Art. VII.—NOTES ON THE LOCALISATION OF DISEASES OF THE BRAIN.*

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Through the observation of patients, conjoined with autopsies, physicians can do something towards solving the problem of the cerebral functions, and my object this evening is simply to introduce the subject of the localisation of diseases of the brain, by the presentation of some notes upon cases, with a few remarks on their import and bearing. For the sake of brevity, unessential details have been left out in reporting these cases, but nothing has been omitted that would affect their interpretation.

My first case was a married woman, aged 41 years. She came under observation four months before her death. Fourteen months before this time she had suddenly become paralysed on the right side. Several years previous to this attack she had had rheumatism. An aortic regurgitant murmur was discovered. It was ascertained that, when first stricken with paralysis, she had some difficulty in deglutition, with positive facial paralysis and aphasia.

On examination, she was found to have slight right facial paralysis, the lower part of the face only being affected. The forehead and eye were not involved. She was decidedly aphasic, but could pronounce a few simple words, and seemed to understand what was said to her. She had well-marked paralysis of the right upper extremity. The shoulder was stiff; the forearm was semiflexed on the arm, and the thumb and fingers were also bent inwards on the palm; but these parts could be straightened by force, only, however, to return again to their unnatural position. The entire limb was a little wasted, and she frequently complained of pain in it. Her right lower extremity showed some loss of motor power, but not the distinct paralysis observable in the upper limb. It was paretic rather than paralysed, and exhibited no contractures. No loss of sensation could be made out. Farado-contractility was good on the paralysed side.

This patient had phthisis, and also, as revealed by autopsy, an inter-thoracic growth, probably carcinoma, and a tumour of the

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liver. She died exhausted, suffering much the week before her death, from dyspnœa and pain in her chest and right side.

The autopsy was held eighteen hours after death. No disease of the skull or membranes was detected. An area of yellowish-white softening was found, involving a small portion of the hinder part of the third left frontal convolution, the lower end of the ascending frontal, the entire surface of the island of Reil, and a narrow segment of the adjoining temporal convolution. The substance of the left hemisphere was paler than usual. The left corpus striatum and optic thalamus were normal. No other cerebral lesion was discovered. Slight vegetations were present on one of the crescents of the aortic valve, besides the other lesions to which I have alluded.

The second case was a man, 40 years old, who had had two attacks of paralysis of the left side, from which he had in both instances, in a few weeks, almost completely recovered. The arm and face had been more affected than the leg. Examination revealed a paretic condition of the lower part of the face, on the left side; the mouth was drawn very slightly to the right; the eyes and upper face were unaffected; he had no aphasia. The left upper limb was weak, but he could elevate it to a horizontal line, and perform all movements with it, but not vigorously. Neither arm, forearm, nor hand exhibited paralysis or contractures in distant groups of muscles. The left leg was a little weaker than the right, but that was all. No loss of sensation or interference with the special senses was present. The right side of the body was normal. While under my care he had two local spasmodic seizures, each lasting not over a minute, one involving the left arm and the same side of the face, the other only the lower part of the face. No change in his paralytic symptoms, and no subsequent stroke, occurred, but he died, a week after coming under observation, of what was supposed to be uræmic poisoning.

The post-mortem examination was made nineteen hours after death. The skull and dura mater presented nothing abnormal. The pia mater was œdematous and moderately congested, particularly over the right hemisphere. Anterior to the fissure of Rolando, on the right side, was an arc of distinct softening. It began above, about an inch from the longitudinal fissure, at the border of the fissure of Rolando, extending forwards and outwards so as to involve slightly the posterior extremities of the first and second frontal convolutions, and then bending inwards and backwards again towards Rolando's fissure, the edge of which it reached once more near its inferior termination. The area of softening was irregularly crescentic in shape, and varied in width from one-fourth to three-fourths of

an inch. The inner edge of the crescent and its ends, which were enlarged, were situated in the ascending frontal convolution. The portion of this convolution between the softened space and the fissure of Rolando remained unchanged. The diseased mass had invaded deeply the grey matter, and at each of its extremities had encroached upon the white substance. A small cylinder of the softened tissue reached to the median surface of the brain, about half an inch beneath the convexity. The arc of softening was the only discoverable lesion of the brain.

The lungs were œdematous. A cheesy focus was found at the base of the right lung. Both kidneys were highly granular.

My third case, reported in full elsewhere (*Medical Bulletin*, vol. i. No. 1, p. 13, January 1879), was a man, 66 years old, who for at least eighteen months before his death had had hemiplegia of the right side, with aphasia, the paralysis being most decided in the arm. He had marked loss of sensibility in the right arm, forearm, and hand, and the same condition, but less pronounced, in the right lower extremity. Fourteen days before he died he had two severe attacks of right unilateral convulsions, and a week later he had a similar seizure. His hearing was defective, and he was irritable and emotional.

Post-mortem examination showed destruction, by softening, of the following parts: a small outer rim of the island of Reil; a posterior segment of the third frontal convolution; the lower thirds of the ascending frontal and ascending parietal convolutions; the upper border of the first temporal convolution; the Sylvian border of the lower parietal, and the posterior portion of the upper parietal convolution.

Several examples of what I have supposed to be facial monoplegia have fallen under my observation, but I have not yet had the opportunity of confirming my supposition by an autopsy. In the *Philadelphia Medical Times* for October 26 and November 9 and 23, 1878, is a series of "Lectures on a Case of Facial Monoplegia," by John Guitéras, M.D., Physician to the Philadelphia Hospital. I had the pleasure of seeing the specimen from the case, which is ably detailed and discussed in these lectures. The lesion which probably caused the partial facial paralysis present was a distinctly defined area of softening, which involved one-inch of the length of the ascending frontal convolution.

The cases here reported may be looked upon as additional evidence that destructive lesions of certain districts of the cerebral cortex cause paralytic symptoms more or less extensive and permanent. They also indicate, from the local spasms occurring in the second case, and the unilateral convulsions in the third,

that a destructive lesion of the cortex may at the same time be irritative, or that it may become so temporarily. In the first case, the aphasia, and facial and brachial paralysis, were due to a lesion of portions of the areas usually regarded as the centres for speech and for face and arm movements. The ordinarily given leg-centres, high up in the ascending frontal and ascending parietal convolutions, were not involved, although the right leg exhibited some loss of power. The transient character of the dissociated hemiplegia, which occurred twice in the second case, is of interest. The cutting off of blood-supply from the lodging of an embolus, in a case of this kind, may, in the first instance, include a larger area than subsequently undergoes softening. The effects of a sudden lesion also probably radiate for a time, for a certain distance, into neighbouring parts. It will be recalled that the paralysis produced in animals by destruction of cortical areas was commonly transient. The arc of softening found in this second case was so situated as to involve only small portions of the general districts or centres for leg, arm, and face. The third case illustrated aphasia and tolerably complete hemiplegia from an extensive destruction of the cortical motor zone. The paralytic symptoms present in this patient resemble somewhat closely those produced by lesions of the basal ganglia. The unilateral convulsions were also similar to those which sometimes result from disease of the corpus striatum. The defective hearing, without disease of the ears, and the marked loss of sensibility, on the paralysed side, especially in the arm, are interesting, from the fact that some physiologists have located sensory centres in both the inferior parietal lobule and first temporal convolution.

I have notes of three unreported cases of hemorrhage into the optic thalamus, in all of which incomplete hemiplegia with hemianæsthesia had been produced. In each case the hemorrhage was large. The anæsthesia in two was pronounced; in one it was slight, and better made out in the arm than elsewhere. No spasmodic symptoms were observed. In one case the crus cerebri adjoining was involved in the hemorrhage, this patient being markedly hemianæsthetic.

In two cases of hemorrhage into the corpus striatum, well marked motor paralysis of the usual type had been exhibited. Anæsthesia was not present, or, at least, could not be recognised. The lower fibres of the facial nerve were partially paralysed; and the paralysis of the arm and leg seemed to me more decided than in the cases of hemorrhage into the thalamus opticus. In one case the clot was confined to the nucleus caudatus or intra-ventricular part of the corpus striatum; in the other, portions of both nucleus caudatus and nucleus lenticularis were

included in the lesion. I give these cases simply because they are additions to actual experience, avoiding lengthy details, as the symptoms observed did not differ from those frequently reported. Recently, however, I presented to the Pathological Society of Philadelphia specimens from two cases of peculiar interest. In one of these the lesion was triple, consisting of a large clot in the right optic thalamus, a small cyst in the right corpus striatum, and a large cyst in the left corpus striatum, the symptoms being left hemiplegia and hemianæsthesia, without right hemiplegia. In the second case a small apoplectic cyst was present in the right corpus striatum, the patient not having been hemiplegic.

In still another case, never before reported, I found softening, involving the entire right island of Reil, a portion of the second and third frontal and of the third and fourth temporal convolutions, where they bound the Sylvian fissure, and two-thirds of the corpus striatum within the lateral ventricle. The symptoms observed during life were mental hebetude, slowness of speech (but not aphasia), dulness of hearing, slightly impaired sensibility on the left side, and general muscular weakness. He was not hemiplegic, as we clinically understand the term hemiplegia. The muscular weakness was a little more evident on the left than on the right side, but he used both arms and both legs with almost equal facility, and no contractures were present.

From a study of these examples of lesion of the great basal ganglia, it will be seen that, while partial destruction of the corpus striatum usually causes typical hemiplegia, such is not always the result. In some instances little or no paralysis occurs. Hemorrhage may occur into the optic thalamus also, without motor paralysis, although in all of my cases more or less complete hemiplegia was present. According to Nothnagel, indeed, lesions of which the thalamus opticus is the exclusive seat are not followed by motor paralysis at all. He also says that it may be regarded as demonstrated that lesions in the interior of the thalamus opticus cause no disturbance of sensibility. ("Ziemssen's Cyclopædia," vol. xii. pp. 148 and 149.)

Both the corpus striatum and optic thalamus are connected by fibres with the convolutions above, and below with the mesencephalon. In addition, a bundle of white fibres, called the internal capsule, is supposed to pass, compressed into a small compass, between the outside of the optic thalamus and the nucleus lenticularis, or portion of the corpus striatum which lies beyond the ventricles in the substance of the hemispheres. It is probable that within this internal capsule are included both

the great sensory and motor tracts which go to and proceed from the convolutions, and it may be, as has been supposed by some, that true paralysis only occurs when the internal capsule is implicated directly or by pressure.

Time will not permit me this evening to go into any lengthy discussion of the various theories of localisation and the question of the real nature of paralysis.

The broad fact that one-half of the body is controlled by the opposite half of the brain is of itself a strong point in favour of the general doctrine of localisation. Cases without number, similar to those given in this paper, have been recorded to prove that paralysis usually appears on the side opposite to the brain lesion. Brown-Séquard's array of opposing cases is, after all, probably only sufficient to show that we may have exceptions to a great rule. This is especially likely, since recent embryological researches have shown that the decussation in the medulla oblongata is variable in character.

The tracts which go to and from the cortex also, doubtless vary somewhat in their directions, and special centres may differ according to the age and habits of the individual.

In regard to the nature of paralysis, my personal experience has not as yet been sufficient to enable me to come to an absolutely satisfactory opinion. With Bastian ("Paralysis from Brain Disease," p. 50), I incline, at present, to think that several explanations may be allowed, in accounting for paralytic phenomena. Some of the symptoms may be due to irritation, others to destruction of brain tissue, and in still other cases, injuries to the brain, besides causing direct symptoms, may produce stimulating or inhibitory effects upon more or less remote parts. I consider it probable, also, that a special form of inhibitory motor paralysis may result from a strongly irritative lesion of portions of the antero-frontal lobes. I reported to the Pathological Society of Philadelphia a case of fibroma, involving the first and second frontal convolutions, and convolution of the corpus callosum, in which the paralysis present appeared to be of the true inhibitory type (*Philadelphia Medical Times*, January 18, 1879). I believe it not unlikely that we will learn to distinguish between paralytic symptoms due to inhibitory action, and those which are the result of pressure or tissue-destruction.

In concluding these brief notes, I would say to those who may question the value of such investigations, that even direct practical results from a study of cerebral localisation have not been entirely wanting. They have been obtained chiefly in the domain of surgery. Thanks to the labours of such men as Broca, Bischoff, Turner, and others, cranio-cerebral topography is now

pretty well understood. The physician or surgeon can determine with considerable precision such points, for instance, as the relations of the fissures of Rolando and of Sylvius to cranial sutures, the superior levels of the great cerebral ganglia, and the situation with reference to external areas of such important convolutions as the third frontal and angular gyrus.

The surgeon's trephine may be guided with greater certainty than ever before to the seat of a lesion. Broca, in 1871, successfully located an abscess of the third frontal convolution of the left side, and reached the lesion by operation. Even supposing the position of an abscess of the brain to be accurately determined, it may be said that an operation might be dangerous or impossible, and I recognise the fact that operative interference would only hold out hope in a limited number of cases. In the matter of organic cerebral affections, however, a little advance is a great gain. Huguenin ("Ziemssen's Cyclopædia," vol. xii. p. 819) mentions an instructive case, in which Renz succeeded, after extracting the blade of a knife, in emptying an abscess which lay deep in the brain by successive introductions of a subcutaneous syringe. The patient was cured. He lived eight years and a half free from all brain symptoms, and died from hemorrhage of the lungs (or stomach?). It is true that in this instance an external opening was present; but it shows the possibility of emptying and healing an abscess deeply situated in cerebral tissue.

M. Proust (*Medical Times and Gazette*, December 16, 1876) communicated to the French Academy of Medicine the case of a young man who had received a bayonet wound on the left side of the head, and subsequently had partial aphasia and incomplete paralysis of the right face and arm, with other symptoms. With the aid of M. Terillon, trephining was performed. The aphasia and arm paresis instantaneously improved. Hebetude, which had been present, disappeared, and the patient eventually recovered. Trephining has been successfully employed in a similar case by another French surgeon, M. Lucas Championnière, who has given to the profession certain data for determining the "line of Rolando" (*Lancet*, July 7, 1877). Aphasia, monoplegia of the face, arm, or leg, imperfect hemiplegia, limited convulsions, strabismus, and nystagmus, are among the symptoms which can now be successfully employed by the surgeon in deciding upon cranial operations.

In medicine a more reliable prognosis can be given in intracranial affections if we can locate with accuracy the seat of disease. Regional diagnosis also is often a great aid to general diagnosis; knowing *where* a lesion is, we can frequently come to a more satisfactory conclusion as to *what* it is; and thus we

may be able sometimes to discriminate to the advantage of our patients between such conditions as clot, tumour, softening, sclerosis, and meningitis. Mental diseases are becoming better understood; some of them, for instance, being found to be due to lesions of the cortex, macroscopic or microscopic. Circumscribed cerebral meningitis is an affection which cannot always be recognised from the general picture drawn of it in ordinary text-books; but a knowledge of the varying effects produced by the disease, according to the region of the brain covered by the inflamed membrane, will often help greatly to a correct conclusion.

The substitution of one region of the brain for another whose functions have been annulled by disease, through some system of development by training, is a new path in cerebral therapeutics, which holds out some promise, and is an outcome of the study of localisation.

A study of the symptoms produced by involvement of successive districts of the cortex has done much to clear up the mists which have enveloped that interesting affection known as general paralysis of the insane.