ON SOME SYMPTOMS OF ORGANIC BRAIN DISEASE.

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I.—ON SUDDEN PARALYSIS IN CEREBRAL TUMOUR.

PARALYSIS, the result of the presence of a growth in the brain, is commonly of slow, gradual development. Such paralysis is rightly regarded as characteristic of tumour. Loss of power of sudden onset is sometimes regarded as incompatible with the diagnosis of a growth. But this is certainly incorrect. Cerebral tumour is occasionally associated with paralysis of onset as sudden as is ordinarily thought to be characteristic only of hæmorrhage or softening. In some cases this sudden paralysis is not due to the tumour—it is due to an associated vascular lesion. But in other cases such associated cause cannot be found, and the sudden paralysis appears to be the direct effect of the growth. These cases are of much pathological interest, and all are of clinical importance on account of possible errors in diagnosis. There is nothing new in the fact, but it has scarcely received the attention it deserves.

In speaking of sudden paralysis, the cases may be distinguished in which the onset of the paralysis is accompanied by a convulsion. It is very common for a convulsive attack to leave considerable weakness in the part convulsed. After an epileptic fit such weakness is transient. After a convulsion from organic disease, as tumour, it may be permanent. It is, in that case, the result of the presence of the growth

in a motor region, which means the irritative destruction of that region. The weakness resulting from the after-effects of the "discharge" may render the effect of the graver damage produced by the growth suddenly apparent. But these cases, although they may be distinguished, cannot be entirely separated from those in which the paralysis results suddenly from the direct action of the growth without convulsion.

The causes of paralysis of sudden onset in cerebral tumour may be enumerated as follows:—

Associated conditions . Haemorrhage into and around growth.

Softening from (a) associated vascular disease.

(b) Pressure on or Invasion of vessels.

Direct effects . . . Inhibition (?)

Exhaustion from convulsion.

The first-mentioned cause, hæmorrhage, with growth, is one of the rarest. The condition is common, but does not occur, as a rule, until the tumour is of considerable size. The resulting hæmorrhage does not produce paralysis unless the tumour is situated in a motor region; but the growth so situated has commonly caused paralysis from its size, before the hæmorrhage occurred.

Softening, from direct invasion, and consequent obstruction, of vessel, artery or vein, is also rare. It is well known how constantly vessels escape the effects of pressure or invasion by such growths as occur within the skull.

Softening from associated and independent vascular disease is, however, a common incident in cerebral tumour. It may occur in the old, in whom chronic endarteritic changes occasionally co-exist with a growth. It occurs, however, more frequently in the young, in cases of syphilitic disease. It is exceedingly common for syphilitic disease of vessels and a syphilitic growth independently of vessels to co-exist, and each may produce its own symptoms; if the tumour is not in a motor region, there may be only headache and optic neuritis to declare it, and the only paralytic symptoms may be due to the vascular disease, and may be of instantaneous onset. The following case is an instance of the co-existence of these two conditions, and in it the sudden paralysis must, I think

be ascribed to the vascular disease. The case is also of interest from the long interval between the primary syphilis and its latest manifestation.

T. N., a strongly-built artisan, aged 35, was first seen at the National Hospital for the Paralysed and Epileptic, on May 30. He was suffering from slight right hemiplegia, and gave the following history. Fifteen years before, he had well-marked constitutional syphilis. Four years before, after having pain in the back of his head for some weeks, he had what his wife termed a "fit," in which there was loss of consciousness for a few minutes, but no convulsion. It was followed by slight right-sided weakness, which lasted for some months, and then gradually passed away. The headache, however, continued, and he had occasional attacks of giddiness, in which he walked unsteadily. A year after the first, a second similar attack of loss of consciousness left him slightly weak on the right side, as did the other, but was succeeded by total, or almost total, loss of sight. He could see perfectly until the occurrence of the loss of consciousness. After four months' blindness, the sight gradually returned, but not completely. The giddiness became less marked, but the occipital headache continued until a year ago. He was pretty well until a fortnight before he came under observation, and then two convulsive attacks occurred, apparently general. After the last, control was lost over the bladder and rectum.

When first seen he seemed to understand slowly what was said to him. The expression of the face was dull and heavy. He did not speak, except when asked questions, and then sometimes remained silent and looked puzzled, at others answered them intelligibly, speaking slowly. No paralysis could be detected, either in the limbs or of any cranial nerve; but the stools and urine were passed involuntarily. Ophthalmoscopic examination showed the right optic disc to be well coloured, but clear, outline distinct; around it, however, were several white patches with soft-looking edges, over which the vessels of the retina passed without change of position or calibre. The vessels were normal. Examination of the left eye was impracticable, on account of an old injury.

Under the influence of perchloride of mercury he improved

rapidly. In a few weeks he regained control over his sphincters, and spoke in his ordinary manner. On July 1, however, he complained of general malaise, but had no headache or giddiness. Next day his wife observed his manner to be strange. On July 3, he was drowsy, sleeping most of the day; his manner was dull and listless. He spoke as usual when asked questions, but did not care to speak unless spoken to. On the following morning, July 4, his wife, on waking, found him unable to speak or to move his right arm or leg. She had noticed nothing like a convulsion in the night, and thought that she must have done so had there been one. He could not open his mouth farther than would admit the tip of a spoon, but seemed to swallow liquids without much difficulty. Next day he seemed to notice what was going on, and after a few days to understand what was said to him, but he did not speak, except that when pressed by his wife to say if he was better, he said "Better."

He was seen on July the 10th; he seemed to understand whatever was said to him, and attempted to execute any movement he was told. He could not be made to say "Yes" or "No" voluntarily. Once he uttered correctly two or three words in answer to a question. No other speech could be obtained. There was very slight paralysis of the lower part of the face on the right side, noticeable at rest and on slight movement, but disappearing on stronger movement. He could not be made to protrude his tongue, nor to contract his masseters. No paralysis of any other cranial nerve could be detected. He could not move his right arm or leg. By a strong effort to move the leg he could just contract the muscles of the right thigh. The limbs were not rigid. There was distinct hyperæsthesia of the paralysed arm and leg. Ophthalmoscopic appearances were unchanged.

In this state he continued for three days; and then, on July 14, violent convulsions came on, and recurred so frequently that his wife described them as "one continuous fit." Both sides, according to her account, were equally involved in the convulsion, both arms being very rigid. The attacks continued until his death, four days later (July 18).

At the post-mortem examination a small growth was found

in each hemisphere. That in the right hemisphere was about the size of a cherry, and situated in the lowest portion of the parietal lobe, in the supramarginal gyrus, above the posterior limb of the fissure of Sylvius. It reached but did not project from the surface of the convolution. On section it was firm, and the cut surface was uniform, greyish, and semi-translucent.

In the left hemisphere a tumour the size of a walnut existed in the inner part of the frontal lobe, about two inches from the anterior extremity of the hemisphere. It involved the grey matter of the marginal convolution, but was elsewhere situated only in the white substance of the hemisphere, which was much softened, and mottled with vessels in the vicinity of the tumour. Its surface was greyish red and translucent. On section it was very firm, having nearly the consistence of soft cartilage. The greyish-red superficial layer enclosed a central opaque greenish-yellow mass. This "cheesy" centre was irregular in shape, irregularly lobulated, the portions being separated by reddish-grey translucent tissue, continuous with that of the periphery, but of a deeper red.

On slicing the hemisphere it was seen that while the posterior third above the ventricles was firm, and presented more visible vessels than normal, the anterior two-thirds were much softer than natural, and contained very few visible vessels. The softer portion was reddish in the neighbourhood of the tumour, white in the middle of the softened portion, and yellowish behind. The walls of the lateral ventricles were quite firm. The corpus striatum and optic thalamus were perfectly healthy. The cerebellum was free from any abnormal appearance.

The vessels at the base of the brain were diseased at several points. On the left vertebral, on the side of the medulla oblongata, was a semi-opaque swelling, about half an inch in length, and in thickness about equal to the diameter of the artery. On section the thickening had only slightly diminished the calibre of the vessel. The right vertebral was larger than the left, and tortuous. The branch of the posterior inferior cerebellar artery, which runs along the anterior edge of the cerebellum, also presented a large nodule, the size of half a pea, semi-opaque, and similar in appearance to the other, and,

like it, not obliterating the artery. Another nodule of similar appearance lay in the middle of the left Sylvian fissure, being attached to the most anterior of the two arteries, into which the middle cerebral divides, and this part of the vessel was closed. Branches of the other unaffected artery, given off further on, could be seen passing onwards and forwards over part of the nodule to supply the contiguous brain substance, which presented no trace of softening.

I think that the anæmia and softening of the anterior part of the left hemisphere must be regarded as the cause of the sudden paralysis and loss of speech, and that this anæmia must be ascribed to the co-existing disease of the middle cerebral artery. No symptoms can be ascribed with certainty to the growth in the right supramarginal gyrus. The sight of the opposite eye had been long lost from injury.

There remain for consideration the still more interesting cases in which a cerebral tumour causes a sudden paralysis without there being any discoverable cause for the suddenness of the onset. Of this the following cases are examples :—

A woman, aged 53, had been suffering for about six months from headache and rheumatic pains in the chest. Twelve days before, she suddenly fell while walking, but without, according to her own account, any loss of consciousness or any giddiness. For the next seven days there was more headache and irritability, but no weakness. Then, five days before admission, she lost consciousness for three hours, and on recovering consciousness was unable to move the left arm or leg, without, however, any affection of speech. Gradually her mental state became more and more dull. When admitted into the National Hospital for the Paralysed and Epileptic, there was complete left hemiplegia and great mental dulness. She lay on her back taking no notice, making no attempt to change her position, moaning frequently. When spoken to she would not at first answer, but on the question being repeated, especially if simple (as the names of her children), she answered, commonly correctly. After one reply, it was difficult to extract another for some time. She lay in bed with head and eyes inclined a little to the right (towards a window), but they

could be turned to the left freely. There was no other affection of ocular movements. The left side of the face was distinctly weak in the lower part. There was no apparent deafness. Deglutition was unimpaired. The tongue on protrusion deviated slightly to the left.

The left arm was absolutely paralysed and slightly rigid; the rigidity increased on attempts at passive motion. The left leg was also motionless. Sensation on left side was thought to be unimpaired. The right limbs were moved freely.

A few days after admission, the right arm and leg were moved less, and gradually became motionless. Soon after the right side became powerless, the mental state became more and more dull, and she passed into a state of insensibility, lying with eyes closed and mouth open, but occasionally half opening her eyes for a short time. Nothing roused her; but if the left arm was moved, an expression of pain passed over the face. Deglutition became difficult, partly at least from her mental state, food being held in the mouth a long time before being swallowed. The comatose condition increased, and she died a month after her admission—the temperature having been normal until the last day, when it rose to 102°.¹

At the post-mortem examination, two growths were found in the brain; one, small in size, in the right corpus striatum, and a large one in the corpus callosum extending laterally into both hemispheres. The small growth measured three-quarters of an inch from before back, and five-eighths of an inch from above down. It was situated at the junction of the crus with the corpus striatum, its inner limit being just outside the optic tract, which was not, however, in any way damaged. It occupied the anterior and upper part of the radiation of the crus into the striate body. The larger growth had infiltrated the substance of the corpus callosum, so that its white substance was no longer distinguishable. Its antero-posterior extent was an inch and a quarter, the posterior extremity being two inches from the posterior extremity of the corpus callosum; the anterior margin of the growth was just behind, and did not involve the anterior curved portion of the corpus callosum. A few white

¹ The notes of the symptoms of this case were taken by Dr. W. A. Sturge.

longitudinal fibres were visible on the under surface only. On the upper surface it formed a grey prominence. In the left hemisphere it had infiltrated the white substance above the lateral ventricle, extending outwards as far as the junction of the latter with the corpus striatum, and almost to the grey substance at the bottom of the transverse frontal fissure; backwards as far as the level of the anterior extremity of the optic thalamus, and forwards as far as the level of the anterior extremity of the corpus striatum, infiltrating through this extent the white substance of the gyrus fornicatus, and to a less extent that of the marginal convolution; its anterior and vertical measurements being one inch and a quarter. In the right hemisphere its extent was smaller, its position being similar, but its antero-posterior extent only one inch, and its upper limit the calloso-marginal fissure.

The growths were uniformly soft, pinkish-grey, mottled with red dots and lines; nowhere opaque; uniform. Demarcation tolerably sharp; a little white softening outside the growth in the substance of the hemisphere. No hæmorrhage. The microscopical characters were, in each tumour, those of a glioma.

In this case we must associate the left hemiplegia with the growth in the right striate body, and the subsequent right hemiplegia, probably, with the extension into the left hemisphere of the tumour in the corpus callosum. There was nothing to account for the suddenness of the onset of the paralysis.

Another case, which I recorded a few years ago,¹ presents an equally striking case of sudden paralysis from tumour, and may be briefly recapitulated:—A man, while in fair health, lost consciousness for a short time, and afterwards slight persistent weakness of the right side was observed. Subsequently he had one or two other attacks of transient unconsciousness. Two months later he was one day found unconscious, and on recovering, in an hour or two, partial consciousness, the right arm and leg were found to be completely paralysed; he was speechless, and the action of the sphincters was involuntary. In a day or two he recovered slight power over the leg,

¹ *Path. Trans.*, vol. xxvii. p. 18.

and none over the arm. He became able to say a few words, stopping frequently from confusion, and being unable to get at the word he wanted to say. He could not name his occupation, but when it was mentioned he recognised it at once, and could then repeat the name. When he tried to speak quickly he became confused, mumbled, and could not be understood. This state continued for a fortnight. He then had a series of convulsions, epileptiform in the right side, with simultaneous co-ordinated movements in the left which struck the observers as undoubtedly "convulsive." He afterwards had fits confined to the right side, and the left limbs were motionless and apparently paralysed. Optic neuritis existed in the eyes. In this state he died. A tumour (glioma) was found in the left hemisphere, above the lateral ventricle, occupying the greater part of the superior parietal lobule, *i.e.* the parietal lobe above the intra-parietal sulcus. In the lower part of the lobule it projected above the adjacent convolutions in an area of about an inch. It extended downwards to within a quarter of an inch of the roof of the lateral ventricle, and inwards to within half an inch of the inner surface of the hemisphere. The upper extremity of the ascending parietal convolution was not involved, but was pressed upon, and narrowed by, the growth.

In this case also there was nothing to account for the sudden onset of the paralysis. In neither case was there, apparently, any initial convulsion. It is not conceivable that the sudden onset can have been the result of the pressure of the growth on, or invasion by it of, the motor tract, since such paralysis would be as gradual as the process which caused it. The sudden onset may be compared with another effect of tumour equally sudden, convulsion. Convulsion depends on the discharge of a motor centre under the irritating influence of the growth, and these facts seem to suggest that the irritation may exercise an influence on the centre, inhibiting instead of discharging it, or leading to the inhibition of the lower centres. This was the explanation suggested when the specimen from the latter case was shown (in 1875) at the Pathological Society, and in his lectures, since published, Dr. Brown-Séquard has applied a similar explanation very widely. As an

explanation of the phenomenon in question, it derives, I think, additional probability from the facts of the last case I have mentioned. In this, towards the last, there was a paralysis of the limbs on the left side as well as on the right. There was nothing to account for this but the growth in the left hemisphere: there was no evidence of any pressure exerted by it on the right hemisphere. The growth was situated in the midst of the fibres from the corpus callosum, and by its action on these may have exercised an inhibitory influence on the right centres, paralysing the left limbs. In the same manner a growth in this situation may cause, as in one case recorded,¹ convulsions beginning on the same side, in just the same manner as they had previously commenced on the opposite side.

Thus the phenomena of sudden paralysis after convulsion, and sudden paralysis without convulsion in cases of cerebral tumour, cannot be entirely separated, and the two may in some cases be conjoined as the result of the local irritation.

II.—ON SLOW INCO-ORDINATION FROM DISEASE OF THE OPTIC THALAMUS.

Many observations now attest the frequency with which lesions of the optic thalamus are associated with mobile spasm, spontaneous ("athetosis"), or in movement (slow inco-ordination). Charcot found disease of the white fibres outside the thalamus (posterior part of the internal capsule), and believed that it was the implication of these fibres which determined the symptom. In one case, however, in which jerky inco-ordination had been present, I found disease of the middle of the thalamus, not involving the capsule.² The following case is another illustration of the same condition: a spasmodic slow inco-ordination was due to a lesion nearly in the same situation.

Jane M., a married woman, aged 41, was admitted under my care into the National Hospital for Paralysis, in October, 1877,

¹ *British Medical Journal*, 1874. Dr. Buzzard, *Practitioner*, October, 1868.

² 'Athetosis and Post-hemiplegic Disorders of Movement,' *Med. Chir. Trans.* 1876.

with right hemiplegia of six months' duration. Its onset had been sudden, with transient loss of consciousness (for two or three minutes only). The attack had been preceded by much left-sided headache, frontal and occipital. A little "difficulty in getting words out" succeeded the onset: no more marked affection of speech. The arm and leg were powerless for months. She had considerable hypertrophy of the heart: no history of rheumatic fever. Owing to a mistake, her urine was not tested. On admission there was slight paralysis of the lower part of the face for emotional, none for voluntary, movements. The arm was still very weak, she could just raise the hand to the head, grasp = seven kilogrammes. The hand, when held out, was unsteady, and there was considerable irregularity in the movement of the fingers, causing slow inco-ordination, the fingers "sprawling" about. There was a little slow involuntary movement of the fingers. There was much spontaneous pain in the arm and hand. No change in sensibility was noted. A fortnight later the leg was stronger: she could walk alone, and was then sent to the country branch at Finchley. She was there found one morning unconscious. I saw her in the latter part of the day, and she was then lying on her back, incapable of being roused. There was no stertor, but a little froth was coming from her mouth. The left arm, when raised, was held for a moment, and fell gradually. The right arm and leg fell at once on being raised. When the right arm was pinched, it was moved a little. The conjunctivæ were sensitive. The eyelid closed when it was touched. The pupils were rather—not extremely—small: the left a little the larger of the two. The eyes did not quite correspond in position, and on close observation the right appeared to move while the left did not. There was no conjugate deviation. She died an hour later. From the inco-ordination of movement it was suggested that possibly the initial lesion was in the optic thalamus, and from the marked difference in the movement of the two eyes, that the ultimate lesion was in the pons. It proved to be so. A large clot lay in the substance of the pons, about the middle (from above down). It had destroyed the greater part of the left half of the pons, but many extravasations existed in the right half. The lowest

fibres (transverse) were little damaged : those from the anterior pyramids were wholly destroyed on the left side. The clot extended up to the lining membrane of the floor of the fourth ventricle just above the eminentia teres. The upper extremity was about a third of an inch from the posterior border of the corpora quadrigemina, and the lower at the level of the facial nuclei.

The left optic thalamus presented a depression on the upper surface running transversely across, in front of the posterior tubercle. On section a space was found, capable of holding half a nut, filled with ochre-yellow material, and situated nearly in the middle of the thalamus, passing inwards and backwards as far as the left anterior of the corpora quadrigemina, but in such a manner as to leave the posterior tubercle of the thalamus, and its connection with the corpora geniculata damaged. Externally it did not extend beyond the limits of the thalamus, but ended in a jagged border just to the inner side of the white bundles of the internal capsule. No other lesion could be found in the brain. The arteries of the base were normal, except the basilar, which presented some atheroma.

This case is of interest as showing, just as did the other to which I have referred, that inco-ordination of movement may result from a lesion of the optic thalamus itself, not apparently implicating the white fibres outside it. No defect of sensibility was noted; my brief notes of the case mention merely the absence of hyperæsthesia. There is some reason to regard the optic thalamus as both a sensory and a reflex centre. This function is quite in harmony with the circumstance that inco-ordination may result from the perversion or loss of its function. It is interesting to find that just as inco-ordination of movement of the legs results from damage to the tract in the cord through which the sensory fibres pass, and certainly without any correspondence with the affection of sensation, so inco-ordination seems to result from damage to the sensory ganglion of the brain, and, also, without relation to the degree of impairment of sensation. Of the cases of post-hemiplegic mobile spasm published in the paper before alluded to, six out of ten presented no impairment of sensibility.