anthrax bacilli, was inoculated with a minute quantity of anthrax spores from the same culture as had been used in preparing the bacilli employed. In twenty-four hours a copious growth was already apparent. Similarly, with blood serum, Lubarsch[20] has found that not only are spores unharmed by it, but they even produce a vigorous crop without any obvious delay in their development. Apparently, in the act of developing, they acquire tolerance of the harmful influence of the medium. Thirdly, the fact discovered by Buchner that dialysis of blood serum into distilled water destroys, while dialysis into normal salt solution does not destroy, its bacteria-killing power, is a clear indication that the latter is a globulin, since such a body would be precipitated by dialysing away the salts that enabled it to be dissolved. This discovery of Buchner's suggests the presence of a globulin as the bacteria-destroying agent, which most probably is identical with the cell globulin that I have worked with.

My results suggest a possible explanation of Wooldridge's work on immunity against anthrax.[21] He cultivated the bacillus in a solution of tissue fibrinogen, and found (1) that if a scanty growth of the anthrax had taken place, the filtered liquid was capable of producing immunity against anthrax when injected into rabbits; (2) that if the liquid produced a copious growth, no immunity could be produced by means of it; (3) that the growth of the anthrax altered the fibrinogen, causing it to clot easily on heating. This form of fibrinogen can also be obtained by dissolving it in a very slightly alkaline liquid. His conclusions were (1) that the immunity-giving power of the solution has nothing to do with any metabolic products of the microbe, because it is not proportional to the intensity of the anthrax growth; (2) that it is due to a change produced in the fibrinogen by this growth. Therefore, since the only change he knew of was an increase in the tendency of the fibrinogen solution to clot on boiling, he tried the effect of injecting into rabbits an easily coagulable fibrinogen solution in which anthrax had not previously been grown. He found that by this means he was able to lengthen the incubation period of the disease to a week, and in two cases the animals recovered. As regards the experiments in which immunity was produced by means of a filtered culture, in which the anthrax had grown badly, it is probable that he was injecting a liquid containing some fibrinogen and a trace of the anthrax-albumoses. Consequently immunity was produced. He failed to get immunity with those culture fluids in which the anthrax had grown well, because in all probability a large quantity of the albumoses having been produced, he injected more than the requisite immunity-producing dose. That this is likely follows from the facts that I published in the JOURNAL of October 12th, 1889; for I have found that the quantity of anthrax albumoses necessary to produce immunity is extremely minute,[22] and any larger dose (with anthrax inoculation accompanying it) will infallibly be followed by the death of the animal. As regards the increased power of resistance that the rabbits seemed to show after injection of a simple tissue fibrinogen solution, is it too much to suppose that we are here dealing with a defensive proteid?[23] Apparently Wooldridge himself had some difficulty in getting the anthrax bacilli to grow in it. I have completely failed to do so when trying to repeat his experiments. He only obtained these results by injection of a fibrinogen solution that had been but little changed by the addition of traces of caustic alkali; only by adding more caustic alkali (and so still more changing its original nature, as shown by the loss of coagulability on boiling) did he succeed in obtaining a typical culture.

A paper recently published by Fokker[24] describes experiments that lead the author to believe that fresh milk has a bacteria-killing power. Fokker finds that by boiling the milk for a short time this power is completely destroyed, and that, as with blood serum, the bacteria-killing power vanishes in the act of killing the microbes, the primary decrease being followed by an increase in the number of living bacilli present, and the milk rapidly becoming the excellent culture medium that experience proves it to be.

These facts would seem to show that a defensive proteid is present in milk, and it would be worth investigating from this point of view why some microbes when grown in milk precipitate the casein by means of a rennet ferment, but yet do not, so far as is known, redissolve and use it at a later stage.

The fact that I have succeeded in obtaining immunity with the anthrax albumoses is a proof that these bodies form one of the factors in the conflict between the organism and the microbe. If ever it could be shown that cell-globulin has, or tends to have, a similar action in the living body to that which it has in a test-tube, the question would arise as to whether the anthrax bacillus defends itself from the cell globulin by means of its albumoses. For this possibility of an albumose destroying a ferment an interesting parallel can be found. Mr. Dickinson, of Caius College, Cambridge, has recently proved that the active principle of leech extract is an albumose[25]; and just as the leech albumose destroys fibrin ferment, so is it possible that anthrax albumose destroys the ferment-like cell globulin.[26] Whether or not this is the case I hope to be able shortly to decide, so far as this can be done by a test-tube experiment.

[20] *Loc. cit.*

[21] "Versuche über Schutzimpfung auf chemischem Wege:" *Archiv für Anatomie und Physiologie; physiologische Abtheilung*, 1888, p. 527.

[22] About one-millionth of the body weight for mice, and from one-five-millionth to one-ten-millionth for rabbits. My reason for trying to get immunity with these very minute doses after a long series of failures with larger ones was derived from the analogy of the albumose of snake poison, which is also active in such exceptionally small doses. It is stated that one-thirteen-millionth of its body weight of cobra poison is sufficient to kill a mouse.

[23] In my Royal Society paper I have described the effects of the intravenous injection of cell globulin after inoculation with anthrax. In certain cases a prolongation of the incubation period took place. The appearance of phagocytosis, and signs of degeneration which were seen in the bacilli after the death of the animal, pointed to an increase of its power of resisting the microbe.

[24] "Ueber die bacterienvernichtenden Eigenschaften der Milch," *Fortschritt der Medicin*, January, 1890.

[25] I have Mr. Dickinson's kind permission to mention this interesting fact. He is about to publish a paper dealing with the question.

[26] Cell globulin deserves the appellation "ferment-like," not only because of its ferment action in causing blood to clot, but also because of the difficulty met with in rendering it insoluble by the prolonged action of alcohol.

THE CROONIAN LECTURES
ON
CEREBRAL LOCALISATION.

Delivered before the Royal College of Physicians of London.

BY DAVID FERRIER, M.D., LL.D., F.R.S.,
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LECTURE VI.—MOTOR CENTRES.

MR. PRESIDENT AND GENTLEMEN,—I now come to the consideration of the physiological signification of the Rolandic area of the monkey and man, and its homologues in the brain of the lower mammals. I have already described with some detail the movements which are capable of being excited by electrical stimulation of the different regions included within this area. How these movements are to be interpreted is a subject on which there are great differences of opinion. The definite purposive character of the movements, however, their correspondence with the ordinary volitional activities of the animals, and, above all, their uniformity and predictability, harmonise best, in my opinion, with the hypothesis that they are indications of the functional excitation of centres directly concerned in effecting volitional movements, and anatomically a part of the motor apparatus.

It has been established by experiments on monkeys—and now so generally admitted that it is almost unnecessary to enter into detailed proof—that destruction of the centres, excitation of which produces definite movements causes paralysis (*qud* volition) of the same movements on the opposite side of the body, varying in degree, completeness, and duration with the extent of the destruction of the respective centres. When the destruction is complete, the paralysis is permanent, and is followed in due course by descending degeneration of the pyramidal tracts of the spinal cord, and secondary contracture in the paralysed limbs. As an illustration I quote the following experiment on a monkey which was exhibited at the International Medical Congress in London, 1881, eight months after the operation.

The cortex was destroyed as shown in the figure (Fig. 33), in the left hemisphere over an area embracing the ascending frontal and ascending parietal convolutions, except at their upper and lower extremities. The lesion also invaded the base of the superior frontal convolution, and the anterior limb of the angular gyrus. There was thus destroyed nearly the whole of the motor area on the convex aspect of the hemisphere, the centres for the leg, foot, and trunk being only partially destroyed; those

for the angle of the mouth and tongue almost entirely escaping. The result of this lesion was almost complete right hemiplegia with conjugate deviation of the head and eyes to the left side. As in similar cases in man, the deviation of the head and eyes was only of comparatively short duration, and the partial facial paralysis, at first perceptible, also disappeared within a fortnight; but the paralysed condition of the limbs continued very marked. With the exception of slight power of flexion of the thigh and leg, the right lower extremity was helpless, and the right arm was incapable of independent volitional movements. Occasionally, when the animal struggled, associated movements were observed in the right hand, similar to those initiated by the left,

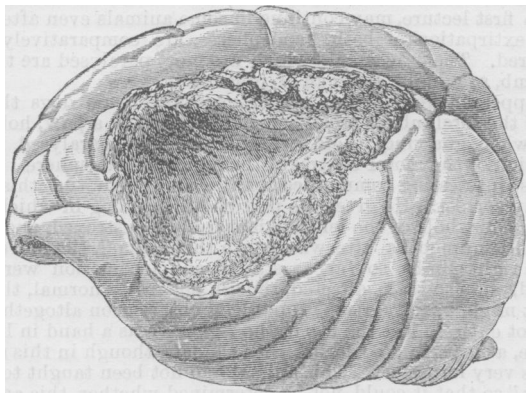

Fig. 33.

but only under such circumstances. The power of prehension was entirely annihilated. Cutaneous sensibility was unimpaired throughout. The slightest touch excited attention; and a pinch, or other painful stimulus, caused signs of sensation quite as vigorous as on the other side. This was the condition in which the animal was when exhibited at the International Medical Congress, and at this time well-marked contracture had become established in the paralysed limbs, with exaggeration of the tendon reactions, as in cases of incurable cerebral hemiplegia in man.

The investigation of the brain of this animal was carried out by a committee appointed by the Physiological Section, and the position of the lesion in the motor zone, and its limitation to the cortex and subjacent fibres, were definitely proved by them. Microscopical investigation also demonstrated the existence of secondary degeneration in the pyramidal tracts of the right side of the spinal cord as far as the lumbar region.

In the case represented in Fig. 34 the lesion, which was estab-

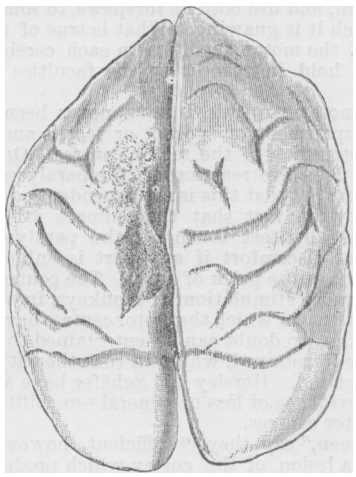

Fig. 34.

lished at the upper extremity of the fissure of Rolando in the left hemisphere, caused paralysis of the right leg, without affection of sensation, followed in due course by contracture of the paralysed muscles. This condition remained unchanged for eight months, at the end of which time the animal was killed. In this case also secondary degeneration was demonstrated in the medullary fibres of the corona radiata, and in the pyramidal tracts of the opposite side of the spinal cord as far as the lumbar region, whence emerge the motor nerves of the lower extremity.

In another experiment the cortex was destroyed in the middle of the ascending parietal convolution and adjacent margin of the ascending frontal convolution of the right hemisphere. The result of this was almost complete paralysis of the left hand, and great weakness of the flexor power of the forearm. The shoulder movements, however, were unimpaired; the animal could stretch its arm forward, but could not grip with its hand what it wished to lay hold of. Tactile sensibility was absolutely unimpaired in the paralysed limb, the slightest touch on it at once exciting the animal's attention, and a painful stimulus, such as a pinch, or touch with a heated wire, caused as lively signs of sensation as on the other side. This condition remained essentially unchanged for the two months which the animal survived the operation.

Many similar experiments have been recorded by Horsley and Schäfer,[1] and their observations on the functions of the marginal gyrus deserve special note. Extirpation of the marginal convolution causes paralysis of those movements which remain more or less unaffected after the destruction of the centres on the convex aspect of the hemisphere; namely, movements of the trunk, those of the hip muscles, as well as some of those of the leg, In order, however, that these movements should be entirely paralysed, it is necessary that the marginal convolution should be destroyed in both hemispheres; as it would seem that the trunk movements are so bilaterally co-ordinated in the marginal convolution, that the removal of one only is not sufficient to cause any very marked effect. When both are removed, however, the most absolute paralysis of the trunk muscles is induced. "The attitude and general appearance of a monkey in which this double lesion has been produced are very striking. Instead of sitting up with back somewhat curved, in the manner normal to monkeys, an animal which has been submitted to this operation lies prone, with legs and feet outstretched (or, at most, with flexed hips), back flat, tail straight and motionless, and arms put forward to clutch at any neighbouring object. The head retains its power of rotation, as well as flexion and extension, and the movements of the eyes and facial muscles appear normal. The animal frequently props itself upon its elbows, but never assumes the normal sitting attitude. If the monkey desires to sit up, it can only do so by dragging itself into the sitting posture by its arms and hands, and holding on by these to the wires of the cage, or to any neighbouring object. If the hold should be detached the animal immediately tends to fall over. Progression is effected almost entirely by the arms, the monkey dragging itself along with the aid of these, assisted by the flexion which occurs at the hips; the legs are quite limp and draggled, the dorsal surface of the toes being drawn over the ground."[2]

Besides the movements of the trunk, there are others which are also bilaterally represented in each cerebral hemisphere. This holds in respect to the upper facial region, as well as those of the larynx. Hence, unilateral extirpation of the centres of these movements causes no, or scarcely any, perceptible impairment; and it is necessary that the centres should be destroyed on both sides in order that paralysis should result. It has been shown by Krause in dogs, and by Horsley and Semon in monkeys, that unilateral extirpation of the laryngeal centres does not appreciably impair the adduction of the vocal cords; whereas phonation becomes volitionally impossible when the centres are destroyed in both hemispheres.

It would appear from the researches of Franck and Pitres,[3] Exner,[4] Lewaschew,[5] Sherrington,[6] that such movements as are not primarily bilaterally represented in each cerebral hemisphere are secondarily associated, in accordance with a hypothesis originally advanced by Broadbent, by commissural fibres connecting the bulbar and spinal nuclei with each other. Though moderate stimulation of the cortical centres of the limbs gives rise to movements as a rule only on the opposite side, yet it not infrequently happens, if the stimulation be increased, that movements occur in the limbs on both sides. These are, however, more pronounced on the opposite than on the same side. In the monkey

[1] *Phil. Trans.*, B. 20, 1888.
[2] See illustration, Fig. 20, *op. cit.*
[3] *Leçons sur les Fonctions Motrices du Cerveau*, 1887.
[4] *Sitzungsb. d. Wiener Akad*, 3 Abth., pp. 185-190, 1881.
[5] *Archiv f. Physiologie*, Bd. 36.
[6] *Journ. of Physiology*, Nos. 4, 5, and 6; and JOURNAL, January 4th, 1890.

as well as in man, it is not unusual to find descending degeneration in both lateral columns as the result of unilateral cortical lesions.

According to the recent researches of Sherrington, if the cortical lesions affect only the centres for the limbs, bilateral degeneration does not occur, to any extent at least; but this is very pronounced if the lesions affect the marginal convolution. The degeneration is confined to the pyramidal tracts on the same side as far as the decussation of the pyramids, but becomes bilateral in the spinal cord. In the case, however, of the more obviously bilateral laryngeal centre, degeneration is well marked in the pyramids of both sides. These facts, as well as clinical observations in man, show that, even in the case of the limbs, each hemisphere represents both sides of the body, mainly the opposite, but to some extent also the same side.

It was first pointed out by Brown-Séquard, and his observations have been confirmed by Pitres[7] and Friedländer[8], that lesions which induce hemiplegia of the opposite side also cause some diminution in the energy of the movements of the limbs on the same side. This is a result which we should expect, if each hemisphere were in relations with both sides of the body.

The bilateral relations of each hemisphere, which exist to some extent in monkeys and man, are, as we shall see, still more pronounced in the case of dogs and the lower animals; and, in dogs, as Sherrington has shown, bilateral degeneration from unilateral cortical lesion is more commonly met with. This bilateral representation accounts for a certain amount of recovery, even when the motor centres of one hemisphere have been entirely destroyed; and this recovery extends more particularly to those movements of the limbs which are more or less habitually associated with those of the other side, and least of all, to those which are more independent and more volitional. Hence, in cortical paralysis, the arm is more paralysed than the leg, and the distal movements of the arm more than the proximal. These facts are of great importance in reference to the hypothesis of the functional compensation for the centres which have been destroyed by neighbouring, or other, portions of the same cerebral hemisphere. It has now been established beyond all question that cortical lesions of the motor zone in man, if they are such as actually to destroy and not merely push aside the grey matter of the respective centres, invariably cause paralysis of volitional motion in the related parts. Such results have invariably followed, not only the destructive lesions of disease but also surgical excisions of the cortical centres. Not only does general hemiplegia result from lesions of the whole of the Rolandic area but limited lesions cause limited paralyses, or monoplegiæ, of the face, arm, and leg, in precise correspondence with the results of experiments on monkeys.

In the Gulstonian lectures on Localisation of Cerebral Disease, which I had the honour of delivering in this College twelve years ago, I brought before you a number of cases, collected from various sources, in support of these statements. Since then many others have been recorded, all confirming the same; and the point has now been considered so completely proved, that clinical observers have practically ceased to record their observations.

Of 483 cases of cortical (including subcortical) disease collected under my direction by Dr. Ewens (excluding as a rule tumours and other lesions likely to cause indirect affection of other parts), I have the records of 110 cases of hemiplegia of the opposite side from general lesion of the Rolandic zone; and 90 cases of monoplegia from limited lesions of this zone. Of these, 11 are cases of crural monoplegia, from disease of the paracentral lobule; 15, cases of paralysis of the arm and leg from disease of the paracentral lobule and upper third of the ascending convolutions; 33, cases of brachial monoplegia, including three cases of surgical excision, from lesion of the middle of the ascending convolutions; 19, cases of paralysis of the arm and face from lesions of the lower half of the Rolandic zone; and 10, cases of facial paralysis from disease of the lower third of this region. In addition to these, I have notes of 20 cases of atrophy of the cortex of the Rolandic zone in connection with congenital or infantile hemiplegia, or as the result of congenital absence, or long-standing amputation, of a limb.

In the monkey and man, therefore, there is no evidence of functional compensation for the paralysing lesions, except in so far as these may be accounted for by the bilateral relations of each cerebral hemisphere. The theory of compensation by other portions of the same hemisphere has been adduced more particularly in order to account for the apparent recovery in dogs and lower animals after complete unilateral destruction of the cortical motor centres. This is a hypothesis, however, which is inconsistent with the principles of localisation otherwise maintained by those who advance it; and for which, moreover, there is no necessity. Though dogs appear to recover from the disorders of movement, which are at first obvious enough after unilateral extirpation of their motor centres, yet, in reality, the recovery is never complete. Only those movements are permanently affected which are the least automatic and most volitional; while those which are most automatic and least volitional, such as those concerned in station and co-ordinated locomotion, and which, as we have seen in the first lecture, may continue in some animals even after complete extirpation of both hemispheres, are comparatively little impaired. The movements which are most paralysed are those of the limb, as a hand, or organ of prehension.

It appeared from Goltz's earlier experiments on dogs that the use of the forelimb as a hand, such as in giving a paw, holding a bone when gnawing it, etc., was permanently paralysed by destruction of the motor centres of the opposite hemisphere. But in his last interesting communication[9] he has shown that this is not strictly correct. He gives the particulars of a dog in which practically the whole of the left hemisphere was destroyed, and which lived fifteen months afterwards. In this animal, the movements of the right limbs, so far as station and locomotion were concerned, were so little affected, though not really normal, that the defect might readily escape superficial observation altogether. It had not entirely lost the use of the right paw as a hand in holding a bone, at least in association with the left, though in this respect it was very imperfect. This animal had not been taught to "give a paw," so that it could not be determined whether this acquired trick could still be exhibited, though Goltz is of opinion that, in rare cases, a dog may perform this act after deep and extensive (but probably not complete) destruction of the motor zone of the opposite hemisphere. The possibility of a certain degree of independent, or rather associated, volitional use of the opposite limbs is, as Goltz's experiments clearly demonstrate, dependent on the integrity of the motor centres of the uninjured hemisphere; for when these are destroyed on both sides, all strictly volitional movements are permanently paralysed. Thus he says: "A dog whose motor centres of both hemispheres have been destroyed cannot feed itself; the movements of the tongue are greatly impaired, while the tongue, in a case of unilateral extirpation, can be moved normally. The animal can, as already mentioned, still walk, but in a very awkward and unsteady manner. All movements of the paws as hands appear to be utterly impossible."[10] This powerlessness depends on the symmetrical destruction of the motor centres in both hemispheres; for, if the motor centres on the one side, and the occipital regions on the other, are destroyed, the dog can eat and drink and move its tongue, walk and run about fairly well, and use both its forepaws, to some extent, in fixing a bone which it is gnawing. What is true of the bilateral representation of the motor faculties in each cerebral hemisphere appears also to hold in respect to the faculties of general and special sense.

Apart from motor paralysis, I have never been able to detect the slightest impairment of special, or tactile and general sensibility after destruction of the motor centres. One may observe absence of, or defective reaction of, the paralysed limbs to sensory stimulation; but that this is not dependent on defective sensation is shown by the fact that the animal's attention is at once attracted by the slightest touch on the paralysed side, and by general signs of discomfort if any part is subjected to painful stimulation, such as the prick of a pin. The contrast between the reactions to sensory stimulation in monkeys, in which the falciform lobe, and those in which the motor centres have been removed, is so striking that no doubt can be entertained that, in the latter case, sensibility is retained, while, in the other, it is abolished or profoundly impaired. Horsley and Schäfer have also been unable to obtain any evidence of loss of general sensibility after destruction of the motor centres.

"We have seen," say they, "sufficient, however, to convince ourselves that a lesion of the cortex which produces paralysis of volitional motion in a part, is not necessarily accompanied, also, by loss of general sensibility of the paralysed part."[11] In order to test the hypothesis which has been advanced by some—that the super-

[7] *Archives de Neurologie*, No. 10, 1882.
[8] *Neurologisches Centralblatt*, No. 11, 1883.
[9] *Archiv f. Physiologie*, vol. xlii, 1888.
[10] *Op. cit.*, p. 448.
[11] *Op. cit.*, p. 15.

ficial cells of the motor cortex are sensory in function—they, in one instance, destroyed the superficial layers of the grey matter by means of the actual cautery. "In spite, however, of the complete blocking of the superficial vessels so produced," they go on to say, "we obtained only an incomplete muscular paralysis as the immediate result of the operation; but, although the superficial layers of the cortex must have been destroyed, there was no diminution of sensibility in the parts affected by paresis. The subsequent softening and disintegration which occurred in consequence of thromboses caused by the cautery was accompanied by a much more complete condition of muscular paralysis; but the general sensibility of the opposite side was still apparently unaffected, and continued so until the death of the animal."[12] One of Goltz's experiments on dogs[13] also shows very clearly that destruction of the cortical motor zone does not impair sensibility on the opposite side. Taking advantage of the well-known fact that dogs snarl when touched while engaged in eating, he touched the right side of a dog so occupied, which had had its motor centres in the left hemisphere destroyed some time previously. The animal responded invariably with the characteristic signs of displeasure on the slightest touch. Similar facts have been demonstrated in the case of cats by Bechterew.[14] "It is a familiar observation that a cat dislikes having its feet wet, so that, if it should accidentally step on a wet place, it will stop and shake its paw dry before proceeding further; or if, while indolently slumbering, a drop of water falls on it, it will start up and make off hastily; or it will close its eyes and contract its ears if its paw is gently touched unobserved." After verifying these facts in a cat about to be operated upon, Bechterew removed the cortex in the region of the sigmoid gyrus. On recovery from the chloroform narcosis, the animal exhibited the characteristic motor disorders of the right limbs, and was unable to use the right paw for any independent volitional act, but touching the ear, or the sole of the right as well as the left foot, induced the same closure of the eyes, and drawing in of the ears as before; and the sprinkling of a few drops of water on its paralysed side caused the animal to start up and make off as before.

It has, however, been maintained by various experimenters, Hitzig, Nothnagel, Schiff, Munk, Tripier, Goltz, Luciani, etc., that the affection caused by destruction of the so-called motor centres is accompanied by, or dependent on, affections of tactile, muscular, or general sensibility, or all three combined, in the paralysed limbs.

My own experiments, as well as those of Horsley and Schäfer, have shown that there is no discoverable impairment or loss of tactile or general sensibility after lesions of the motor zone, and I will not therefore stay to examine in detail the data, so far as they have been given, on which the various authors whom I have mentioned, found their conclusions. They appear to me to be based merely on defective reaction to sensory stimulation, which can be equally well explained on the theory of motor inability as sensory deficiency; or they are due to the destruction of other regions than the motor cortex. This applies more particularly to the experiments of Goltz, in which the lesions of the hemisphere, or hemispheres, have been vague and indeterminate. It is unquestionable also that in man paralysis from lesion of the motor area is, in the majority of instances, an essentially motor affection, and is unaccompanied by any discoverable defect of tactile, muscular, or general sensibility. I have myself recorded several, and have collected many other cases of lesion of the cortical motor zone accompanied by paralysis, in which every variety of sensibility has been carefully tested and found normal. But it is true also that in a good many instances of lesion of the motor area, some degree of impairment of tactile and muscular sensibility, general or limited, has been observed. And several authors, Petrina,[15] Exner,[16] Luciani and Seppili,[17] Starr,[18] Dana,[19] Lisso,[20] have endeavoured to show, from examination of the clinical records of cerebral disease, that the motor centres and the centres of tactile and general sensibility coincide, so that sensory disturbances, frequently at least, if not always, accompany the motor paralysis. The data on which these conclusions have been based appear to me in the highest degree unsatisfactory. The lesions have been microscopical specks of themselves insufficient to cause anything; or, and for the most part, tumours which may cause everything; or multiple foci of disease not limited to the cortex itself. Causation is not established unless invariable and unconditional relationship has been shown to exist between a particular lesion and a particular symptom. In the case of the motor area it has been satisfactorily demonstrated that destructive lesions invariably give rise to motor paralysis, local or general, according to the position and extent of the lesion. A single case, therefore, of paralysis from lesion of the cortical motor area, unaccompanied by any defect of sensibility, is sufficient to overthrow a host of positive instances in which the two symptoms have apparently been caused by the same lesion. Apart, however, from all other considerations, an examination of the clinical records taken indiscriminately from all sources, excluding tumours and such lesions as are obviously calculated to cause widespread and indeterminate disturbances of other parts, is certainly not in favour of the conclusions which the above-mentioned authors have founded on this kind of evidence. For, of 110 cases of general lesion of the Rolandic zone causing hemiplegia, in 52 cases, sensibility was unimpaired (in one of these a large portion of the motor cortex was excised).[21] In 37 the condition as to sensibility is not mentioned; while in 21 it is said to have been to some extent affected. But of these last, in one sensibility is stated to have been blunted over the little finger.[22] In another there was general hyperæsthesia, more marked on the paralysed side. In one all varieties of sensibility were retained, but the localisation of touch was somewhat defective.[23]

In this case, however, the inner table of the skull had been driven into the brain substance, causing general hemiplegia. In two the lesion extended deeply into the white substance. In one case the cortical lesion was complicated by the presence of a large tumour in the centrum ovale.[24] In one sensibility is said to have been blunted on both sides of the body. In another[25] the lesion was a large hæmorrhagic cyst in both central convolutions. In this, and in five others, the island of Reil or external capsule was involved. In one hemiplegia was accompanied by anæsthetic formication of the paralysed foot. In this case there was tubercular deposit implicating the gyrus fornicatus as well as the central convolutions. In seven others there was diffuse meningo-encephalitis or tubercular meningitis. In ten cases of crural monoplegia, from disease of the paracentral lobule, cutaneous sensibility was unaffected in six, its condition not mentioned in two, and affected in two. In the one[26] sensibility to pain was a little diminished in the paralysed limb, but this disappeared the next day. In the other[27] the leg became gangrenous, a condition which was preceded by anæsthesia. Of fifteen cases of paralysis of the arm and leg from disease of the paracentral lobule and upper third of the ascending convolutions, sensation was intact in six, not mentioned in five, and affected in four others. In three of these the paracentral lobule was deeply involved, one of them being a tubercular mass; and in only one was the anæsthesia marked or permanent. In all four the lesion was in immediate proximity to, or actually involved, the gyrus fornicatus. In one case, under my own care,[28] of traumatic cicatrix at the upper third of the ascending frontal convolution, excision of the lesion was followed by loss of tactile sensibility on the dorsum of the two distal phalanges, and inability to indicate the position of the fingers of this hand. This impairment of sensibility ultimately disappeared, while the motor paralysis continued as at first. In this case the lesion was such as to actually implicate the gyrus fornicatus.

Of 35 instances of brachial monoplegia, 5 were cases of excision of portions of the cortex for the cure of focal epilepsy. In two, cases by von Bergmann[29] and Keen[30] sensation was intact. In another reported by Keen,[31] of hemiplegia and epilepsy, resulting from case, depressed fracture, there was, after the operation, slight impairment of sensation in the middle of the forearm and two inner

[12] *Op. cit*, p. 17.
[13] *Pflüger's Archiv*, Bd. 34, 1884, p. 465.
[14] *Pflüger's Archiv*, Bd. 35, 1885, p. 137.
[15] Sensibilitätsstörungen bei Hirnrindenläsionen, *Zeitsch. f. Heilkunde*, Bd. ii, p. 375, 1881.
[16] *Localisation der Functionen in der Grosshirnrinde des Menschen*, 1881.
[17] *Die Functions-Localisation auf der Grosshirnrinde*, 1886.
[18] Localised Cerebral Disease, *Amer. Jour. Med. Sc.*, 1884.
[19] *Cortical Localisation of Cutaneous Sensations*, 1888.
[20] *Zur Lehre von der Localisation des Gefühls in der Grosshirnrinde*, 1882.
[21] Case of J. H., *Brain*, vol. x, p. 95.
[22] Tripier, *Rev. Mens.*, 1880, Case 4.
[23] Bramwell's Case, JOURNAL, August 28th, 1875.
[24] Seguin's case, *Trans. Amer. Neurol. Assoc.*, 1877, p. 115.
[25] Starr's case, 75, *Amer. Journ. Med. Sciences*, July, 1884.
[26] Gouguenheim, *Soc. Méd. des Hôpitaux*, 1878, p. 48.
[27] Ballet, *Archives de Neurologie*, Tome v, p. 281.
[28] Case of J.B., *Brain*, vol. x., p. 26.
[29] *Archiv. f. Klin. Chirurg.*, p. 864, 1887.
[30] *Amer. Journ. Med. Sc.*, 1888, Case 3.
[31] *Ibid*, Case 2.

fingers. But this condition of sensibility was similar to what had existed before the operation. In another[32] there was no obvious impairment in tactile (?) or muscular sensibility. The patient could not distinguish the form of objects owing to the inability to move his fingers. In a fifth case[33] the removal of a tumour from the right lower parietal region, which was the cause of epilepsy beginning in the thumb, was followed by tactile anæsthesia of the whole of the left side, together with loss of so-called muscular sense in the left arm. In this case the sensory tracts for the whole of the opposite side of the body were obviously implicated. Of the 30 others, sensibility was unimpaired in 12, not mentioned in 15, and affected in 3. In one of these the lesion was a gumma.[34] In the second[35] there was a clot compressing the island of Reil; in the third sensibility was said to have been extinguished over the entire surface of the body.[36] Of 19 cases of lesion of the lower half of the Rolandic zone, causing paralysis of the face and arm, sensation was unaffected in 11, not mentioned in 5, and affected in 3. In one of these,[37] however, a blood clot in the island of Reil compressed the subjacent convolutions. In another reported by the same author[38] a small tubercle the size of a hemp seed, situated in Broca's convolution, is described as having caused (!) paralysis of the right side of the face and arm, and anæsthesia of the right side of the trunk. The third, also reported by Petrina, was similar to the second. In 10 cases of disease of the lower third of the Rolandic zone, causing simple facial paralysis, sensation was intact in 4, not mentioned in 5, and affected in 1. In this case[39] there was said to have been anæsthesia, not only of the face, but of half the trunk.

It thus appears that, of 284 cases of lesion affecting the Rolandic zone, general or in part, in 100 the condition of sensibility was not mentioned; in 121 it was stated, and by many of the most reliable clinical observers, to have been intact; and in many of these[40] all varieties of sensibility are expressly stated to have been carefully investigated. In the remaining cases no detailed notes are given as to the different modes of sensibility, and the methods applied for testing them. In 63, some impairment of sensibility was noted. In 28 of these, the lesion was not confined to the Rolandic zone, but implicated adjacent lobes, especially the parietal. The remaining 35 have already been analysed, and it has been shown that, in the majority at least, conditions existed which were calculated to implicate either the sensory centres in the gyrus fornicatus, or the sensory tracts of the internal capsule. Even where these cannot have been demonstrated to exist—and I freely admit that there are such cases—it is more logical to assume that they may have existed than that in some individuals the tactile and motor centres should coincide, while in others this should not be the case. I do not consider that the sensory aura which occasionally precedes, or accompanies a localised epileptiform spasm can be taken as a proof that the motor and sensory areas coincide. It may prove contiguity, functional or anatomical, but not coincidence. For the most careful investigation in a large number of cases has failed to detect the slightest impairment of any of the forms of general sensibility, while the motor affection has been of the most pronounced character. There is also no relation between the degree of affection of sensibility and that of the motor paralysis. The motor paralysis has been absolute, while the affection of sensibility has been slight, and confined to one, or at most two or three, fingers; or the motor paralysis has been limited, while the impairment of tactile sensibility has been general. And in others the affection of tactile sensibility, at first observed, has disappeared, while the motor paralysis has remained. And when, moreover, we take into consideration the fact that tactile and muscular sensibility may be abolished in the absence of motor paralysis, a condition which can be experimentally produced in monkeys by lesions of the falciform lobe, we have a further proof that the motor and sensory centres of the cortex are anatomically distinct from each other, and that we cannot attribute the motor paralysis to any defect in tactile or muscular sense. The occurrence of slight defects in tactile and muscular sensibility, more particularly in the fingers, which have been noted by several as a special characteristic of lesions of the cortical motor zone, are, in my opinion, to be looked upon as the beginning or remnants of a general hemianæsthesia, rather than as indicative of special centres for the tactile and muscular sensibility of the digits in the motor cortex.

In illustration of this, I would mention the details of the following case. The patient was a lady aged 50, suffering from word-blindness and a slight degree of right hemiopia, which, from these and other symptoms, I diagnosed to be due to a tumour in the region of the angular gyrus. There was no paralysis of motion, but there was slight impairment of the localisation of touch and the sense of position in the fingers of the right hand, the face and leg being normal in this respect. An operation was undertaken by Mr. Horsley for the removal of the tumour, but it was found on trephining to be situated beneath the angular gyrus, and could not safely be removed. This was a case in which, without doubt, the sensory tracts of the internal capsule were implicated, but only to a slight extent; hence the limitation of the anæsthesia to the fingers. Had the implication of the internal capsule been more extensive, there would undoubtedly have been loss of tactile and muscular sensibility on the whole of the opposite side of the body.

This case has an important bearing on the hypothesis advanced, among others, by Nothnagel[41] that the centres of the muscular sense are situated in the parietal lobe. Defects of tactile sensibility, and of the sense of position of the limbs, have not infrequently been observed in connection with lesions of this region, sometimes complicated with hemiopia when the lesions have invaded also the occipito-angular region, as in the above case, and in one reported by Westphal.[42] But the real cause of these symptoms I believe to be implication of the sensory tracts of the internal capsule which lie underneath this region, and not affection of the cortex itself; for lesions of the inferior parietal lobe do not, in my experience, produce the slightest affection of general sensibility on the opposite side of the body.

Cortical lesions of the motor zone causing complete paralysis may occur without any impairment whatever of the muscular sense, and loss of the muscular sense may occur without motor paralysis. I agree with Bastian, James,[43] and others—and have furnished experimental proof thereof—who hold, in opposition to the views maintained by Bain, Wundt, and Hughlings Jackson, that the sense of movement, its range and direction, are dependent upon in-going, or centripetal, impressions conditioned by the movement itself, and not on the out-going current or energising of the motor centres.

We have, I believe, no sense of innervation independently of the sensory impressions arising from the parts which are moved. The energising of the motor centres and motor apparatus is revealed in consciousness only through the functioning of the correlated sensory tracts and centres. The idea or conception of a movement is, therefore, a revival in the respective sensory centres of the various impressions which have been associated with this particular movement. Of these the most important are the visual factors and those which are included generally under the so-called muscular sense. In learning a movement our chief guide is vision, which enables us to place our limbs in the position requisite to produce any desired effect, and we associate also with the particular movement a distinct set of muscular sense impressions. The revival of these, singly or conjointly, is the idea of the movement, and this allowed to excite the appropriate muscular combination is the volitional act itself.

I hold that the centres of the sensations which accompany muscular action, and which form in part the basis of our ideas

[32] Lloyd and Deaver's case, *Amer. Journ. Med. Sc.*, 1888, p. 477.
[33] Jackson and Horsley, *Brain*, vol. x, p. 93.
[34] Martin, *Chicago Med. Jour.*, vol. 46, p. 21.
[35] Wood, *Phil. Med. Times*, vol. v, p. 470.
[36] Ringrose Atkins, JOURNAL, 1878.
[37] Petrina, *Zeitsch. f. Heilkunde*, vol. xi, 1881, Case 1.
[38] *Ibid.* Case 6.
[39] Petrina, *sup. cit.*, Case 3.
[40] Mills (*Trans Amer. Cong. of Phys.*, etc., 1888, p. 269), (digest in *Brain*, October, 1889); Delépine (*Trans. Path. Soc.*, 1889); Ferrier (*Brain*, April, 1883, p. 67); Moutard-Martin (*Bull. Soc. Anat.*, 1876, p. 706); Laquer (*Inaug. Dissert. Breslau*, p. 91, Case 10); Mills (*University Med. Mag.*, November, 1889); Ferrier (*Brain*, vol. x, p. 95); Raymond et Derignac (*Gaz. Méd.*, 1882, p. 665); Von Bergmann (*Archiv. für klin. Chir.*, 1887, p. 864); Davy and Bennett (*Brain*, vol. ix, p. 74); Ballet (*Archives de Neurol.*, vol. v, p. 275, Case 1); Lloyd and Deaver, *Amer. Jour. Med. Sciences*, vol. 96, p. 477); Keen (Cerebral Surgery, *Amer. Jour. Med. Sciences*, 1888, Case 3). One might add to these cases not a few of hemiplegia with aphasia (not followed by *post-mortem* examination) in which the symptoms pointed to cortical lesion. In one, recently under my care at King's College Hospital, of absolute paralysis of the right arm associated with word-blindness and word-deafness, the patient was aware of the slightest touch on the paralysed hand, or of a drop of warm or cold water falling on it, and with eyes blindfolded could put her left hand on the spot, in whichever position her paralysed arm was placed. This is a method of testing the sense of position in those who are unable to understand or speak, and is also applicable to the lower animals.
[41] VI Congress für innere Medicin, *Neurolog. Centralblatt*, 1887, vol. 6, p. 213.
[42] Zur Localisation der Hemianopsie und des Muskelgefühls beim Menschen, *Charite-Annalen*, 1882.
[43] *The Feeling of Effort*, 1880.

of movement, are distinct from the cortical centres, through and by which the particular movements are effected. The destruction of the cortical motor centres paralyses the power of execution, but not the ideal conception of the movement itself. A dog with its cortical centres destroyed has a distinct notion of the movements desired when asked to give a paw, but it makes only ineffectual struggles and fails to comply. So, too, it not infrequently happens that a patient rendered hemiplegic by embolism of his Sylvian artery only discovers his infirmity by his inability to execute the movements which he has distinctly conceived.

Voluntary movements are capable of being carried out in the entire absence of all sense of movement. In the well-known case described by Schüppel,[44] the patient, anæsthetic from spinal disease, was able to co-ordinate his limbs perfectly, and move them freely and forcibly with the aid of vision; and even without the aid of vision to employ them with a fair degree of precision and steadiness. A similar condition is met with in hemianæsthesia from organic lesion of the sensory tracts of the internal capsule, and in the functional forms described under the name of hysterical hemianæsthesia.

Though the patient is able to move the anæsthetic limb voluntarily, he has no knowledge of its position or of the resistance which may be offered to its intended movement. Bastian,[45] however, maintains that "the rule has been with the hemianæsthetic patients which have been so thoroughly investigated by Charcot at the Salpêtrière that, although there has been complete loss of tactile sensibility, and usually absolute insensibility to pain in the skin and all other sensitive structures on the affected side, together with paresis of the affected limbs, the so-called 'muscular sense' has been nearly always preserved."

On this question I have appealed to M. Charcot himself. He has favoured me with a reply, from which I quote the following: "Cases of hysterical hemianæsthesia may be seen both in men and women affecting only the superficial integument, and without implication of the muscular sense, but the obnubilation, or complete disappearance of the muscular sense—in particular the loss of the sense of position of the limbs—is each very frequent, one might almost say habitual, in hysterical hemianæsthesia, especially when it is accompanied by paresis or hemiplegia..... Up to the present I have not met with serious impairment, strictly limited to the muscular sense in hysteria, unaccompanied by cutaneous hemianæsthesia. It appears from all this that the abolition of the muscular sense represents the highest degree of the hemianæsthetic scale." He refers me also to cases of anæsthesia[46] in which the sense of position of the limbs was entirely abolished, and "yet the patients were able to move the affected members freely even when the eyes were closed. Under such conditions, however, the movements of the limbs deprived of the muscular sense are uncertain and hesitating."

These and similar facts show that the sense of movement is not essential either to the due co-ordination or power of carrying a movement into effect. Vision may entirely replace the muscular sense, though, as one would naturally expect, volitional movements effected only through the aid of vision are, when the eyes are closed, less certain and precise than those which are accompanied also by a sense of movement. Yet these defects are capable of being overcome in large measure by practice; so that, even when the eyes are closed, the visual conception of the movement is capable of compensating entirely, or almost entirely, for the loss of the muscular sense. That this does not occur in all cases may be admitted, but the essential point is that it may in some, and one case of this kind is sufficient to demonstrate that volitional action is not necessarily bound up with sensations conditioned by the muscular action itself.

It is conceivable that ideas of movements might be formed, and volitional movements effected by a brain consisting only of visual and motor centres. Under these circumstances, however, vision would be largely occupied in directing movements, and the range of muscular action and muscular adaptation would be infinitely less than if these were guided, also, by sensations generated by the movements themselves. By the so-called muscular sense, we are able to conceive and execute movements which we have never seen, but we are unable to conceive or volitionally execute movements which we have neither seen nor felt. But though, under ordinary conditions, the sensations of movement are the invariable accompaniment of muscular action, and are repeated as often as the muscular action itself, this constant association does not imply that the one is dependent on the other, or that the musculo-sensory ideas of movement are the necessary or immediate excitants of the movement itself.

Bastian holds that in addition to the conscious impressions which accompany muscular action, and which he admits may be, chiefly at least, localised in the falciform lobe, there are a set of unfelt impressions which guide the motor activity of the brain by automatically bringing it into relation with the different degrees of contraction of all the muscles which may be in a state of action. To these unfelt impressions he gives the name "kinæsthesis," and he considers that the motor centres are the seat of the "kinæsthesis" or sense of movement. The so-called motor centres are, therefore, according to him, in reality sensory centres which excite the true motor centres of the spinal cord through the pyramidal tracts which connect them therewith. I cannot agree with Bastian in including in the muscular sense, which is so essentially an act of conscious discrimination, the mere afferent or unconscious impressions, through the agency of which the harmonious co-ordination of the different segments of the spinal cord and lower centres is secured, apart from the cerebral hemispheres; nor do I think that impressions, which practically do not rise into consciousness, can be ideally revived or enter into the composition of ideas or conceptions of movement. But if it were the case, as Bastian assumes, that the ideal revival of kinæsthetic impressions is the immediate excitant of the true motor centres in the spinal cord, it would follow that the so-called motor centres would be independent centres of activity, irrespective of the stimuli from the sensory centres of the cortex. Experiments show, however, that the motor centres are not independent centres of action, for it has been found by Marique,[47] whose experiments have been confirmed by Exner and Paneth,[48] that when [the motor centres have been completely isolated, by section of the fibres which associate them with the sensory centres of the cortex, paralysis results of precisely the same character as that which occurs when they are actually extirpated. Marique proved that the same contractions were obtainable on electrical irritation of the respective centres after, as before, isolation, showing that they still retained their excitability and connection with the pyramidal tracts. These experiments indicate, therefore, that the motor centres of the cortex are not independent centres of action, but act only in response to the stimuli which proceed from the sensory centres by way of the associating fibres which connect them together.

If the true motor centres were situated only in the spinal cord, one would expect to find the spinal motor centres developed in correspondence with the motor capacities of the animal. In such case the spinal motor centres of man, in whom the motor capabilities are most varied and most perfect, should be developed far beyond those of other animals, but precisely the opposite holds. For relatively as compared with the brain, and relatively as compared with the size of the animal, the spinal motor centres of man are less developed than those of the lower animals; and they are absolutely less than those of many animals whose capabilities are of the simplest order. The development of the spinal motor centres corresponds with that of the purely reflex synergic muscular combinations of the related metameres or body segments; while that of the cortical motor centres corresponds with the multiplicity and complexity of the motor faculties, volitional and cognitive.

From the various considerations above advanced I conclude that the motor centres of the cortex are not the centres of tactile or general sensibility, nor are they the centres of the muscular sense, whether we regard this to depend on centripetal impressions, conscious or unconscious, or on a sense of innervation, but that they are motor in precisely the same sense as other motor centres; and, though functionally and organically connected, are anatomically differentiated from the centres of sensation general, as well as special.

Frontal Centres.

The region of the brain which lies in advance of the Rolandic area and marked off by the precentral sulcus is one respecting the functions of which there is still considerable doubt. Anatomically it is related to the motor tracts of the internal capsule. These tracts, according to the investigations of Flechsig, lie in the

[44] *Archiv d. Heilkunde*, 1874, Bd. xv, p. 44.
[45] The Muscular Sense, *Brain*, vol. x, 1889.
[46] *Diseases of Nervous System* (Sydenham Society), vol. iii, pp. 304, 445, 463.
[47] *Centres Psycho-Moteurs du Cerveau*, 1885.
[48] "Versuche über die Folgen der Durchschneidung von Associations-fasern am Hundehirn, *Archiv f. d. ges. Phys.*, Bd. xliv, 1889.

inner portion of the foot of the crus, and connect the frontal lobe with the opposite cerebellar hemisphere indirectly through the grey matter of the pons. Destructive lesions of the frontal centres, both of the postfrontal and prefrontal regions, as I have shown experimentally, cause descending degeneration of these tracts[49] not capable of being followed beyond the upper part of the pons. The direction of the degeneration may be taken as a proof of the motor signification of the regions in question. Similar degenerations have been described by Brissaud[50] as the result of lesions of the frontal lobe in man. He has not been able to trace the degeneration into the pyramid, and concludes that the internal tracts of the foot of the crus connect the frontal regions with the motor nuclei of the medulla. Degenerations in this part of the crus have, according to his observations, been always associated with psychical defect, apart from paralysis of the face or limbs. The effects of electrical irritation, combined with those of destruction, more particularly of the postfrontal region, indicate that this part is related to the lateral movements of the head and eyes. Irritation, as we have seen, causes opening of the eyes, dilatation of the pupils, and conjugate deviation of the head and eyes to the opposite side. At the moment of destruction of this region in the one hemisphere there always occurs a temporary deviation of the head and eyes to the side of lesion. This, however, is only transient even when the destruction has been almost if not absolutely complete. In two experiments which I have described[51], after bilateral destruction of the postfrontal area the animals were unable to turn the head or eyes to either side for a day after the operation. At first they were unable to look round when sounds were made in proximity to the ear; or, if they did, they moved the trunk and head *en masse*. The removal of the prefrontal regions alone caused no discoverable physiological symptoms, either sensory or motor. But I found in several instances that after the symptoms which followed destruction of the postfrontal area had entirely disappeared, the subsequent destruction of the prefrontal area induced paralysis of the head and eyes of exactly the same nature as before. I have confirmed these observations in a recent experiment. After apparently the most thorough cauterisation of the whole of the excitable frontal area, convex as well as mesial aspect, the animal, which at first exhibited marked distortion of the head and eyes to the side of lesion and inability to turn them to the opposite side, recovered within three days to such an extent that the defects were no longer perceptible.

A month later, extirpation of the prefrontal region, in advance of the former lesion, induced the same condition as before, namely, deviation of the head and eyes to the side of lesion, and total inability to turn them to the opposite side. The conjugate deviation of the eyes continued for some time after the movements of the head had been recovered, but within three days no defect was any longer capable of being made out in this respect. These facts indicate that the prefrontal regions have the same functional relations as the postfrontal. The transitory duration of the symptoms would be explained by the fact that the postfrontal centres were not entirely destroyed. It is difficult to remove the whole frontal area without inflicting injury on the head of the corpus striatum.

In one case in which I removed the frontal lobe on both sides by a transverse incision immediately anterior to the precentral sulcus, the animal lived only twenty-four hours. There was no paralysis of the facial muscles or limbs, though the right limbs were used with somewhat less energy than the left. Though the animal could extend its head and trunk, it was unable to maintain an upright position, or to move its head and eyes laterally. The eyes were kept shut except when it was in any way disturbed. Sight, hearing, and tactile sensibility were unimpaired. Except the inability to move the head and eyes there was no defect observable, sensory or motor. In this case the corpora striata were also injured, more on the left side than the right.

I have recently extirpated practically the whole of the frontal region of the left hemisphere (see Fig. 35). When the animal began to move about, a few hours after the operation, it was observed to turn round from right to left, and the head, when at rest, tended towards the left side. The right eyelid drooped considerably, and the right pupil was distinctly smaller than the left. Next day the conjugate deviation of the eyes continued, and they could not be turned to the right, but the lateral distortion of the head was not so pronounced. The inclination of the head towards the left gradually diminished, but the inability to turn the eyes to the right continued during the whole time of the animal's survival. It died suddenly from cerebral hæmorrhage ten days after the operation. In this case the conjugate deviation of the eyes continued longer than I had observed it in any former experiment, and this was no doubt in relation with the almost if not complete removal of the frontal lobe.

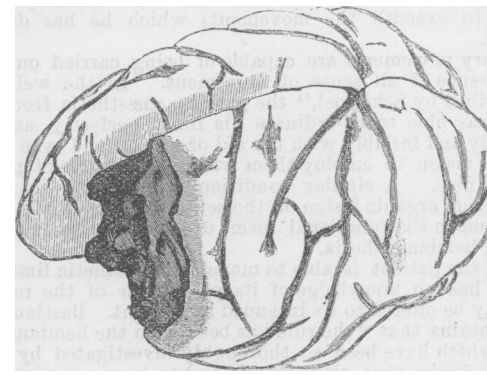

Fig. 35.

This experiment shows that destruction of the frontal region causes not only conjugate deviation of the head and eyes, but also a temporary paralysis of those movements which are also excited by electrical stimulation, namely, elevation of the eyelids and dilatation of the pupils. This confirms a similar observation which I had previously recorded. There is reason for believing, therefore, that the lateral movements of the head and eyes are not capable of being permanently paralysed, unless every portion of the frontal region be completely destroyed.

Beyond these, I have not been able to discover any other physiological symptoms on removal of the frontal lobe. I have never observed any affection of vision. Hitzig,[52] however, states that this occurs on destruction of the prefrontal region in dogs. I cannot corroborate this from my experiments on monkeys. What looks somewhat like impairment of vision towards the opposite side after unilateral extirpation of the frontal region is due to the conjugate deviation of the eyes to the opposite side, so that the animal, being unable to turn its eyes to the opposite side, does not see an object until it crosses the middle line; but the field of vision is otherwise normal. Munk finds that destruction of the frontal area in dogs causes paralysis of the trunk muscles, and he terms the frontal region the sensory sphere of the trunk, though he distinctly states that he has been unable to discover any evidence of anæsthesia. My own experiments, as well as those of Horsley and Schäfer, Hitzig, Kriworotow, and Goltz, are opposed to the statements of Munk in this respect; and Horsley and Schäfer have shown that the centres for the trunk muscles are in the marginal convolution. It is probable, therefore, that any affection of the movements of the trunk which Munk may have observed has been due to direct or indirect implication of these centres. In addition to the paralysis of the movements of the head and eyes on destruction of the frontal lobes, I have also observed, and my observations have been confirmed by Hitzig and Goltz, a noteworthy psychical defect—a defect which I have endeavoured to correlate with the inability to look at, or direct the gaze towards, objects which do not spontaneously fall within the field of vision. It is a form of mental degradation which appears to me to depend on the loss of the faculty of attention, and my hypothesis is that the power of attention is intimately related to the volitional movements of the head and eyes. On this point, however, which I have elsewhere discussed, I will not on this occasion dilate further. The recorded cases of injury and disease of the frontal lobes in man are in accordance with the negative character of experimental lesions, unilateral or bilateral, so far as relates to the sensory and motor faculties in general; and in several a certain intellectual deficiency and instability of character have been observed, not unlike those occurring in monkeys and in dogs. Of fifty-seven cases of lesion of the frontal region, collected from various sources, in two there was conjugate deviation of the head and eyes; twelve in which intelligence

[49] See Fig. 122, *Functions of the Brain.*
[50] *Contraction Permanente des Hemiplegiques*, 1880.
[51] Experiments 19 and 20, *Phil. Trans.*, Part ii., 1884.
[52] *Archiv für Psychiatrie*, 1887, Vol. 15, p. 270.

was specially impaired; and in all a total absence of paralysis of the limbs.

Though I have occupied so much of your time, I have only been able to treat of—and that in many respects very imperfectly—the functions of the cortical centres so far as concerns sensation and motion. There is another question which I have not considered at all, namely, the relations of the cerebral hemispheres to the functions of organic life. This, however, is a subject which is still involved in so much obscurity, and in reference to which there are at present so few facts which are not susceptible of different modes of interpretation, that I think it well to wait for further light before hazarding any definite views of my own. And I feel it all the less necessary to do so, seeing that the subject in one of its principal aspects, namely, the relation of the hemispheres to the thermic functions of the body, has been recently so ably placed before you by my predecessor, Dr. MacAlister.

On the psychical aspects of cerebral localisation I have touched only incidentally. This of itself would require a volume, and that mainly of speculation. As to the questions which I have treated more fully, and on which so many differences of opinion at present exist, and will probably still continue, I shall be content if the facts and considerations which I have brought before you contribute to their solution, if only by stimulating work on the part of others, with a view to arriving at conclusions which shall be acceptable alike to physiologists and physicians. For the true conception of the functions and relations of the cerebral hemispheres and their constituent centres is not only of the highest scientific and philosophical interest, but of direct and important practical bearing on the diagnosis and treatment of cerebral disease.

THREE LECTURES
ON
SOME POINTS RELATING TO INJURIES TO THE HEAD.

Delivered at the Royal College of Surgeons of England.

By WILLIAM HENRY BATTLE, F.R.C.S.,

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LECTURE II.

MR. PRESIDENT AND GENTLEMEN,—I would now direct your attention to other evidences of fracture of the base of the skull, the escape of blood from the seat of fracture, and its appearance under the conjunctiva or over the mastoid process. An effusion of blood into the cellular tissue of the orbit is one sign of the presence of a fracture of the anterior fossa of the base of the skull. This effusion of blood may be limited to the eyelids at first, and then spread to the ocular conjunctiva; it may take place into the back of the orbit, and then spread forwards, only showing itself in the conjunctiva after the lapse of some hours or days, and then spread to the lids; it may be small in quantity, or sufficient to produce exophthalmos. The value of orbital extravasation as a sign of fracture is largely dependent on the question of its position (subconjunctival or not), and the quantity in which it presents. Other signs, such as epistaxis from the nostril of the one side on which the fracture is, and the position of the injured part must be considered. Most are familiar with the appearance of the eye when there is this hæmorrhage under the ocular conjunctiva; in extreme cases it forms an elevated brownish-red circle, which encloses the cornea, and contrasts strongly with the colour of the iris of the patient. Usually disappearing in the course of a fortnight or so, it may last a long time, the blood not being absorbed for weeks; I have watched its gradual disappearance over a period of from six to seven weeks. In four cases, all of which recovered, there was much protrusion of the eye on admission, and that to an extent sufficient to interfere with the action of the ocular muscles. In one case, that of a man of 25, there was slight proptosis on admission; next day there was more marked but still moderate protrusion of the eyeball, which was displaced downwards and towards the middle line, the movements being limited in every direction, chiefly outwards. There was no ophthalmoscopic change. On the third day there was considerable subconjunctival ecchymosis. In sixteen patients there was orbital effusion in the lids when they came in, which subsequently spread to the conjunctiva. In four subconjunctival ecchymosis appeared on the second day, in three on the third, and in one on the sixth. Of the fatal cases, ecchymosis of the conjunctiva was not an invariable symptom, for in two out of fourteen there was none, the fracture being correctly diagnosed by the severe epistaxis, and in one, where the lesser wing of the sphenoid was splintered, the hæmorrhage did not show in the conjunctiva for at least ten hours after the injury. Again, in the case in which the anterior clinoid process was the only part of the base broken, contusion of the lids and extravasation in the conjunctiva, were evident from the first.

In fractures of the malar and superior maxillary bones blood may be extravasated into the lids and under the conjunctiva; this is a rare occurrence which might lead to error in diagnosis. Mr. Holmes[1] has recorded a case. Another sign of fracture of the base of the skull is the appearance of extravasated blood in the mastoid region after an injury to the head. I must ask you to consider this sign somewhat fully, for I consider it under certain circumstances to be a most important indication that the posterior fossa of the skull is the seat of the fracture. Sir Prescott Hewett writes: "Extravasation of blood, and consequent discoloration of the skin, appearing in the mastoid region some hours after a severe injury to the head, may lead to the suspicion of a fracture involving the posterior part of the base; and all the more valuable will this sign become if the injury did not bear directly upon this region, and especially if it bore upon the opposite side of the head."[2] Observations on this subject lead me to the following conclusions with regard to "mastoid ecchymosis." That it appears in the first place in front of the apex of the mastoid process. That it often spreads upwards over the mastoid in a line, slightly curved, and with the convexity backwards, its direction being approximately that of the outline of the external ear, from which it is distant half to three-quarters of an inch. At the end of three or four days after its appearance it diffuses itself forwards and backwards, chiefly in the latter direction, is most marked in the original line, and then gradually disappears. That it usually shows from the third to the fourth day after the injury, but its appearance may be delayed until the twelfth or fourteenth day. If the injury which is in the occipital region is to the right of the middle line, the ecchymosis will appear on that side over the anterior part of the apex of the mastoid process. That it may be accompanied with œdema and tenderness over the process. That the duration of the time which elapses between the receipt of the injury and the appearance of the extravasation is to some extent an indication of the distance of the fracture from the process, the blood being compelled to travel by a certain anatomical route in order to reach the surface. That unless search be made for the extravasation it is very apt to be overlooked, as the ear conceals it, especially is the ear is large and the head of patient has not been shaved. Should the fracture have taken place in the middle line of the occipital bone, or should there be two or more lines of fracture diverging towards the foramen magnum from a point in the middle line the extravasation may appear on both sides and at a varying interval.

The following examples illustrate these statements. A man, aged 41, fell and received a scalp wound over the posterior part of the left parietal bone. He was admitted into hospital unconscious, bleeding freely from the ears and nose. On the fourth day there was ecchymosis behind the left ear, from which flowed clear watery fluid. A woman, aged 50, knocked down by a hansom cab, had considerable hæmorrhage from the right ear, which ceased during the first night. On the fourth day there was well-marked ecchymosis over the right mastoid process. A man, aged 24, thrown from the driving seat of a van, was admitted into the wards unconscious, with very free hæmorrhage from the right ear. On the third day there was an extravasation of blood over the mastoid continuous in a less degree down the side of the neck. Over this area there was much tenderness. This extravasation disappeared in sixteen days, but the tenderness remained for some time longer. A man, aged 40, was knocked down, and struck the back of his head on the pavement. On the sixth day there was considerable discoloration over the occipital bone and behind the ear, and he threw his arms about when touched in that situation. A man, aged 32, was admitted on

[1] JOURNAL, 1885, p. 967.
Holmes, *A System of Surgery*, edited by Hulke, vol. i, p. 591.