nor alteration in electro-motor contractility. The lesion was situated in the ascending parietal convolution of the right hemisphere, in the position of a line drawn back from the second frontal. It was an area of softening surrounding a tubercle of the size of a millet-seed, and about a centimetre in diameter; the softening affecting the subjacent medullary fibres more than the cortex. A smaller area of softening of the size of a pea was situated near this, but within the fissure of Rolando. The whole lesion could be covered with the pulp of the finger.

M. Sabourin* records a case of sudden partial right hemiplegia without loss of consciousness, which soon disappeared, leaving paralysis of the right hand and arm, which continued till death seven days afterwards. A focus of red softening was found in the left hemisphere of the size of a two-franc piece, the centre of which, where the softening was greatest, corresponded with the junction of the ascending parietal convolution and supramarginal lobule. The softening extended halfway up the ascending parietal convolution, somewhat concealed within the fissure. The ganglia were intact.

M. Boyer† has recorded a case of sudden paralysis of the left arm and leg. After four or five days, the leg regained its power, while the left arm remained permanently paralysed. The affection happened in 1872. In 1877, the condition was rigidity of the left arm and atrophy of the muscles of the forearm. Sensation was unimpaired. The patient died of bronchopneumonia. The left hemisphere was normal. In the right hemisphere, a patch of atrophy, caused by an old yellow softening, existed in the ascending parietal and ascending frontal convolutions; and an extension of the same existed in the temporo-sphenoidal region. The position of the lesion is indicated, from a drawing sent me by M. Boyer, in the diagram before you.

M. Bourdon‡ has collected several other cases, from which I quote the following.

A case related by M. Pierret, of an apoplectic attack with early rigidity of the left arm and retraction of the right angle of the mouth, followed by partial paralysis of the left arm. Death occurred twelve days afterwards, from sudden coma and epileptic convulsions. In the right hemisphere, at the junction of the middle frontal with the ascending frontal convolution, a focus of red softening of the size of a franc-piece existed. A similar focus existed in this case in the median occipital of the same hemisphere.

Another case, observed by Darolles, is given, of cerebral affection resolving itself ultimately into paralysis of the right arm. A hæmorrhagic extravasation was found in the left hemisphere, embedded in the cortical substance, three millimetres in extent, at the upper part of the ascending parietal convolution. A patch of softening also existed in the median occipital lobe of the right hemisphere in this case.

In a third case, there was an association of aphasia with paralysis of motion of the right arm. The brachial monoplegia continued till death. After death, a small hæmorrhagic extravasation was found at the upper part of the ascending frontal convolution of the left hemisphere.

M. Bourdon also quotes an interesting case of double brachial monoplegia communicated to him by M. Vermeil. This was the result of a cranial injury. The arms only were paralysed as to motion, sensation being unimpaired. Death occurred two days after the accident. There was no depression of the skull at any part. On the surface of the brain were two small superficial hæmorrhagic extravasations of about fifteen millimetres in diameter. The one of these was situated towards the upper part of the ascending frontal in the left hemisphere. The other, in the right hemisphere, was situated at the junction of the ascending parietal with the supramarginal lobule.

Several other cases of brachial monoplegia (Raymond's, Demongeot de Confrevron's) are on record; but the characters of the lesions (tuberculo-meningeal) are not altogether such as to admit of their being quoted as exactly circumscribed. I will only refer to one other case of brachial monoplegia, respecting which there exists some doubt as to its harmony with the other facts related in reference to limited paralyses. The case is related by M. Cotard.§ A woman, who died at the age of seventy-one, had been seized with convulsions, followed by paralysis of the left arm, about the age of two years. The left arm continued paralysed and rigid. She walked with difficulty; but this was apparently due to fracture of the neck of the left femur a few years before. Formerly no difficulty was noted in this respect. The right hemisphere was smaller than the left. "Behind the upper extremity of the fissure of Rolando, there was a longitudinal depres-

* I use the term monoplegia here to indicate paralysis of a distinct group of movements, and not as meaning paralysis of a muscle or a limb.

† *Op. cit.*, p. 185.

‡ *Bulletin de la Société Anatomique*, December 8th, 1866.

§ *Op. cit.*, p. 85.

|| *Bulletin de la Société Anatomique*, July 1876.

* *Le Progrès Médical*, 1877, p. 391.

† *Bulletin de la Société Anatomique*, May 4th, 1877.

‡ *Op. cit.*

§ *Atrophie partielle du Cerveau*, p. 21.

sion of the cortex, which extended parallel with the longitudinal fissure as far as the occipital lobule, five *centimètres* in length by one *centimètre* in breadth." It is not clear in this case what extent of the ascending parietal convolution was involved, or how much of the postero-parietal lobule, if any, as it is only said that the linear depression was parallel to the longitudinal fissure. M. Charcot, in whose *clinique* the case occurred, is unable to give me further particulars as to the exact seat of the lesion. The uncertainty in this case is as regards the extent of the affection of the centre for movements of the leg, which, whether they were affected at first or not, do not seem to have been permanently so.

A somewhat similar uncertainty also exists in reference to Boyer's case already quoted, in which, though the leg was at one time paralysed, yet recovery took place, though some lesion of the leg-centre still appears to have continued. It is, therefore, a question whether the leg may regain complete power, notwithstanding the existence of partial lesion of its cortical centre. These cases would seem to indicate this, granting always that the observations were thoroughly exact and minute.

M. Charcot, in a private communication to me respecting Cotard's case, thinks that cortical lesions in infancy do not have precisely the same effect as similar ones in the adult, owing to the functions of the cortical centres not being as yet well defined. This is worthy of particular investigation, and would seem to be justified by the experiments of Soltmann in reference to the development of the cortical centres in young animals.*

With the exception of Cotard's case, which must remain doubtful, the seven cases I have quoted of limited lesions with brachial monoplegia are entirely in harmony with the localisation of the head and arm centres in the monkey; for, though the lesions recorded did not occupy the same position in all, yet they were all in regions included within the area in which these centres are situated, viz., the ascending parietal and upper part of the ascending frontal convolutions.

In those in which it was noted that the hand in particular was affected, the lesion invaded the ascending parietal convolution, in accordance with the facts of experimental localisation. In others, no differentiation was made, or was possible. It would have been most interesting if this had been done in Vermeil's case of double brachial monoplegia, as the lesions occupied centres for different movements of the upper extremity.

As with the leg, so also in regard to the arm, some cases of amputation, or congenital deficiency, of the hand or arm are on record in which atrophy has been described in certain convolutions of the opposite hemisphere.

A case is reported by Chuquet† of amputation of the right arm six years before death. In the left hemisphere, there was atrophy of the upper third of the ascending parietal convolution and corresponding part of the internal surface of the hemisphere. The length of the atrophied part was two *centimètres*.

M. Boyer‡ examined the brain of a man who died at the Bicêtre, having had his left arm amputated thirty years previously. The ascending convolutions were atrophied at their superior extremity, and the ascending frontal was throughout very slender and flattish.

Dr. Gowers has kindly given me the particulars, accompanied by a photograph of the brain, of a case of congenital absence of the left hand. The middle part of the ascending parietal convolution in the right hemisphere is, as clearly shown in the photograph, much smaller than the corresponding convolution in the left. A microscopic examination had not been made at the time Dr. Gowers gave me these particulars, though he intended carrying this out.

The cases of atrophy—and of these Dr. Gowers's seems the most satisfactory, inasmuch as the lesion was congenital—if we cannot place absolute reliance on them as yet, are not opposed to the localisation of the hand and arm centres as determined by physiological experiment or the facts of disease.

Brachio-facial Monoplegia.—The combination of brachial with facial paralysis is a much more frequent occurrence than brachial or facial paralysis singly in connection with cortical disease; and very commonly, and for obvious reasons, it is associated with aphasia when the disease is in the left hemisphere. This, however, is not always the case; and I have recently seen two cases of right brachio-facial monoplegia without aphasic symptoms. Many such occur, but recover and are lost sight of. *Post mortem* examination has, however, been made in several.

Dieulafoy§ has recorded the case of a woman, aged 60, who was sud-

denly seized with paralysis of motion in the right arm and right lower facial region. Death occurred the next day from coma. The necropsy revealed a hæmorrhagic extravasation, of the size of a nut, surrounded by a zone of softening in the ascending frontal convolution of the left hemisphere. The exact position of the lesion in the ascending frontal is not given more accurately in the record.

Troisier* gives a case of a man, who died of phthisis, who had been suddenly seized with paralysis in the right arm, at first showing itself in the muscles supplied by the musculo-spiral nerve, and followed by complete paralysis of the limb, with the addition of right facial paralysis; sensation was intact. In the left hemisphere, a patch of hyperæmia and yellow granulations, of seven to eight square *centimètres* in extent, was found posterior to the third frontal convolution. The lesion was not very definite in this case, and it was further complicated by the simultaneous occurrence of granulations in the membranes behind the posterior parietal convolution.

M. Hippolyte Martin† has recorded a case of left facial paralysis, with paresis of the left arm, more particularly shown in the first three fingers. The affection had come on suddenly, without loss of consciousness or other paralysis, five or six months previously. The necropsy revealed a patch of yellow softening in the lower fifth of the ascending parietal convolution of the right hemisphere. The softening extended up the fissure of Rolando, to a level with the posterior extremity of the second frontal convolution. The softening penetrated about one *centimètre* from the surface. The basal ganglia and the rest of the brain were normal.

Cruveilhier‡ gives a case, illustrated by a figure, of paralysis suddenly occurring on the right side of the face and tongue (aphasia), with paralysis and rigidity of the right arm. The lesion in this case was a patch of red softening of about 2 x 4 *centimètres* in extent, situated at the lower third of the fissure of Rolando, and affecting also very specially the ascending parietal convolution.

A case reported by Anton Frey§ is referred to by M. Pitres||, as showing that brachio-facial monoplegia may occur from lesions limited to the medullary fibres of the middle pediculo-frontal and frontal section. The symptoms were paresis of the left arm and left side of the face, without affection of sensation. Death occurred from gangrenous erysipelas of the face. In the right hemisphere, a small focus of hæmorrhage, formed by the juxtaposition of three minute extravasations, each of the size of a mustard-seed, was found in the medullary fibres at the junction of the middle frontal with the ascending frontal convolution.

Other similar cases are given by Pitres, which I need not, however, quote. I might cite many other instances of this form of monoplegia in connection with cortical lesions; they are not, however, all described with the degree of topographical accuracy which is desirable. I would refer the reader to M. Landouzy's work, where most of them are collected and details given, and also to Grasset's recent memoir, *Localisation dans les Maladies Cérébrales*, 1878.

A glance at the diagrams before you, and a comparison of the position of the lesion in these five cases with the situation of the hand and facial centres (areas [7] [8] [11], fig. 15), will show how close is the correspondence between them. The lesions causing brachio-facial monoplegia are all towards the middle of the ascending frontal and parietal convolutions or somewhat lower, regions which include facial and brachio-manual centres, as indicated by experimentation on monkeys.

Facial Monoplegia.—Facial paralysis of the cerebral type—i. e., paralysis limited almost exclusively to the lower facial region—is not a very common occurrence by itself in connection with cortical disease. Usually, it is complicated with brachial paralysis or with aphasia; with the latter more especially when the lesion is in the left hemisphere. This is naturally to be expected from the proximity of the facial centres ([7] [8], fig. 15) to those of the arm and hand already indicated, and to the lingual and oral centres ([9] [10] [11], fig. 15).

Brown-Séquard¶ reports a number of cases of facial paralysis, "seemingly caused" by lesions in various parts of the brain, such as the frontal, occipital, and temporo-sphenoidal lobes; but as there is not even a common, to say nothing of a constant, relation between such lesions and motor paralysis of any kind, and as there seems to have been no attempt at distinction between peripheral and central facial paralysis, the facts have little or no value as regards localisation.

The cases on record in which paralysis limited to the face has been

* *Jahr. für Kinderheilk.*, Band ix.

† *Bulletin de la Société Anatomique*, November 1876.

‡ *Soc. Anat.*, April 1877.

§ *Gazette des Hôpitaux*, 1868, p. 150.

* *Bulletin de la Société Anat.*, 1872, p. 262.

† *Revue Mensuelle*, No. 3, p. 136.

‡ *Atlas*, liv. xx, pl. 4.

§ *Archiv für Psychiatrie*, 1875, p. 327.

¶ *Op. cit.*, p. 76.

¶ *Lancet*, April 1877.

found in connection with localised cortical disease have been due to lesion of the right hemisphere; but in none can the lesion be said to have been accurately defined or uncomplicated. Charcot and Pitres* give a case, with a figure (fig. 19), of a lesion, extensive in one sense, but limited as regards the motor area of the brain involved. This was

Fig. 19.

a case of apoplexy with facial paralysis and early rigidity of the limbs on the left side. The rigidity of the limbs disappeared, but the facial paralysis persisted till death. An extensive area of softening was found in the right hemisphere, invading the third frontal, the lower extremities of the ascending convolutions, and a large extent of the parietal, temporo-sphenoidal, and insular lobes.

Hitzig† relates the case of a French soldier, who, two months after a bullet-wound on the right side of the head, began to be affected with clonic spasms in the left side of the face. These were followed by transient, but complete, paralysis of the left side of the face and left side of the tongue. Clonic spasms occurred also in the left hand. After death, an abscess was found corresponding to the seat of the injury, situated in the ascending frontal convolution, between the pre-central fissure and the fissure of Rolando. It should, however, be noted that there were indications of meningeal inflammation over the whole surface of the right hemisphere, though there was no cerebral softening except in the neighbourhood of the abscess. A similar case is reported by Wernher.‡ Had the lesions been in the left instead of the right hemisphere, we should in all probability have had the symptoms of aphasia superadded, as in the following case—to select one from a host—related by Hervey.§ This was a case of right facial paralysis with aphasia. A focus of softening was found anterior to the fissure of Rolando, at the junction of the third frontal with the ascending frontal convolution of the left hemisphere.

Dr. Gowers|| has reported a case of left hemiplegia, which gradually recovered, with the exception of very marked paralysis of the inferior facial muscles. Under the influence of emotion, however, the muscles acted equally well on both sides. This was found to depend on a hæmorrhagic extravasation in and beneath the upper half of the pre-central sulcus, which had pressed upon the convolutions bounding it—viz., the posterior extremity of the middle and superior frontal, and corresponding part of the ascending frontal of the right hemisphere.

Aphasia—Oro-lingual Hemiparesis.—I would now make a few remarks on what may be described as an objective-subjective monoplegia; in the one aspect, oro-lingual hemiparesis; in the other, aphasia. It is not my intention to discuss the intimate pathology of aphasia, nor the relation between the objective and subjective aspects of the lesion on which this depends. Nor do I think it necessary, in the present state of clinical medicine and pathology, to take up time with the enumeration of instances of aphasia with lesions in what is termed Broca's region.

The occurrence of aphasia in the immense majority of instances in connection with disease of the left hemisphere and with disease in a region which, as you will see by reference to the diagrams, corresponds with the oral and lingual centres in the monkey ([9] [10] figs. 15 and 16), is a fact which can no longer be disputed. It is also a fact that, in the great majority of instances, aphasia is associated with a greater or less

degree of right hemiplegia or right monoplegia, of which the most common is brachio-facial or facial monoplegia.

It is necessary that those who dispute the validity of inferences as to causal relationship between the lesions indicated and aphasia, should clearly understand what is contended for by those who consider this relationship established. Aphasia, in the strictly limited sense of the term, or Broca's aphasia, does not mean speechlessness from paralysis of articulation, nor speechlessness from general cerebral disturbance, such as emotional shock, etc., but the inability to express thoughts in articulate speech, or to think in words, and all that this implies.

It is not contended that there is an absolute restriction of the speech-centre to the left hemisphere. This, though the rule, is not an absolute rule, but only an approximate generalisation, and therefore exceptions may be admitted without invalidating the localisation of the speech-centre in one side or the other, which is what is really maintained. Hence, to overturn the localisation of a speech-centre, it is not enough to bring forward a case of lesion of the left speech-centre without aphasia. This is admitted by all; and it is a very significant fact, that in several at least of the cases of aphasia with disease of the right speech-centre, the patients have been left-handed.

It is incumbent upon the opponents of this localisation to bring forward a case in which, with bilateral lesion of this centre, no aphasia occurred. But, I need scarcely say, no such evidence exists.

The effect of a bilateral lesion would be, according to the results of experimental physiology, both aphasia and anaesthesia, or paralysis of articulation.

A beautiful, and I believe a unique, instance of this has been put on record by Dr. Barlow.* A boy aged 10, the subject of aortic disease, of which he ultimately died, was seized with right hemiplegia, chiefly brachio-facial, and aphasia. Of this he had apparently recovered at the end of a month. Three months afterwards, he was seized with left brachio-facial monoplegia. This time there was not only aphasia, but paralysis of all voluntary movements of the mouth and tongue. Reflex deglutition, however, was unimpaired. There was no affection of sensation in the paralysed parts, either in the skin or in the mucous membranes of the palate, etc., and the muscles reacted normally to the faradic current. "To sum up the cerebral condition", says Dr. Barlow, "there appeared to be loss of voluntary motor power over the muscles concerned in deglutition and articulation." This lasted till death, while the arms improved somewhat in power. Intelligence was fair, and comprehension good. On *post mortem* examination, a lesion was found in each hemisphere, and in exactly corresponding situations. The region involved by the lesion—which was yellow softening—was

Fig.

"the lower end of the ascending frontal and the hinder end of the middle and inferior frontal convolutions". (Fig. 20.)

This case, in all its features, whether we look at it as an experiment

* *Revue Mensuelle*, 1877, p. 181.

† *Archiv für Psychiatrie*, 1872, Band iii, p. 231.

‡ *Archiv für Path. Anat. und Physiologie*, Band lvi, p. 289.

§ *Bulletin de la Société Anatomique*, 1874, p. 29.

|| *Pathological Transactions*, 1876, p. 35.

of disease, approaching to the conditions of an exact and precise experimental lesion, or to the care with which it was observed and distinguished from peripheral or bulbar paralysis, is one of the most satisfactory and conclusive demonstrations of the harmony between human pathology and experimental physiology with which I am acquainted.

Some ingenious—if we cannot call them successful—attempts have been made to overthrow the causal relationship between lesions in Broca's region in the left hemisphere and aphasia, and to make it a relationship merely of co-existence.

As embolism or thrombosis of the left middle cerebral artery or one of its branches, is perhaps the most frequent cause of aphasia, it has been suggested by Jaccoud and others, that the greater frequency of aphasia with left cerebral lesion might be accounted for by the greater frequency of embolism in the left middle cerebral artery than in the right. That this is so appears to be an indisputable fact, and perhaps to be accounted for by the physical conditions of the blood-supply. Meissner* found in 38 cases of embolism, 26 in the left and 12 in the right; and Bertin found 31 cases of embolism of the left to 7 of the right middle cerebral. Aphasia, however, does not always depend on embolic softening; and if we take the relative frequency of softening due to any cause, in the right and left hemisphere respectively, we find, according to Andral's statistics, that the right hemisphere is more liable than the left. Thus out of 169 cases, 73 were in the right, 63 in the left, and 33 in both hemispheres. And in reference to diseases of the hemispheres generally, Charcot and Vulpian found the relative proportions in the two hemispheres very nearly equal. They found 58 cases of left hemiplegia with disease of the right hemisphere, to 52 cases of right hemiplegia with disease of the left. As regards the relative frequency of aphasia with right and left hemiplegia respectively, Seguin found, from an analysis of 266 cases of hemiplegia with aphasia, 243 with right hemiplegia, and 17 with left hemiplegia; i.e., a proportion of 14.3 : 1. Now, if we take Bertin's statistics as to the relative proportion of embolism of the left and right middle cerebral as 4.4 : 1, and Seguin's association of aphasia with lesion of the left hemisphere as 14.3 : 1, we have, in favour of the association of aphasia with lesion of the left hemisphere, a preponderance of 10 : 1, which cannot be accounted for by mere fortuitous collocation; and even if the arguments founded on the relative frequency of disease in the left and right hemisphere respectively, had not been thus shown to be without foundation, they would be at once disposed of by cases of aphasia resulting from traumatic lesions of the left hemisphere. Several interesting cases of this kind are on record.

Sydney Jones† gives a case of aphasia resulting from fracture of the left side of the skull by the kick of a horse. After death, an abscess of the size of a nut was found in the medullary substance of the third left frontal convolution.

Simon‡ gives a case of a healthy man who fell from horseback, and who on getting up was found by a physician who came up to be aphasic, and without any signs of paralysis. Death occurred from meningitis. A small wound with depressed fracture of the left side of the skull was found; and corresponding to this was a cerebral softening—surrounded by meningeal inflammation—of the third left frontal (in which a spiculum of bone was imbedded), the second frontal, and the island of Reil.

A similar case of aphasia from fracture of the left side of the skull, in which recovery took place on trephining, has been recorded by MM. Proust and Terillon§.

That gross anatomical lesions have not always been discovered in Broca's region in cases of aphasia may be true, but this is a fact of no value against the localisation of the speech-centre here. Perhaps they might have been discovered more frequently if careful search had been made, for on more than one occasion they have been found, when supposed absent, in the subjacent medullary fibres. Of instances of this kind several have been collected by M. Pitres.||

The clinical evidence alone is amply sufficient to establish the relation between aphasia and lesion of Broca's region as an empirical generalisation; but when we take into consideration also the facts of experimental physiology and the light they throw on the motor substrata of mind, the connection between lesion of Broca's region and aphasia is no longer a merely empirical generalisation, but a derivative law, which in my opinion is established on as firm grounds as any other fact in scientific medicine.

Diagnosis of Cortical Paralysis.—Apart from considerations as to the mode of onset, etc., of the affection, there are no features which clearly enable us to distinguish between hemiplegia depending on general destruction of the motor area of the cortex, and hemiplegia due to destructive lesions of the corpus striatum, more particularly those involving the anterior two-thirds of the internal capsule. There is the same relative affection of the different movements; those being most paralysed which are most volitional, at least after the first rude shock of the disease has subsided. The facial paralysis is seen especially in the lower facial region, or in those movements which are most independent; while the frontal and the orbicular muscles of the eye are but slightly affected. The movements of the leg are less paralysed than those of the arm, and the proximal movements of the arm less than those of the hand. In neither case is sensation affected, if the lesion be strictly limited to the cortex, or to the anterior two-thirds of the internal capsule; and in neither case is the nutrition or electric contractility of the paralysed muscles directly impaired. The same tendency exists in the development, sooner or later, of descending sclerosis of the motor tracts of the crus, pons, medulla, and spinal cord, and the appearance of late rigidity or contracture of the paralysed limbs. This late rigidity seems, according to the views of Charcot, Bouchard, Bastian, etc., to be essentially dependent on the degenerative process, and has its analogue in lateral sclerosis of protopathic origin. Hughlings Jackson, however, looks on late rigidity as a species of tonic distortion, caused by the cessation of the cerebral influence, and the consequent unopposed action of the cerebellar and mesencephalic centres; for, says he, "there is unimpeded cerebellar influx, and hence rigidity of the muscles which in health the cerebrum chiefly innervates".* I see serious objections to this view. If it were true, I should expect the antagonism or cerebellar influx to show itself at the beginning of the paralysis; for distortion, consequent on the cessation of an antagonist, is always most marked at the commencement, whereas, in the rigidity of hemiplegia, it is usually weeks or months before it sets in. And if it were cerebellar influx, I should further look for the rigidity in the extensor muscles of the trunk and legs, owing to the more especial relation of the cerebellum to these movements; whereas the very contrary holds, as the rigidity is most marked, and, in a large majority of cases, is confined to the arm and flexors more especially. The rigidity, in fact, seems to affect those muscles most which are most paralysed by destructive lesions of the hemispheres. If it be objected—and this seems to be the main objection—that the remittent or intermittent character of the late rigidity, in its early stages, militates against the idea of its being dependent on a permanent organic lesion, we can find analogous phenomena. The neuralgic pains which are associated with progressive sclerosis of the posterior columns of the spinal cord are not constant, but remit, intermit, or vary under different conditions, which modify the nutrition and activity of the nerve-centres. It seems to me more in accordance with all the facts to attribute, with Charcot, etc., the late rigidity to irritation set up by the process of degeneration of the motor tracts, just as in protopathic lateral sclerosis.

As regards the temperature of the paralysed limbs, in central and cortical disease respectively, there seems to be some uncertainty, and we cannot speak definitely on this point as yet. It is, however, generally stated† that there is less difference in temperature between the two sides when the paralysis depends on cortical than on central disease. It is certainly less marked than in paralysis due to mesencephalic lesions. Experiments have been made on animals, with a view to determine the question; but the results do not seem quite in harmony with each other. Eulenburg and Landois state that vaso-motor paralysis occurs in dogs, in consequence of destruction of the cortical motor centres in this animal, and in this they are supported by Hitzig. Vulpian, however, fails to detect such phenomena. And it would appear, from a preliminary communication by Küssner,‡ that, in rabbits at least, no vaso-motor paralysis occurs from cortical lesions. It may be that the representation of the vaso-motor centres in the hemispheres follows the same laws as the motor centres in different orders of animals, and that the vaso-motor paralysis, at first resulting from cortical lesions, subsides more rapidly than when the lesion is central; and this seems to me highly probable. That vaso-motor should accompany motor paralysis from cortical lesions is, I think, what might be expected, from what we know of the physiology of motor and its correlated vaso-motor innervation; and that such vaso-motor paralysis should subside more rapidly in cases of cortical lesion would, I think, be in harmony with the comparative escape of movements independently organised in lower centres. To the correlated vaso-motor disturbances, I would ascribe the subjective sensations of numbness, tingling, and the like, which so frequently

* For these various statistics I am indebted to Küssmaul, *Die Störungen der Sprache*, Ziemssen's Handbuch, vol. xii, Anhang. 1877.

† *Lancet*, 1873, vol. ii, p. 449.

‡ *Berliner Klinische Wochenschrift*, 1871.

§ *Acad. de Médecine*, November 1876.

|| *Op. cit.*

* *The Medical Examiner*, April 5th, 1877.

† Bastian, *Paralysis from Brain-Disease*.

‡ *Centralblatt für die Medicin. Wissenschaften*, November 10th, 1877.

usher in paralysis or spasm in connection with lesions of the motor centres, rather than look upon them as projected central sensory irritation.

Hemiplegia, complete from the first and permanent, is not, however, the most common type of paralysis depending on lesion of the cortex or subjacent medullary fibres. More frequently, paralysis of cortical origin is fractional or dissociated, or is a succession of dissociated paralysees or monoplegiae. In cortical affections, we frequently find a hemiplegia, at first complete, resolving itself into a monoplegia; or a monoplegia becoming a hemiplegia by progressive advance of the disease to other motor centres. This latter is a significant indication of cortical disease. Paralysis of voluntary motion of the arm and leg; of the arm and face, or this combined with aphasia, if the lesion be in the left hemisphere; or paralysis of the inferior facial region; of the arm alone; or of certain movements of the hand and arm; or of the leg alone; without affection of sensation, and without qualitative or quantitative changes in electrical contractility, or direct impairment of nutrition, may be looked upon as depending on lesions of the cortex or subjacent medullary fibres. Monoplegia is very often associated with monospasm or early rigidity of the paralysed limb, or of the muscles governed by the centres surrounding the lesion. Sometimes the paralysed limb may remain motionless while convulsions occur in the others. Cortical paralysis is frequently erratic and transitory, more especially in connection with superficial meningo-encephalitis; appearing and then vanishing, first on one side and then on the other. According as the lesion is superficial, or deep, and invading the whole depth of the cortex and subjacent medulla, we get transitory paralysis, or a paralysis which remains permanent, and is followed by descending sclerosis and late rigidity.

Whereas early rigidity is of frequent occurrence in cortical disease, it is rare in central cerebral disease; and then particularly, it would seem, when the medullary fasciculi of the fronto-parietal region are irritated—a condition generally found in connection with hæmorrhagic effusions into the lateral ventricles. Consciousness is less frequently lost in cases of sudden cortical lesion than when similar disease occurs in the central ganglia. This is to be accounted for by the special tendency in the latter case to sudden displacement of the cerebro-spinal fluid, and, through this, general disturbance of the cerebral circulation, in the manner indicated by Duret.

As an accessory element in the diagnosis of cortical lesions, may be taken the fact, noticed by Callender* and others, that cortical lesions are more frequently accompanied by localised pain in the head; and I have frequently observed that, even when pain is not spontaneously complained of, it may be brought out by percussion over the seat of lesion.

While we cannot be quite certain of the position or extent of a cortical lesion causing a sudden and complete hemiplegia, we may take a monoplegia of the leg, or of the arm and leg, as an indication of lesion of the upper extremity of the ascending convolutions close to the longitudinal fissure; brachial monoplegia, as a sign of lesion of the upper part of the ascending frontal convolution, or, if the paralysis affect the hand more particularly, of the ascending parietal convolution; brachio-facial monoplegia as indicating lesion of the mid-fronto-parietal region; while facial and oral monoplegia, or this combined with aphasia, indicates lesion of the lower part of the ascending frontal convolution, where the third frontal unites with it.

SURGICAL MEMORANDA.

CATGUT DRAINS.

MR. PLAYFAIR is, I have no doubt, correct in his criticism upon my views as to the physics of capillarity, and I am quite ready to accept his statement; but he will allow me to say that the experiment to which I referred was one likely to lead to the view I took of the subject. More explicitly, the experiment referred to was of the following nature. One end of a skein of catgut, not tied together, and therefore with the separate strands at some distance from one another, was placed in an urine-glass filled to the brim with water. The other end hung over the side of a second glass of the same height. In a little time, the water began to run over; but after about an hour the flow completely and finally ceased. The water in the full glass had sunk about a quarter of an inch. I do not venture to argue the physics of the question; but such are the facts. This experiment was performed in one of the wards of the infirmary, and was watched by several of the students.

S. MESSENGER BRADLEY, Manchester.

* Barth. Hospital Reports, 1869.

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.

GENTLEMEN,—It has long been a heavy burden on my conscience that, while you are receiving your instruction in physiology here, you are told but little of the history of science—that your attention is only rarely and incidentally called to the various steps by which we have reached our present outlook. I fear that such historical statements as do reach you are sadly limited to the controversies of the last decennium. The names you hear quoted are too often those of the authors of the most recent memoirs and of the last new contradiction; and I shall probably be going not far wrong when I say that, were your knowledge limited to what is uttered here, you would be almost justified in supposing that, except for the changes embodied in about the last dozen volumes of Pflüger's *Archiv*, Physiology might have been, like man, created in its present form in the Garden of Eden. This state of things, though undesirable, is, in large measure, unavoidable. Our time is so short, and the body of knowledge we have to traverse so great, that it is absolutely impossible for us to devote an hour's study to the history of our science without foregoing a corresponding quantity of the science itself; and, on the whole, we do well to choose the knowledge itself rather than the story of how we have come into possession of it. The recent death of a great physiologist, Claude Bernard, has, however, determined me to spend the whole of to-day's lecture in pointing out to how large an extent the modern doctrines of physiology have been influenced by his brilliant investigations.

It is not my purpose to dwell upon his personal history; indeed, there is happily but little to tell. Men of science are fortunate when their personal histories are uneventful; they live in their work and not in their lives. And Claude Bernard was in this sense fortunate. All I know of him is that he was born in the sunny South of France, at Saint-Julien, near Villefranche (Rhône), on July 12th, 1813; and that, when nineteen years of age, he came to Paris as an aspirant for literary fame, carrying in his pocket an unaccepted tragedy and a comedy which had enjoyed an obscure provincial success. Arrived in Paris, he was told by a judicious friend (Saint-Marc Girardin) that "poetry was a bad trade, but might be made a profitable amusement".* Other lovers of poetry in general, and of their own in particular, have at divers times received the same valuable advice. Bernard was in some respects singular, in that he obeyed that advice to the letter. The transition from dramatic poetry to the dissecting-room is great and startling, but he did not shrink from it; he became a medical student. In 1839, he became *interne des hôpitaux*. So far, he had given no token of his future eminence; but, in 1841, he was made assistant to Magendie, and then it was that he found his calling.

It is impossible to understand Bernard's life unless the character and influence of Magendie be rightly appreciated; and here, again, I fear that, save in the matter of the functions of the roots of spinal nerves, the name of Magendie conveys no particular ideas to your minds; and yet it occupies a most honourable place in the history of physiology. Magendie was born in 1783 and died in 1855, so that the lives of himself and his pupil Bernard extend over just about a century. During that century, the progress of physiology has been immense; and if I were to ask any of you what has been the distinguishing token of the physiology of this nineteenth century, you would answer "Experimental investigation, the application of chemical and physical knowledge and methods to the solution of biological problems". Now, Magendie was *par excellence* an experimentalist; his whole life was a prolonged effort to unravel physiological phenomena, by the help of chemical and physical methods, applied in a direct experimental manner. He is, in a certain sense, the father of modern experimental physiology. You must remember that, in his time, Germany did not possess the institutions and appliances of which she is now justly proud, and which

* Paul Bert, Preface to *La Science Expérimentale*, par Claude Bernard. Paris, 1878.