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TWO ANOMALOUS CASES OF SYRINGOMYELIA.

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THE two cases of syringomyelia which form the subject of this paper offer some points of interest from both the pathological and clinical standpoints. In neither of the cases was the disease diagnosed during life, and in both complicating conditions were found in the central nervous system on *post-mortem* examination.

It is now generally admitted that syringomyelia is not a pathological entity, but a condition which may develop either primarily, or more rarely secondary to different pathological processes involving the spinal cord. The difficulty, which has been recognized by Schlesinger and other authorities, in defining syringomyelia in terms of pathology has undoubtedly led to the inclusion of many cases under this name on entirely inadequate grounds. It seems to us, however, that the chief lesions in the cases we report conform so completely to those which characterize syringomyelia that they must be included in this disease, no matter what definition of it be adopted.

CASE I.

Syringomyelia and Syringobulbia associated with Syphilitic Pachymeningitis.

J. S., a journalist, aged 43 years, was admitted in September, 1907, to the National Hospital under the care of Dr. Ormerod, to whom we are indebted for

permission to use the clinical notes. As he was an educated man and took an intelligent interest in his disease it was possible to obtain a full and accurate history of his case.

At the age of 18 he contracted syphilis, and later had well-marked secondary symptoms, including condylomata; he neglected to follow the treatment prescribed. Three years later he developed a double-hemiplegia with optic neuritis; for a fortnight, according to his own account, he was totally paralysed, but recovered quickly under anti-syphilitic treatment. A cerebral gumma was diagnosed. After this he underwent several "cures" at Aix and other places.

Thereafter he remained fairly well till four years before his admission to hospital, though for years he had complained of unsteadiness of gait and difficulty in walking in the dark. Within the past four years his gait has become worse, his legs more stiff and rigid, and he has tired very easily. During the past year he has required two sticks to get about at all, owing to the progressive weakness of his legs and the difficulty in balancing himself. For years he has also had difficulty in starting micturition.

For two years he has had diplopia, and his sight has deteriorated. Recently he has noticed ataxia of his arms; he found in using his typewriter that "his fingers would strike the wrong letters."

The patient was under treatment by several physicians, but we are ignorant of the diagnoses which were made; apparently the disease was generally regarded as syphilitic, but by an eminent French neurologist he was told he had "*tabes combinée.*"

On examination smell, taste and hearing were found normal, but vision was reduced to $\frac{6}{18}$, and ophthalmoscopic examination revealed optic neuritis subsiding into consecutive atrophy; the swelling had disappeared, but the edges of the discs were blurred, the physiological pits filled in, and the vessels reduced in size. There was slight internal strabismus and diplopia, and all ocular movements, but especially conjugate lateral deviation, were accompanied by nystagmus. The pupils were small, but regular in outline; their reaction to light was sluggish, but they contracted well on accommodation. The facial muscles and the movements of the tongue and palate were intact, and deglutition, phonation and articulation were unaffected.

The chief symptoms of the disease were seen on examination of the motor system. The muscles of the head and neck were unaffected, and in the upper extremities the muscles were well developed; none were wasted, their tone was good, and the strength of the larger movements was excellent, but the finer movements were ataxic. No local atrophy or paresis was detected in the muscles of the trunk; the patient was able to rise from the prone to the sitting position without the aid of his arms. There was no wasting in the lower limbs, but both were very spastic, and their movements were feeble, especially dorsiflexion of the feet. His gait was very spastic and ataxic, the knees were held rigidly extended and the toes dragged along the ground. Romberg's sign was well marked.

Examination of sensation revealed a relative analgesia of considerable degree

on the ulnar aspect of both upper extremities, and on the trunk from the level of the second costal cartilages to midway between the xiphoid and the umbilicus. There was also some diminution of tactile sensibility, as tested with cotton-wool, over the same areas, but of considerably less degree than the analgesia. Thermal sensibility, as far as it was tested, was intact. Joint-sense and the appreciation of pain on deep pressure were also normal in all limbs. There was no history of shooting pains or other paræsthesiæ.

The tendon-jerks of the upper extremities were brisk; the knee-jerks and ankle-jerks were much exaggerated; ankle-clonus was easily obtained, and both plantar responses were of the extensor type.

The patient left hospital in a practically unaltered condition; some weeks later he died in an attack of acute pneumonia, and through the courtesy of his medical attendants we were able to obtain a *post-mortem* examination.

The autopsy was performed about eighteen hours after death. The brain appeared but little altered, and there was no evidence of disease on its surface. The soft membranes at the base and on the ventral surfaces of the pons and medulla were opaque and slightly thickened, and the walls of the larger arteries were thickened. Even before cutting open the spinal dura mater it was evident that there was gross disease of the spinal cord; it appeared shrunken and flattened antero-posteriorly. The membranes were also very much thickened, especially in the cervical and upper dorsal regions, and it was in many places impossible to separate the dura mater from the thickened and opaque pia-arachnoid, by which it was bound down to the surface of the cord.

The meningeal disease was greatest over the cervical and upper dorsal segments; in the lumbar and sacral regions it was inconsiderable, though even here the soft membranes were more opaque and more firmly bound down to the cord than normally.

When the cord was cut across, a cavity was seen in its centre extending from the mid-lumbar region to the highest cervical segments. It was largest in the upper dorsal region, and to the naked eye it appeared to extend into the medulla.

Microscopical Examination.

The exact condition of the spinal cord could be determined only by microscopical examination. The nervous system was first hardened in 10 per cent. formalin in normal saline solution; blocks from each segment of the cord and from the medulla oblongata were then prepared for Weigert's medullary sheath stain, and sections from each of these blocks were also stained by hæmatoxylin and van Gieson's method. Sections of other blocks from various regions were stained by Mallory's method for the demonstration of neuroglia.

The Meninges.—The thickening of the meninges becomes well marked at the level of the decussation of the pyramids, and is very intense over the cervical enlargement. It lessens slightly over the upper dorsal segments, and increases again in the middle and lower dorsal regions, but finally disappears at about the level of the third lumbar segment. Over the lower two lumbar segments and

the sacral cord there is practically no thickening of the meninges, but some small round cell infiltration persists similar to that to be described in the higher levels. The thickened meninges are densely sclerosed; where the thickening is most intense, calcareous concretions and layers of bone lie in them. In places, as in the ventral surface of section shown (fig. 2), it was necessary to cut away the bone before preparing the section.

The dura mater, as well as the pia-arachnoid, is involved in the thickening, and firmly bound to it by adhesions. The thickened pia mater is also abnormally adherent to the surface of the cord. The majority of the meningeal vessels are diseased; their walls are thickened and frequently hyaline: in other vessels there is considerable proliferation of the intima, but this change is less common. Further, around many of the vessels, as well as in the less compact portions of the thickened membranes, there are foci of small round cell infiltration, and in places among these cells typical plasma-cells. As already mentioned, the cellular infiltration extends over the lumbar cord where there is little or no thickening of the meninges; the same condition may be observed in the medulla. In many places there is marked perivascular infiltration around the intramedullary vessels of the cord and brain-stem.

Spinal Cord.—The topography of the intramedullary disease is most easily traced in Weigert preparations, but at each level its histology was also studied in sections stained by hæmotaxylin and picro-fuchsin, and in many segments in Mallory preparations as well.

In the sacral and lower lumbar segments, the only disease is secondary degeneration of the crossed pyramidal tracts. The definite syringomyelic disease appears in L_4 segment as a dense gliosis in the grey commissure and the base of the left dorsal horn, which in the next higher segment becomes much more extensive and produces a considerable increase in size of the left side of the cord. The disease is most prominent in the dorsal horn and the neighbouring part of the dorsal column; in the periphery of the dense glial tissue many Deiters's spider-cells with long branching processes can be seen, while the centre has undergone partial rarefaction, but as yet there is no sharply defined cavity. In Mallory preparations the glial tissue appears much more extensive than in sections stained by other methods; it extends across the grey commissures and into the base of the right dorsal horn.

In the L_1 and L_2 segments a large cavity occupies the grey commissure, the ventral portion of the dorsal column and practically the whole of the left dorsal horn, and an evagination from it extends into the left lateral column. Its walls are composed of dense neuroglial tissue with many spider-cells; they contain many blood-vessels, the walls of which are as a rule thickened, and some of them are hyaline. The gliosis is much more extensive than the cavity; it extends along the dorsal median septum, and across the commissure into the right dorsal horn, in the centre of which there is some rarefaction. The ventral horns are intact at this level, but there is intense degeneration in the lateral column in the position of the pyramidal tracts. In the lowest two dorsal segments the disease is similar in nature and extent.

In D_{10} the cavity is considerably larger; it occupies almost the whole of each dorsal horn, and extends between them through the grey commissure, involving also the ventral half of the dorsal column; it lies entirely dorsal to the central canal, which is represented by a solid column of ependymal cells. On the right side a projection of the cavity extends into the lateral column, and on the left a separate but smaller cavity lies in the inner portion of the lateral column, parallel to the lateral border of the dorsal horn. As a rule the walls of the cavity consist of a densely sclerosed neuroglia, but in places the tissue is less compact, and coarse glial fibrils and vessels with thickened walls project into its lumen. The ventral half of the dorsal column, and the dorsal and inner portions of each lateral column, are invaded by the sclerotic gliosis which surrounds the cavity; where the gliosis is advanced, practically no myelinated fibres can be found. About this level the thickening of the meninges becomes more prominent, and secondary thereto there appears some

FIG. 1.

Section of the fourth dorsal segment; owing to collapse of the cavity the cord appears much deformed. There was considerable meningitis around the cord; part of the meninges had been cut away.

degeneration in the periphery of the white columns; this persists through all the higher segments of the cord, being most pronounced on its ventro-lateral surface. Further, a partial degeneration of the spino-cerebellar tracts commences at this level.

In D_6, D_7, D_8, and D_9, there is little change; a large cavity, which sends branches into each lateral column, extends from the extremity of the one dorsal horn into that of the other. The gliosis around it reaches the periphery of the cord at, and lateral to, the entry of the dorsal roots. The meninges are very much thickened, and the peripheral degeneration of the ventro-lateral columns is well marked. The central canal is represented by an irregular row of ependymal cells in the ventral wall of the cavity.

The extent of the cavity in D_1 is represented in fig. 1; the fifth segment is

very similarly affected. Here the walls of the cavity consist mostly of dense sclerotic tissue, giving a fairly smooth surface to its lumen.

In the upper three dorsal segments the disease is perhaps more extensive than in any other region of the cord. As in the segments which have been described, it occupies almost the whole of each dorsal horn and a considerable part of the dorsal column; part of each lateral column is also involved. The cavity wall consists of dense glial tissue with few nuclei, but in places irregular nodules or tongue-like projections of densely sclerosed tissue containing large numbers of round darkly staining nuclei jut into the cavity; these are often very rich in blood-vessels, the walls of which are frequently thickened and hyaline. The less compact glial tissue, on the other hand,

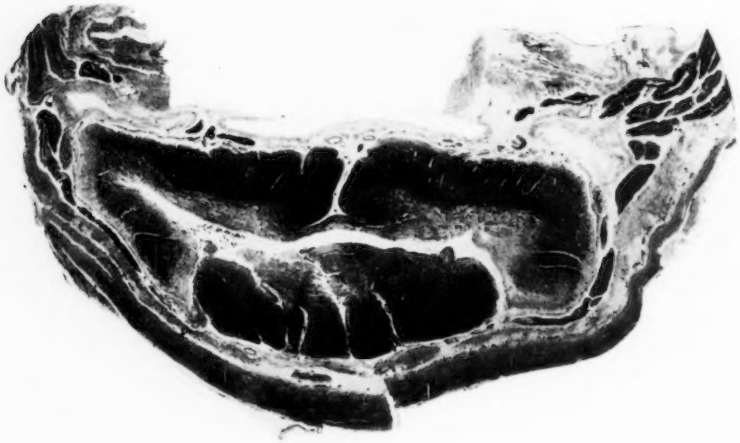

FIG. 2.

Section of the fifth cervical segment. The thickened meninges had to be cut away on the ventral surface of the cord before the section was prepared, owing to the presence of bone in them.

contains larger nuclei, which are granular and stain less deeply. Many of these seem to belong to Deiters's cells.

The surrounding gliosis in each segment extends a considerable distance dorsalward along the dorsal septum, so that in Weigert sections a pale triangular area with base ventralward occupies the centre of the dorsal columns. In the lateral column the gliosis involves the inner portions of the pyramidal tracts, and extends along the dorsal horns to include part of Lissauer's zone. In the second dorsal segment, part of the ventral wall of the cavity is lined by ependymal cells, but in the neighbouring segments the central canal, represented by a column of cells, is separated from the cavity, as in every other segment there is considerable gliosis around it.

In the lower two cervical segments there are two separate cavities, one in each dorsal horn. They are separated by the gliotic tissue which surrounds the central canal. The gliosis at this level extends into the dorsal column and a short distance into each ventral horn; the latter appear somewhat rarefied.

In C_3 the cavity in each dorsal horn again communicates with its fellow across the commissure and the ventral portion of the dorsal column (fig. 2). On the left it passes beyond the grey matter into the lateral column. The ventral horn, though flattened out, contains an almost normal number of large cells. Even at this level the crossed pyramidal tracts are severely degenerated, and there is a considerable degeneration of the dorsal and ventral spino-cerebellar tracts, as well as slight degeneration of the periphery of the ventral columns.

The dorsal part of the dorsal columns is relatively little affected, but there is marked degeneration of their ventral two-thirds, partly ascending system degeneration, partly the result of their invasion by the gliosis. At this level the fibrosis and thickening of the meninges is extreme.

The nature and extent of the disease is almost unchanged in the upper three cervical segments. In each the single cavity which involves the whole of the dorsal horns and the grey commissure sends a large evagination lateralwards into each ventro-lateral column in the plane of the commissure; on the left this almost reaches the periphery of the cord, and on the right more than half-way across the white column. The gliosis that surrounds the cavity is extensive, especially in the ventral part of the dorsal columns. The degeneration of the pyramidal tracts is much less than in the lower segments, and almost disappears towards their decussation. The degeneration of the spino-cerebellar tracts remains distinct, and there is considerable sclerosis of Goll's columns and some irregular degeneration in Burdach's.

At the decussation of the pyramids a large amount of dense gliotic tissue surrounds the central canal and extends dorsalwards on either side of the dorso-median fissure, forming in Weigert sections a pale pyramid with its base ventralward and its apex on the dorsal periphery. The syringal disease is here represented by slit-like cavities which extend almost directly lateralwards to the periphery of the cord at the ventral margin of the substantia gelatinosa Rolandi. There is relatively little gliosis around these cavities. Projections from the central gliosis also extend between the gracilis and cuneate nuclei; each is somewhat rarefied in its centre, but the rarefaction has not advanced to cavity formation.

In higher sections the pathological changes alter but little. At the level of the fillet decussation an excess of sclerotic neuroglia still surrounds the central canal, which has not yet opened out; and from its dorsal portion two slit-like cavities extend ventro-lateralwards towards the ventral margin of the spinal fifth root, running parallel, but slightly ventral, to the outgoing vagus roots. This, with the ascending degeneration of the spino-cerebellar tracts, is the only disease at this level.

The disease remains practically unchanged after the central canal has opened out into the fourth ventricle (fig. 3). The irregular nodules which were visible to the naked eye along the median furrow of the lower end of the ventricle were found to be due to a proliferative gliosis of this region. From here slit-like cavities run, as in the sections just described, towards the spinal fifth root, but they do not reach further than half the distance to the periphery; the internal arcuate fibres bend round the lateral margin of each cavity, and each fasciculus solitarius lies dorso-lateral to it and seems but little affected by it. The disease ceases completely in about the upper third of the medulla oblongata.

Examination of the forebrain revealed no important changes, excepting hydrocephalic dilatation of the lateral ventricles. The foramina of Munro were

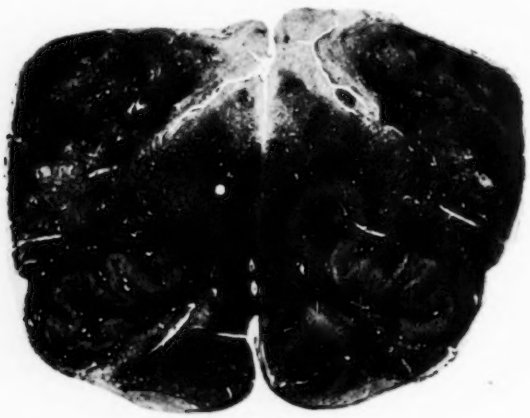

FIG. 3.

Section of the lower end of the medulla showing the gliosis around the calamus scriptorius, and the cavities extending from it ventro-lateralwards.

patent and the aqueduct of Sylvius was much dilated, evidently a result of the internal hydrocephalus. Microscopical examination of the walls of the aqueduct revealed an interesting condition, as in many places its ependymal lining was absent, and here and there a considerable amount of tissue was eroded away or torn through by the distension; at the level of the third nuclei this change extended so deep that the dorso-lateral margin of the nucleus on one side was involved in the secondary gliosis. The view that the rupture of the ependymal lining was due only to mechanical causes is supported by the fact that the ependymal cells which persist are much flattened. The hydrocephalus probably resulted from occlusion of the foramen of Majendie by the meningitis, but as the membranes were unfortunately torn here in removing

the brain it cannot be with certainty asserted that the foramen was completely blocked.

Some of the clinical and anatomical features of this case seem worthy of discussion. The condition which was found on *post-mortem* examination of the nervous system may be dealt with first.

Of the nature of the spinal pachymeningitis there can be scarcely any doubt. In addition to the very definite history of syphilitic infection and of its sequelæ, and of the improvement of some of the symptoms under antisyphilitic treatment, the view that the meningitis was syphilitic was confirmed by the histological changes which we found, *i.e.*, the intimal disease of many of the vessels in the meninges and throughout the nervous system; the hyaline degeneration of the walls of others; and, finally, the presence of perivascular infiltration by small round cells and by plasma-cells. Although the meningitis was most extensive on the spinal cord there was also some on the basal surface of the brain-stem.

On the other hand, there can be no doubt but that the gliosis and the cavity in the cord and the medulla oblongata correspond with the condition which is characteristic of syringomyelia. The cavity occupies the region which is typically occupied by syringomyelia, and the distribution of the disease in the bulb is so characteristic of syringobulbia that there can be no hesitation in including the case in this class. Further, the glial walls of the cavity and its relation to the central canal conform to the changes of syringomyelia.

The nature and the significance of the association of these two processes, the meningeal and the syringomyelic, is extremely interesting and very important in relation to the pathogenesis of syringomyelia. This association is not infrequent; it has been long recognized, but it is not our intention to attempt to review the literature on the subject here; this has been recently done by Rhein [11], who succeeded in collecting thirty-five cases, and added one of personal observation.

The occurrence of a thickening of the meninges, and especially of the pia mater, is often observed over spinal cords in which there has been extensive parenchymatous degeneration, the meningeal being secondary to the medullary disease, but independent of its etiology. That such secondary thickening of the membranes may occur in a cord which has been severely or for long involved by syringomyelia is not surprising, but such cases must be excluded from consideration here. There remain, however, a considerable number in which the association of the two conditions can be explained only by the assumption

of a coincidence, or of a common etiological factor, or by regarding the syringal disease as secondary to the meningeal.

The proportion of cases of syringomyelia in which a meningitis occurs is so considerable that it is scarcely possible to assume a mere coincidence. But before attempting to decide on the other alternatives it is necessary to determine, as far as possible, the nature of the meningeal changes which occur in these cases.

In a certain number, as in the cases reported by Phillipe and Oberthür [9], by Müller and Meder [8], and by others, there was only a fibrotic thickening of the meninges without changes specific of any cause; in one recorded by Zenoni [18] it was regarded as tuberculous, and in one of Saxer's cases [13] it was a sequel to acute cerebro-spinal meningitis. In a larger series of cases—as in those reported by Japha [6], Schwarz [15], F. Taylor [16], Rhein [11], and in our own—there could be scarcely any question of the syphilitic nature of the pachymeningitis. In Japha's case there was a diffuse gummatous meningitis, and in Taylor's a gumma was found in the spinal dura. The conclusion must be, therefore, accepted that the pachymeningitis occasionally associated with syringomyelia is not always of the same nature.

Against the first alternative, that the spinal and meningeal changes are due to a common cause, it must be urged that the latter are undoubtedly rare in syringomyelia. According to Schlesinger [14] the membranes are quite unaffected in the majority of the cases. On the other hand, a considerable proportion of the cases of syringomyelic pachymeningitis have been syphilitic, and there is certainly little ground for the view that any form of syringomyelia, apart from that associated with such meningitis, is due to syphilis, which it would be necessary to assume if the association of the two conditions is to be ascribed to a common cause.

It seems more probable that the spinal disease is secondary to the meningitis, and due in some way to compression of the cord or interference with its blood or lymph circulation. In favour of this is the fact that when the cord is compressed, or the spinal circulation disturbed, softening is more liable to occur in the region which syringomyelia generally involves—that is in the base of the dorsal horns and the adjacent part of the commissure—than in any other area. It is, however, difficult to determine the exact mode of origin of the cavities and of the gliosis which surrounds them. That a cavity may arise directly by necrosis and absorption of tissue secondary to anæmia and lymph stasis, and become later surrounded by sclerosed neuroglia, is certain; it is

occasionally observed with compression paraplegia or associated with a myelitis, but it is very doubtful if an extensive and regular cavity, such as was present in our case, can originate in this way. A process similar to that described by Schwarz [15] is more likely; the circulatory disturbances excite a primary change in the neuroglia, which proliferates and later assumes a homogeneous appearance with faintly staining properties, owing to degenerative changes or a transudation into it; or the neuroglial changes may be degenerative from the onset. Cavities arise secondarily by rarefaction of these areas, and their walls may remain composed of rarefied or reticular tissue, or become sclerosed.

This view is the more interesting, as it brings the pathology of these cases into line with that of primary idiopathic syringomyelia, in which it is generally believed that a gliosis, or other change in the neuroglia, precedes the cavity formation. Further, the disturbance of circulation and the lymph stasis which excite or produce the glial lesions have been recognized as causal agents, combined with a central gliosis which may be due to a developmental anomaly, in ordinary syringomyelia; Schlesinger attributes great importance to the vascular changes which are almost constantly found in the spinal cord in this disease.

There are, however, difficulties in accepting the view that in such cases the meningeal lesions are the sole agent in the production of the syringomyelic changes; there is the fact that in our case the cavity formation was not in any way parallel to the degree of meningitis. In the bulb, for instance, the lesions, which were typical of ordinary syringomyelia, were associated with only a very slight amount of local meningitis or vascular disease. In the cases of Rhein and Saxer, in which there was also syringobulbia, there was more meningitis about the medulla.

On the whole it is most reasonable to accept the view which has been repeatedly advanced that the condition we call syringomyelia is not always of the same nature or due to the same cause, and to conclude with Schlesinger that there is a sub-variety which is the result of meningeal and vascular disease, and unlike the more common form is independent of developmental anomalies. Though the existence of this sub-variety be admitted it seems impossible to associate it at present with any definite group of symptoms. Our own case as well as Taylor's and others show that it is not always, as Rhein states, "characterized clinically by the presence of sharp pains in the distribution of the nerves and other subjective sensory phenomena."

The existence of an internal hydrocephalus was another point of

interest in our case; to its onset, or perhaps to an acute exacerbation in its course, the paralysis of the limbs and the symptoms of increase of intracranial pressure of which the patient gave such a definite history were doubtlessly due. The co-existence of hydrocephalus with syringomyelia is not exceptional; Hinsdale [4], in searching through the literature, found mention of this condition in 15 out of 150 cases of syringomyelia. In some of these the hydrocephalus was apparently idiopathic, or more correctly its cause could not be ascertained; in our case and in others the existence of a diffuse meningitis may explain its origin. The extraordinary dilatation of the aqueduct of Sylvius indicated that the obstruction must have existed at or behind the foramen of Majendie.

Optic neuritis, such as existed in our case, has also been observed, but only very rarely, with syringomyelia. In a case reported by Saxer [13] the diagnosis was confirmed by autopsy. There was a cavity in the cervical cord, which was also much enlarged, and some hydrocephalus. To the latter condition the optic neuritis was undoubtedly directly due, while it probably resulted from blocking of the flow of cerebro-spinal fluid by the enlarged cord. It was thus that Taylor and Collier [17] have explained the occasional association of optic neuritis with tumours and myelitis in this region of the cord. Bullard and Thomas [1] also found optic neuritis associated with syringomyelia in a boy, aged $6\frac{1}{2}$; in this case, too, there was considerable hydrocephalus. The diagnosis in Weisenberg and Thorington's case [18] was not confirmed by autopsy.

Case II.

Syringomyelia without Symptoms, Associated with Intracranial and Spinal Tumours.

A. N., aged 46, was admitted to the National Hospital in July, 1905, under the care of Sir William Gowers, to whom we owe our thanks for permission to report the case.

He complained of headache and gradual loss of sight, and according to his history he had been subject to general epileptiform fits at long intervals for the preceding seventeen years. For some months he had also had true "dreamy states" unassociated with convulsive attacks.

On examination the optic discs were found in post-neuritic atrophy, with corresponding loss of vision. Beyond slight paresis of the left external rectus there was no abnormality of the ocular movements, excepting nystagmus on conjugate deviation to the right; the other cranial nerves were unaffected.

All movements of the head and neck were natural, and in the upper extremities nothing abnormal was found beyond slight tremor of the hands

when held outstretched; there was no weakness, wasting, spasticity, or ataxia. The muscles of the thorax, spine and abdomen also contracted well and equally on the two sides. All movements of the lower limbs were well performed; there was no spasticity, wasting or ataxia, and gait was perfectly normal. No unsteadiness was observed when the patient closed his eyes and stood upright with his feet together.

On testing sensation it was found that the appreciation of pin-pricks and of cotton-wool touches was everywhere acute, and the discrimination of temperature was everywhere accurate. There was no astereognosis or atopognosis.

The deep reflexes were diminished in both the upper and lower limbs, and the abdominal and epigastric reflexes were not obtained; the abdomen, however, was flabby and pendulous, a fact sufficient to account for their absence. The plantar reflexes were invariably of the flexor type on both sides. At no time was there any affection of the sphincters, beyond a slight delay in starting micturition.

About a week after admission a tumour which had been diagnosed in the right tempero-sphenoidal lobe was removed by Sir Victor Horsley; it was circumscribed and of oval shape, measuring 6 × 4 cm. The patient, however, died in an attack of respiratory failure some time after the operation.

At the *post-mortem* examination it was found that the tumour which had been removed had grown from the dura mater of the anterior part of the middle fossa of the skull. Another tumour of almost equal size lay imbedded in the tip of the left frontal lobe; while several smaller nodules of growth, of the same firm consistence, were attached to the dura mater covering the surface of the hemispheres, and particularly to the falx cerebri and tentorium cerebelli. To the naked eye all tumours were similar in structure; all undoubtedly took origin from the dura mater and compressed, but did not invade the brain, being everywhere separated from it by the pia-arachnoid, which was stretched out unbroken over their surfaces.

Histologically, all these tumours were identical in nature and conformed to the structure of the psammomata. They were rich in nuclei, but very little cytoplasm could be distinguished around them; the nuclei were generally spindle or oval in shape. Throughout large areas the cells were arranged irregularly, but much of the tumour consisted of cells arranged in concentric whorls. Following the development of these whorls, it may be seen that as they increase in size the cells, and particularly those of the inner layers, become flattened concentrically. The next stage in their evolution is apparently hyaline degeneration, which commences in the centre, so that we have a core which is structureless, or perhaps contains the badly staining remnants of nuclei, surrounded by cells which are as yet intact.

Occasionally two or more whorls become enclosed in a common sheath of flattened endothelial cells, which later may also undergo hyaline degeneration. After the development of this retrograde change calcareous granules are often deposited in the hyaline centres of the whorls; these may gradually accumulate and form homogeneous calcareous masses, which either lie free in the tissue of

the tumour, or remain enclosed in a capsule of flattened cells. The vessels of the tumour have well-formed walls, but a few of them are in the earlier stages of hyaline degeneration.

The structure of these tumours, though not directly connected with our subject, is given in detail, as it is important to recognize clearly that both in structure and nature they are entirely distinct from that which will be later described in the spinal cord.

No part of the brain-stem was compressed by any of these tumours, and none were found in the spinal dura mater.

The membranes of the cord were normal. When removed, the spinal cord appeared natural from the bulb to the fourth dorsal segment; at this level it increased considerably in size, and then remained fairly uniform in diameter to the level of the eighth dorsal roots. The ninth and tenth segments were much more enlarged, but in the last dorsal segment the size diminished again rather abruptly to the normal. The lumbar and sacral segments appeared unaffected to the naked eye.

After hardening in formalin blocks from each segment were prepared for the Weigert method; sections from each of these blocks were also stained with hæmotoxylin and picro-fuchsin; from a few levels pieces of tissue were taken for Weigert's neuroglia stain.

The topography of the disease is most easily studied in the Weigert sections: in these, as in van Gieson's preparations, no disease is visible in the upper seven cervical segments. In the eighth cervical segment, however, dense fibrillar neuroglia appears round the central canal, and extends into the dorsal columns along the dorsal septum, and into the base of each dorsal horn: in the latter there is some rarefaction of the tissue, but as yet no definite cavity formation.

In the first dorsal segment, similar sclerosed neuroglia surrounds the central canal, which is here dilated into a tube of considerable lumen, completely lined by ependymal cells. A certain amount of sclerosis still extends into the dorsal columns, along the dorsal septum.

In the second dorsal segment the changes are similar, but in places the ependymal lining has been broken through, and coarse thick neuroglial fibrils encroach on the lumen of the central canal.

In the third dorsal segment the central canal is still more distended, and has a less complete lining of ependyma. Here a second cavity appears in the middle of the right dorsal horn: it is irregular in shape, and is limited only by loose reticular but sclerosed neuroglia, which stretches a considerable distance from its edges. From a study of the sections stained by different methods it seems certain that the cavity has arisen by rarefaction of a pre-existing gliosis.

In the fourth dorsal segment the disease is more fully developed; a large cavity, very irregular in shape, occupies the whole of the right dorsal horn and the greater part of the grey commissure. A second cavity, which is evidently continuous with the dilated central canal, lies to its left and near the base of

the left dorsal horn; its wall is formed by irregularly proliferated ependymal cells, while the larger cavity is lined by sclerosed neuroglia alone [fig. 4]. Both are enclosed in a common mass of dense neuroglia, which also invades the ventral portion of the dorsal columns, especially along the dorsal septum.

The cavity in the right dorsal horn is still larger in D_5, while the central canal is almost obliterated; its lumen is lined by a single layer of typical ependymal cells; other ependymal cells form a solid column along its ventral margin, and infiltrate the sclerosed glia.

In D_6 a mass of dense neuroglia occupies almost exactly the position in which in the section above lay the larger cavity; this is here represented only by a narrow slit, while the central canal is marked by an irregular column of ependymal cells, such as is found in many normal cords.

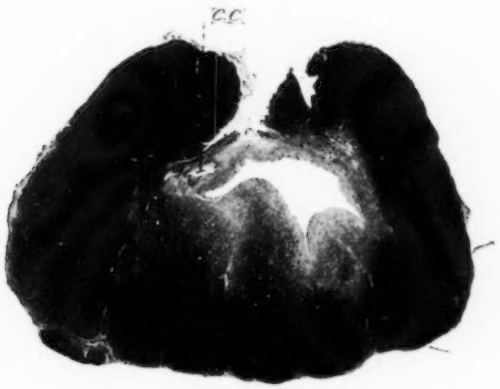

FIG. 4.

Photograph of a section of the fourth dorsal segment stained by Weigert's method; *c.c.* is the central canal.

In the seventh dorsal segment two irregular cavities lie within the mass of sclerosed neuroglia in the right dorsal horn and the neighbouring part of the commissure (fig. 5). The central canal is displaced to the left, but still lies ventral to the gliosis. The cavity ceases between the seventh and eighth dorsal segments, and in the upper part of the latter there appears the large central tumour which was visible to the naked eye. To establish the relationship between the two it is necessary to study serial sections of this region. At the upper level of the change we find first a considerable proliferation of the ependymal cells of the central canal. These form a solid mass which lies at first ventral and to the left of the gliosis and its cavity, but gradually becoming larger spreads along the inner part of the left dorsal horn, so that it comes to lie to the left of the syringal disease. In serial sections it may be easily seen

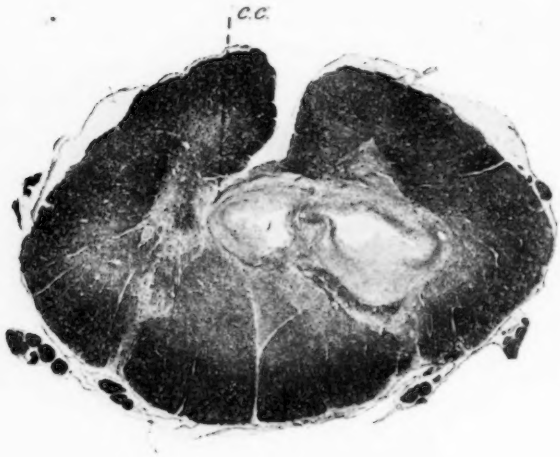

FIG. 5.

Photograph of the seventh dorsal segment; *c.c.* indicates the position of the central canal.

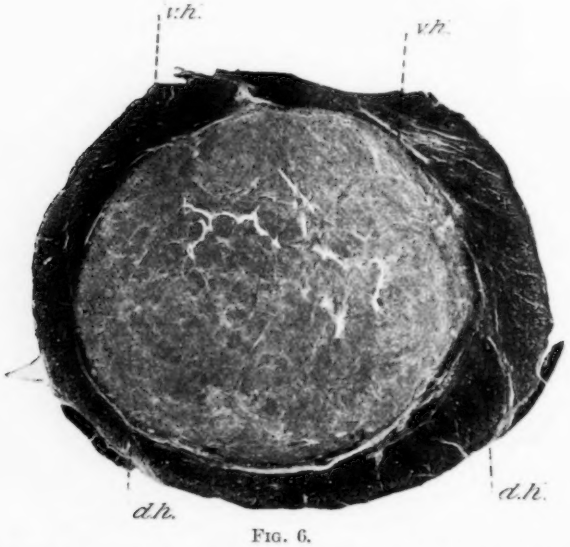

FIG. 6.

A section of the middle of the ninth dorsal segment stained by Weigert's method; *v.h.* the ventral horns; *d.h.* the dorsal horns. The unstained mass in the centre of the cord is the tumour.

that the tumour arises in connexion with, or evidently from, the ependymal cells of this irregular representative of the central canal. It increases very rapidly in size, and in the middle of D_8 it occupies the whole of the centre of the cross-section of the cord; in lateral diameter it here measures about 10·5 mm. and 9·75 mm. in dorso-ventral diameter, while its cross-section is approximately 78 s.mm., that of the whole enlarged cord being 132 s.mm. As the tumour enlarges the syringal cavity and gliosis disappear; in the middle of this segment no trace of them is visible, while the tumour, which is almost circular in cross-section, is surrounded by an almost regular layer of nervous tissue, varying from 1 mm. to 2 mm. in thickness, uninfiltrated and apparently normal in Weigert preparations. In D_9 the tumour is still larger (fig. 7); its area in cross-section is about 113 s.mm., while that of the whole cord is 154 s.mm. The zone of normal tissue that surrounds it is narrower than in the section above, but the tumour nowhere comes to the surface. The ventral horns, which are very much flattened out by it, are the only parts of the grey matter that can be easily recognized; the dorsal horns can be located only by the entry of the dorsal roots. The tumour ends rather abruptly in about the middle of the D_{10} segment. Below the next segment there is no visible disease, and despite the extent and intensity of the focal lesions which have been just described there is no descending secondary degeneration of the pyramidal tracts or of any of the other systems of the lumbo-sacral cord. There was also no trace of ascending degeneration recognizable by the ordinary methods in the upper cervical segments.

The walls of the large syringal cavity are nowhere lined by ependyma; they consist only of gliotic tissue of variable density, in which the glial strands or fibres are irregularly arranged without any trace of membrane-like formation. Where it is less dense the gliotic tissue appears reticular, and consists of irregularly interwoven thick glial fibres, which branch and anastomose with one another. Where denser it has often a gelatinous appearance under a low magnification. The gliosis contains a considerable number of Deiters's spider-cells; the terminal portions of their processes may be often seen to become continuous with the glial fibres. The gliotic tissue has, as a rule, very few nuclei, especially towards the edge of the cavity; here the tissue appears to be undergoing rarefaction at a very slow rate. It is more dense as well as more rich in nuclei in its outer portions; it is not, however, anywhere sharply limited from the normal spinal tissue, into which dense glial strands penetrate irregularly.

The relation of the central canal to the syringomyelic cavity, and more especially to tumour, must be accurately described. In the upper cervical segments the canal is represented, as is usual in the adult, by a solid column of ependymal cells. In C_8 it becomes patent, and is completely lined by a layer of ciliated ependymal cells. In the first and second dorsal segments its lumen is considerably larger, and is still lined, or almost completely lined, by ependyma. Here there is a typical hydromyelia. As the syringal cavity develops, the lumen becomes smaller, and the canal is compressed and displaced

towards the base of the left ventral horn. In D_6 and D_7 the canal is again represented only by a column of ependymal cells, which are much more numerous than normal, but it still lies in the typical way ventral to the gliosis. In the upper part of D_8 the proliferated ependymal cells begin to form a definite tumour-like mass, which spreads at first dorsalwards into the dorsal column and the neighbouring portion of the dorsal horn, and finally comes to occupy practically the whole of the centre of the cord in the eighth, ninth, and tenth dorsal segments (fig. 7).

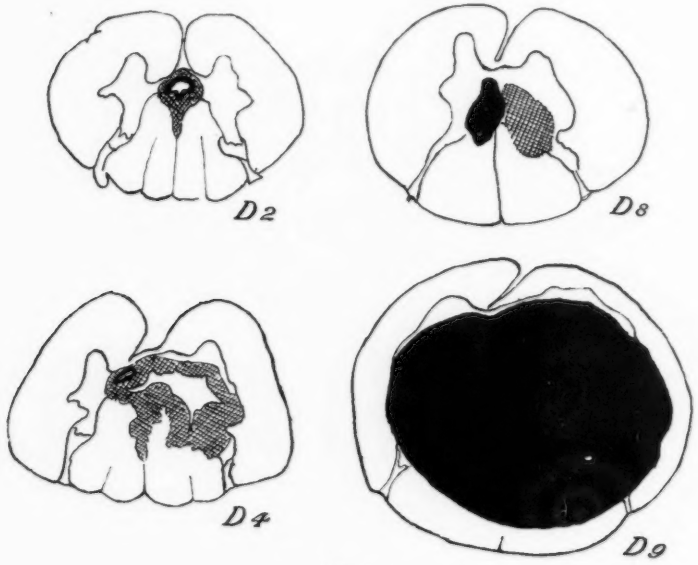

FIG. 7.

Drawings to show the relation of the central canal and its ependymal cells to the spinal tumour and to the syringomyelia. Ependyma black, the syringal gliosis cross-hatched.

The structure of the spinal tumour is simple; although it does not infiltrate the spinal tissue that surrounds it, it is not sharply limited from it by any capsule or membrane. It is composed of very irregularly arranged strands of fibrillar tissue, in which there lie large numbers of fairly large nuclei, oval when cut in length, round when cut in cross-section; but in many places, and especially around the vessels, there is a complete absence of such nuclei. They stain rather deeply and contain coarse chromatin granules. The fibrillar tissue has the structure and staining reactions of sclerosed glia, and along the margins

of the tumour its strands run into and fuse with the neuroglia of the normal spinal tissue. The vessel walls are well formed, but many are thickened, and some are hyaline. At the lower end of the tumour there is an excess of vessels. In places in the outer portions of the tumour a considerable amount of cytoplasm collects around some of the nuclei, and often runs out into irregular branching processes, which may or may not be connected with the fibrils of the stroma of the tumour. There is no visible degeneration, and remarkably little evidence of reaction or sclerosis in the nervous tissue around the tumour, despite the compression to which it had been subjected. The tumour apparently started in the centre of the cord and slowly displaced, but did not destroy, the tissue in which it grew.

Histologically, the tumour is undoubtedly a glioma which has arisen from the ependymal cells of the central canal, but there is nowhere a tendency to a glandular arrangement of the cells, such as Rosenthal [12] has described in a somewhat similar case.

It is our intention to limit our remarks on this case, which is as remarkable from the clinical as from the pathological point of view, to as small a compass as possible.

In the cord were found two conditions, distinct from, though possibly related to, one another.

The extensive primary gliosis in the dorsal horns, the grey commissure and the neighbouring portions of the dorsal columns, with the cavity formation within it, is typical of syringomyelia. On the other hand, the large tumour which lay in the lower dorsal segments was undoubtedly a glioma, which apparently grew from the ependymal cells of the central canal, these having assumed the *rôle* of glioblasts.

It is difficult to say how far the syringomyelic lesions were dependent upon the existence of this tumour. We may, in the first place, exclude the possibility that that cavity has arisen by softening and disintegration of portions of this tumour which had previously extended through the upper and middle dorsal segments; this process, it is well known, occurs in spinal gliomata and produces extensive intramedullary cavities, but in our case the structure of the tumour is entirely distinct from the gliosis which surrounded the cavity; and there is no evidence of the previous existence of such a tumour in the upper dorsal segments. Finally, where the two conditions co-exist, as in the upper portions of D_8, they remain anatomically distinct.

On the other hand, it must be remembered that intramedullary cavities not infrequently arise in connexion with spinal tumours, or even secondary to compression of the cord. It appears improbable,

however, that in this case the tumour was an etiological factor in the development of the syringomyelia, more particularly as from its structure it appears to be more recent than the gliosis which surrounds the cavity.

More probably both conditions arose independently of one another, though possibly dependent upon a common developmental anomaly; the existence of tumours in other tissues, as in the cerebral dura mater, makes it probable that there was some inherent bioplastic anomaly in the central nervous system and its membranes.

The association of the syringomyelia with the cerebral tumours is probably to be explained by the same assumption—that both were dependent on developmental anomalies. It is, however, of interest to note that the association has been not infrequently observed. In the cases reported by Gowers [2] and by Harris [3] there were, in addition to sarcomatous tumours in the pons Varolii, secondary deposits within the spinal theca; owing to this complication these cases need not be further considered.

Langhans [7], however, found in three cases in which tumours lay in the region of the fourth ventricle cavities in the cervical and dorsal segments of the cord, which, though in places connected with the central canal, were probably syringomyelic. In these cases there were no secondary deposits in the cord, and as one tumour was a melanotic sarcoma, and the other two sarcomata, they could scarcely have been directly related to the cavity formation. According to Langhans, the latter resulted from the blood-stasis in the cord which was produced by the compression of the spinal veins as they enter the skull through the foramen magnum.

Pick and Schultze [10], however, found syringomyelia associated with a tumour of the corpus callosum, and Homén [5] with one in the tempero-sphenoidal lobe. In the majority of the recorded cases the tumours have been gliomata, as might be expected since these are the most frequent of all cerebral tumours; but tumours of other nature, as in our own case, have also been found.

From the clinical point of view the complete absence of all symptoms and physical signs pointing to a spinal lesion is remarkable; and this despite the fact that, owing to the difficulty in the accurate localization of the intracranial disease, the patient was repeatedly examined with all possible care. It is impossible to offer a full explanation of this fact, but it may be again pointed out that the spinal tumour displaced and compressed the spinal tissue rather than destroyed it.

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A CASE OF HÆMORRHAGIC MYELITIS.

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THE following case is remarkable for the slight changes found in the central nervous system after death, in comparison with the severe symptoms present during life, and which led directly to a fatal termination :—

Mrs. F., aged 46, had always enjoyed good health, except for an attack of scarlet fever, followed by nephritis, eighteen years previously. The nephritis was completely cured after two years. At the beginning of March, 1907, she helped to nurse a friend who was suffering from a rapidly fatal attack of acute lymphatic leukæmia; the nursing involved night-work and a good deal of lifting. After a few days she felt very tired, and on the evening of March 8 she was suddenly seized with palpitation, a sensation of constriction in the chest, and loss of power in the arms and legs, so that she had to sit down and support her head and shoulders on the table. After a short time she recovered and sat up that night with her patient, although her legs felt heavy and weak. She went on fairly well until March 10, when, after a good night in bed, she had an attack of general trembling. She got up, however, but on sitting down to breakfast she suddenly lost all power in her limbs and back, the muscles being flaccid and helpless; she would have fallen from her chair, but was caught and lifted into a large easy chair. The arms felt as if tied to the body, both arms and legs were powerless, and she could feel nothing on the trunk or limbs below the neck. Evidence of vasomotor disturbance was present in the form of extreme pallor of arms and hands, and of flushing of the face. Two or three hours later she was able to move her arms, and felt "pins and needles" in arms and legs. Later severe pain appeared along the left

lower costal margin, and during the next few days she suffered also from shooting pains in the limbs and trunk.

There was no loss of consciousness in either of the initial attacks, and no pain. The pulse during the second attack fell to 40, and remained at this rate until March 11, when three attacks of palpitation occurred, in which the pulse rose to 110. On this day also

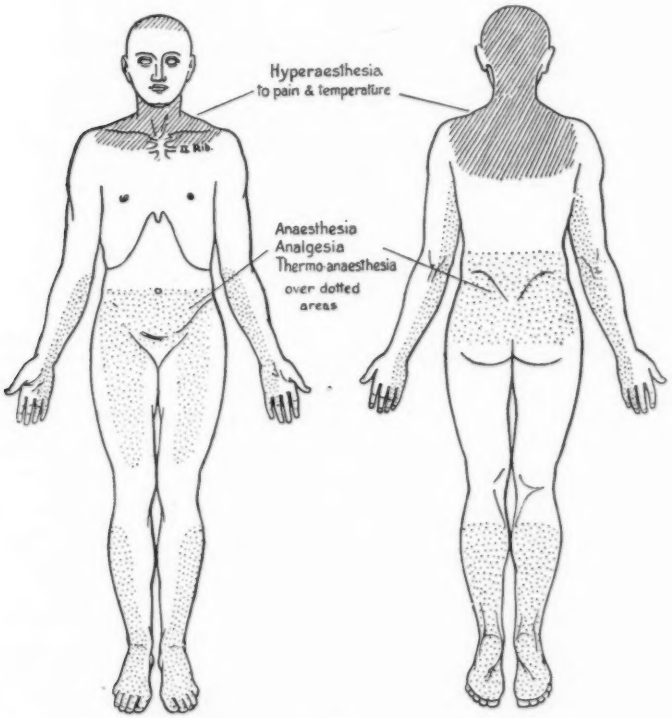

FIG. 1.

obstinate vomiting set in and lasted for three days. There was no affection of the bladder or rectum.

On March 15 her condition was as follows: The pulse was 40, hard and regular, the temperature normal; and respiration 20. She was unable to stand or walk, paralysis of the legs being almost complete. She was able to grasp things with the hands, and had fair power of

movement in the arms. There was some incoördination in both arms and legs, and some loss of the sense of position of the limbs.

She complained of a good deal of pain in the scapular regions, neck, and arms. The main nerve-trunks of the arms, and in the legs the peroneal and posterior tibial nerves, were tender to pressure. The limb-muscles generally were also somewhat tender to pressure. Hyper-

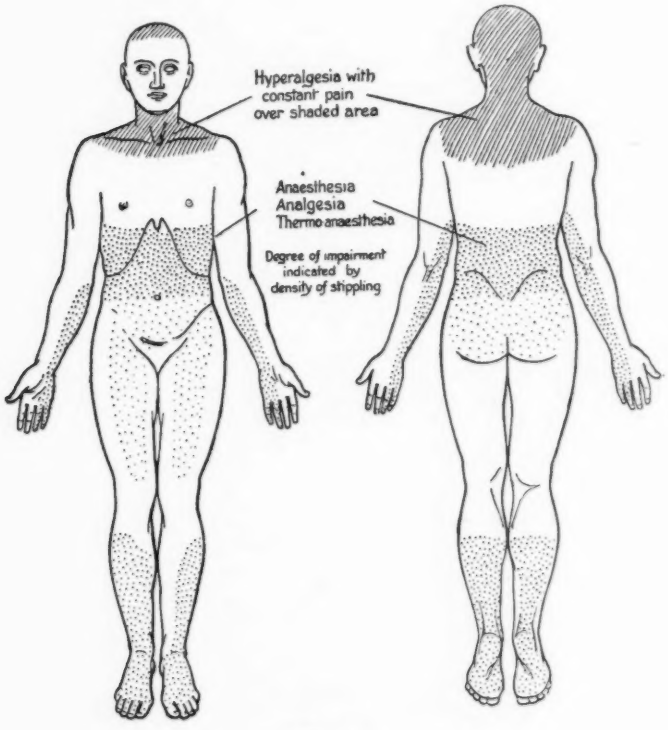

FIG. 2.

æsthesia of the skin was present over the suprascapular regions, over the front and back of the neck, and the posterior aspect of the scalp as high as the vertex. Sensation to touch, pain and temperature was lost over the areas supplied by each ulnar nerve and by the musculo-cutaneous and external popliteal in the legs (fig. 1).

The knee-jerks were exaggerated; there was no ankle-clonus; the abdominal reflexes were present, the plantar absent.

The patient's condition remained much the same for some weeks. But some wasting of the thenar and hypothenar eminences in each hand, and to a less degree of the interossei, made its appearance. She complained of a persistent sweet taste in the mouth, neuralgic pains in the cheeks and about the orbits, and a sense of oppression in breathing, due to partial or complete paralysis of the diaphragm; coldness of hands and feet appeared, with great irregularity of the pulse-rate, which varied many beats within a few minutes.

On May 10 the motor paralysis was unaltered. Figs. 1 and 2 show the disturbance of sensation at this date and on May 21. Some incoördination in both arms and legs was observed. The reflexes remained as before. The muscles were generally soft and flaccid, except those of legs, which showed slight spasticity.

Reaction of degeneration was present in the wasted muscles of thenar and hypothenar eminences; elsewhere some diminution of excitability to faradic current.

We may mention here that the temperature was normal or subnormal throughout, and the urine contained no albumen.

No further change occurred in the condition until June 14, when profound nausea with a persistent sweet taste in the mouth came on. There was no sugar in the urine or saliva. The tongue was clean and moist. The nausea and loathing of food became so extreme that she finally refused absolutely to take any nourishment, and had to be fed by enemata. The nausea was accompanied by three or four attacks of vomiting daily, quite irrespective of taking food, and not controlled by the various drugs tried.

Until June 23 she suffered much from pain and tenderness in the neck, which prevented her from moving her head. After this date the cervical pains disappeared, and with them the hyperalgesia of the occipital and nuchal regions. On June 28 retention of urine appeared for the first time, and on this day she also complained of oppression in breathing and the respiratory rate fell to 16, whilst the pulse-rate, which for some time had been at 60, rose to 85, but remained regular. On June 29 the respiration fell from 16 to 12, and on one occasion to 6 per minute, and with this the pulse went up to 106, the highest pulse-rate being associated with the slowest respiratory rate. On the 30th, respiration 6 to 4 per minute, pulse 160; the temperature, previously subnormal, rose to 99·5° F. On the evening of this day the respiration rose to 30, but

at 9.30 p.m. fell to 6 and then to 2, when she died, with a full, regular pulse of 120, the cause of death being evidently respiratory failure.

Dr. Mackay obtained leave with great difficulty to examine the brain and cervical spine, but all further examination was positively refused. The brain appeared healthy in all parts. As to the cord, to the naked eye there was marked congestion of the dūra mater on its posterior aspect, and it was swollen at the level of the third and also at the sixth and seventh cervical nerves. The vessels of the pia-arachnoid were congested, intensely so on the posterior aspect, beginning above at the sixth cervical, and increasing in intensity to the third dorsal roots. The posterior roots themselves appeared reddened, the anterior perhaps slightly so. On incising the cord a few minute red points and streaks were seen, more especially in the posterior root-zone on either side, from the seventh cervical to the third dorsal levels, and the cord was softened at the level of the eighth cervical and first dorsal segments. After three to four days in 4 per cent. formalin the pons, medulla, and cord were transferred to the various fluids necessary for the following stains: sections were cut in close series and stained by Marchi's and Busch's methods, Weigert, Nissl, and van Gieson stains, hæmatoxylin, rubin and rosin, and by a general stain for micro-organisms.

It may be said at once that Marchi's and Busch's methods gave negative results and that no micro-organisms were seen. Sections stained by Weigert's method also showed that the white matter of the cord was healthy and that the fine plexus of nerve fibrils in the grey matter was almost everywhere intact. In a few places, however, where the vascular changes described below were most marked, the fine fibrils were deficient in number. The vascular changes were very conspicuous by this stain, and in some of the perivascular channels there were fat droplets.

By the remaining methods the chief changes found were (1) intense congestion and dilatation of vessels. In the upper part of the cervical cord, as low as the sixth or seventh cervical roots, this affected almost entirely the anterior spinal artery and its branches; below this the posterior spinal arteries were equally affected; the injected vessels showed, from the number of leucocytes they contained, that there was a well-marked leucocytosis going on; (2) escape of red blood-cells into the perivascular channels; (3) microscopic hæmorrhages with a little (never great) destruction of grey matter in their immediate periphery; (4) extensive proliferation of epithelium of central canal.

The injected vessels were most conspicuous in the cervical cord at

the inner side of the base of the anterior cornu, and often also at the base of the posterior. The dilated vessels were either themselves so distended as to render inconspicuous the perivascular sheaths, or the latter contained red blood-cells; the capillaries and minute arteries from these dilated blood-vessels could be easily traced through the grey matter; they were greatly distended, the sections often looking like an injected specimen of the cord. In some of the minute arterioles and veins there was noticed swelling and proliferation of the lining endothelium, and in some places a similar appearance in the cells lining the perivascular space. There were one or two microscopic hæmorrhages into the tip of anterior horn at the level of the second and third cervical roots. As low as the fifth cervical the grey matter was unaltered, and, except for excessive pigmentation in places, the anterior horn-cells were normal, even in the immediate contiguity of the most striking vascular changes. From the fifth to eighth cervical segments the vascular changes were even more pronounced, and here a small number of the anterior horn-cells had suffered, showing mostly the first degree of change by Nissl's method; a very few were altogether degenerated. These injured cells were always in the immediate neighbourhood of the most marked vascular affection, and belonged to the anterior mesial, antero-lateral and posterior lateral groups respectively at different levels, and the whole number affected was but small; in fact, a striking point was the integrity of the nerve-cells and grey matter generally side by side with the great vascular congestion. On the whole, the grey matter was most damaged at the base of the anterior horn around the group of large branches of the anterior spinal arteries.

From the first to fourth dorsal segments the pia mater on the dorsal surface of the cord, unaffected above this level, was swollen, its vessels injected, and there was inflammatory exudation into it. The vessels entering with the posterior roots and their capillary branches were enormously distended, and contained a very large number of leucocytes; many capillaries were apparently thrombosed; the grey matter of the posterior horn was swollen and had a homogeneous appearance. Here the anterior horns were little affected, but below this again, so far as available material reached, they showed well-marked changes.

Similar appearances were found in the medulla. At the lower part of the decussation of the pyramids the left tubercle of Rolando was deeply injected, the capillaries filled with leucocytes, a very few of which were passing into the surrounding grey matter, which in places appeared granular, and stained badly.

At the level of the tip of the calamus scriptorius, and for a short distance above it, the vessels lying just below the dorsal surface and supplying the vago-accessorial nucleus were intensely injected, even the smallest capillaries standing out clearly; in some the red cells had escaped into the perivascular space. Ventralwards, an enormously distended vein was seen in the position of the nucleus ambiguus. In all these vessels there was evidence of a marked leucocytosis. The cells of these nuclei, however, seemed intact and healthy. At the upper end of the hypoglossal nucleus a microscopic hæmorrhage had occurred in a small part of its dorsal area; the capillaries here were full of leucocytes and contained hyaline thrombi; the rest of the nucleus, constituting about two-thirds of the whole, appeared intact. The respiratory bundle, "slender column," and nerve-roots were healthy.

Above the level of the sixth nucleus, close to the raphé and just below the grey matter of the fourth ventricle, was another small hæmorrhage, and around it the capillaries were distended and full of white cells; the vessels on each side of the raphé were distended in most sections. The vessels in the pia mater on the ventral surface of the pons were injected, and in the pons itself those about the central grey matter and raphé also appeared congested, but no hæmorrhages were found.

The cells of the various nuclei, the reticular formation, and the longitudinal and transverse fibres of the pons and upper part of the medulla were normal.

In spite of the great injection of the vessels and the number of leucocytes, there was no evidence, except in one slight and unimportant instance, of any invasion of the surrounding nerve tissue, either grey or white matter, by leucocytes.

Even allowing for the variations in the appearance of the central canal in normal cords, as regards its patency and the number of cells of epithelial type which surround it, the changes in this case call for some remark.

In the upper dorsal cord the normal ring of columnar epithelium was intact, the canal was of normal size, and in it could be counted in different sections from six to as many as thirteen lymphocytes. The epithelium was proliferating at its base, so that the canal was surrounded by several rows of these proliferated cells which were passing into the tissue of the commissure. Above this the canal was more or less obliterated throughout, and in the greater part of the cervical region its place was occupied by a rounded or oval mass of cells, obviously derived from the lining epithelium. These cells had a fairly abundant protoplasm, staining lightly and diffusely, the outline or

border of the cell being difficult to distinguish and containing a clearly defined large oval nucleus with an abundant chromatin network of the type of the nucleus of an epithelial cell. This change affected the canal right up through the medulla until it opened out into the fourth ventricle. Where it was most advanced the original line of epithelium was undistinguishable, but where this epithelium remained it could be plainly seen that the proliferated cells originated at its base and passed off to invade the surrounding tissue, often for a considerable distance.

Finally, both anterior and posterior nerve root fibres appeared healthy at all levels and contained no degenerated fibres. As it has been mentioned that the vessels contained many leucocytes, and the patient was at the time of onset engaged in nursing a case of acute lymphatic leukæmia, it may be well to mention that there was no suggestion of this disease in the present case.

Some of the pathological changes found explain fairly some of the symptoms present; thus the slowing of the pulse and respiration, both early in the illness and again towards its close, would be accounted for by recurrent microscopic hæmorrhages in the region of the vago-accessorial nucleus. The persistent nausea and vomiting would also attend a lesion of this nucleus, and possibly the persistent and nauseating sweet taste, of which the patient so greatly complained, might have its origin in the medullary lesion. Even here, however, the integrity of the cells of the nucleus indicated that there was no absolute destruction of it, and that the disturbance was a functional one. The pains around the chest, the early loss of sensation on the ulnar side of the forearms, and the paræsthesia felt here may be referred to the great injection of the posterior roots, the inflammation of the dorsal pia mater, and the swelling of the grey matter of the posterior horns in the cord, beginning at the level of the eighth cervical and extending to the fourth dorsal roots.

The other pathological changes in the cord, however, consisted, with the exception of a very few microscopic hæmorrhages and also of a few altered nerve-cells (showing chromatolysis, with very few exceptions, in an early and recoverable form), merely of intense vascular congestion. Although the affected vessels were those that supply the grey matter, this showed slight alteration, and only in a few places. It had apparently sustained no extensive or widely spread damage. A striking feature was the way in which the grey matter often appeared healthy, both in its cells and fibres, and by different methods of staining, in the immediate neighbourhood of the most marked vascular congestion.

The white matter appeared uniformly healthy.

It is in respect of the slight extent of the pathological lesions when compared with the severity of the clinical symptoms, and especially the completeness of the paralysis, that the case seems worthy of record, in spite of the fact that the whole cord could not be obtained for examination. This was most unfortunate, but from the onset of symptoms in the arms, neck and chest, and the subsequent paralysis of the arms, it may be fairly inferred that the changes in the lower part of the cord were of the same nature. The case also in no way corresponded to one of peripheral neuritis. The suddenness of onset at once suggested a vascular lesion, either thrombosis or hæmorrhage.

It is conceivable that in a case in which, following an intense microbic or toxic infection of the cord, a rapidly spreading paralysis is followed by death after a certain number of hours, *e.g.*, in the most acute forms of ascending spinal paralysis, no striking changes should be found in the cord even by modern histological methods, but that in one where the paralysis was so marked and lasted for nearly twelve weeks so few indications of disease should be found was contrary to all expectation.

So far as can be judged from the character of the lesions, they were due to the action of the poison, possibly infective ; unfortunately there is no further evidence of its nature. The changes in the central canal, the grouping of the lesions around it, and their greater intensity in its neighbourhood, are suggestive that, whatever the nature of the poison, it obtained access to the central canal, and thence proceeded to affect the cord. There is good evidence that the central canal is in connexion with the lymphatic system of the cord; it may have been infected by this route. The observations of Schlegel [1] on an infective myelitis (meningo-myelitis hæmorrhagica) in horses are important in this connexion. This affection Schlegel found to be due to a streptococcus, and its course is much more acute than in our case, mostly ending fatally within some days, but the lesions he describes are very similar. They affected especially the pia mater and the grey matter of the cord, and in the latter were situated around the central canal, extending thence towards the periphery, and consisted of injection and dilatation of vessels, extravasations around them, and hæmorrhages.

References.

[1] *Berlin. tierärztl. Wochenschr.*, No. 25, s. 463 ; abs. *Jahresber. Neurol. u. Psych.*, 1907, s. 603.

RECENT WORK ON APHASIA.

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DURING the past three years disorders of speech resulting from lesions of the brain have been the field for much work and of much valuable controversy. At the commencement of this period the subject was surrounded by many difficulties. Ideas of the physiological mechanism of speech had been built up through deductions from the clinical aspects and pathological findings in cases of focal disease of the brain in which the pathological examination had been, almost without exception, incomplete; such cases are therefore relatively inconclusive in the light of our present knowledge of the methods of examination essential in local cerebral lesions to an accurate localization of function. Often authors have reasoned from the external examination of the surface of the brain only, and, indeed, Broca's original observations and his original theory were founded upon such superficial examination. The majority of observers have relied upon the examination of the surface of the brain and serial macroscopic sections; and since it is impossible by the latter method to distinguish the living from the dead tissue with any degree of accuracy, it follows that deductions based upon this method of examination are also inconclusive. No worker had set up the standard of serial microscopic sections throughout the brain as the essential basis of the localization of the lesion in cases of aphasia. One of the results of the recent controversy has been the exclusion, as inconclusive, of all cases in which the localization has not been arrived at by this method.

The many schemes with their accompanying diagrams of the localization and interconnexions of the speech-centres, that have been brought forward by high authorities upon this subject, have been founded more upon theory than upon trustworthy pathological evidence; upon these schemes classifications of the clinical varieties of aphasias have been based, which from the first have been open to the criticism that varieties of aphasia were included and described which

were purely hypothetical, and which had not been met with clinically. These were types of aphasia which should have resulted from lesions in certain situations, had the scheme which gave them birth been a correct representation of the physiological mechanism of speech. The multiplicity and complicity of the schemes and diagrams that have been brought forward are in themselves a proof of the difficulties that have arisen in the attempt to apply them to clinical cases. In many instances cases which have during life shown speech-defects corresponding exactly with a lesion in a certain position according to a particular scheme have, upon pathological examination, shown that the lesion, either in position or extent, was entirely incompatible with the correctness of the scheme. The very cases upon which a new scheme has been built up have proved, years after, upon pathological examination, the undoing of the scheme which had been based upon them. No hypothesis had been raised which was not thrown into doubt by some clinical variety or other.

The very foundations of our ideas of the speech-mechanism, the registration of the elements of speech as "visual word-memories," "auditory word-memories," and "kinæsthetic word-memories," were of necessity purely conjectural, however well they may have served as a working hypothesis. The generally accepted ideas of the localization of the speech-functions in the cerebral cortex were open to just criticism. The localization of the "visual word-centre" postulated by Wernicke in 1876, and placed by Dejerine in the angular gyrus, has received but little support from facts, though perhaps not negatived by them, and it was still a theoretical centre. Lichtheim's "ideational speech-centre" has been abandoned even by its author as a separate centre.

The recognition of the fact that the focal lesions which are productive of the aphasias are never strictly limited to the cortex, that when involving the cortex they are at once both cortical and subcortical, and generally much more subcortical than cortical, has introduced a further difficulty in the localization of these centres, inasmuch as the subcortical portion of the so-called "cortical lesion" may by isolation render functionless regions of the cortex not directly involved in the lesion and apparently healthy.

The localization of the chief speech-centre in the left temporal lobe was proved beyond doubt, but the existence of an "auditory word-centre" in the posterior half of the second temporal gyrus was not proved, the best evidence of such a localization being the case reported by Dejerine [14], in which, as a result of chronic polioencephalitis,

the cortex alone of this region was destroyed, the second temporal convolution being chiefly affected; the clinical picture was that of word-deafness.

At the epoch under consideration the state of opinion as to Broca's localization of the motor speech-centre in the left third frontal gyrus may be gathered from the writings of Dejerine and of his pupil Bernheim, both ardent supporters of Broca's localization.

Dejerine [16], while assured that this centre occupies the "cap" and "foot" of the third frontal gyrus, thought that it extended also to neighbouring gyri, very probably to the second frontal gyrus and to the frontal operculum, possibly, but improbably, to the anterior convolution of the insula. Bernheim [6] was forced to admit that there was not one case on record which had been examined by recent methods and which proved beyond all doubt the truth of this localization.

The distinction between the clinical varieties of aphasia is not always a matter of simplicity. In the recent controversy this fact has been well exemplified, since the same cases have been claimed by one author as instances of "motor" aphasia and by another author as instances of "sensory" aphasia. As this possibility of confusion between the so-called motor and sensory types of aphasia has an important bearing upon the issue dealt with in this paper, it is necessary here to define certain of these types and to point out where confusion may arise. Concerning the types "pure motor aphasia" and "pure word-blindness" there is neither difficulty nor dispute. "Pure motor aphasia" results from a subcortical lesion isolating the speech-centres upon the outgoing side, and its clinical features are loss of or difficulties with spoken language, with the complete conservation of internal language and intelligence, articulation remaining natural. "Pure word-blindness," the type of Dejerine, results from a subcortical lesion which isolates upon the incoming side the speech-centres from the primary visual centres in either hemisphere, its characteristics being inability to appreciate written language, with complete preservation of internal speech, outgoing speech and intelligence. It is to the "pure motor aphasia," as above defined, that Marie applies the term "anarthria," a term that has been used by all previous writers as signifying defects of articulation only.

The "motor aphasia of Broca's type" may be defined as loss or disturbance of spoken language, with some interference with internal speech, and associated almost invariably with alexia, agraphia, and with intellectual defects, these associates varying much in degree and in

persistence. A lesion of the "cap" and "foot" of the left third frontal convolution was held responsible for this variety of aphasia.

The "sensory aphasia" of Wernicke resulted from a lesion involving the posterior two-thirds of the left second temporal gyrus and perhaps also of surrounding gyri. This type was characterized by conspicuous defects of internal speech and of intelligence, by complete or partial inability to comprehend spoken and written language, and consequent on the destruction of this, the chief speech-centre, severe defects of outgoing speech, paraphasia and jargon-aphasia were often present.

A comparison between these two types of cortical aphasia, the "motor aphasia of Broca" and the "sensory aphasia of Wernicke," shows at once that the difference between them is rather one of degree than of kind, and may not therefore be an essential difference, since the aphasia of Broca involves troubles on the sensory side, alexia and amnesia, and the aphasia of Wernicke involves troubles with outgoing speech. That the troubles upon the sensory side in Broca's aphasia are usually slight in degree and transient does not necessarily make an essential difference between the two forms, and while there can be no confusion between well-defined cases of the two types, every gradation from the one type to the other is met with clinically. The question has therefore arisen, "Is the separation of the so-called 'cortical' aphasias into 'motor' and 'sensory' types a just one?" and "Is the difference between them determined by a different localization of the lesion, or by the different degree of damage to function caused by lesions in more or less the same position?"

Westphal, in 1884, drew attention to the fact that cases of Broca's aphasia present some of the characteristics of Wernicke's aphasia, and many authors since that time have emphasized this fact. Von Monakow [8], writing in 1905, states that the distinction between these types is often a matter of the greatest difficulty, and since he has formally admitted the existence of paraphasia in the clinical picture of Broca's aphasia he has removed another factor for the distinction of the two forms. It is argued also against Bastian and Wyllie that in their writings they have repeatedly confused the two types both clinically and pathologically. In the recent controversy this same argument has been hurled from both sides at the opposition.

The type to which the term "total aphasia" ("type Broca-Wernicke," "type mixte") is applied results from lesion in both Wernicke's and Broca's regions, and it comprises severe defects of intelligence and of all the elements of speech. In this connexion it is very necessary to

point out that in deducing localization from cases where there have been multiple lesions involving the speech-centres, one of which exceeds the others greatly in magnitude, it has been almost invariably the custom of writers to attribute the speech-defects to the larger lesion, to the neglect of the smaller lesions. The recorded cases of aphasia in which recovery has occurred after extensive lesions, and those in which the aphasia resulting from small lesions has remained permanent, make the fallacy of such deductions obvious; and further, the extent of a lesion small to the unaided eye can only be correctly determined by the microscopic examinations of serial sections. For example, many cases have been used as cases of pure Broca's aphasia without hesitation by eminent authorities in which small lesions have admittedly existed in Wernicke's zone.

The above considerations will give the reader some idea of the necessity for the reconsideration of the whole subject of aphasia which Pierre Marie demanded in 1906. The whole subject was complicated, confused, and too much beset with schemes and diagrams. There was no working hypothesis that was universally and easily applicable to the clinical varieties of aphasia; no classification of the clinical varieties that did not entail confusion. The very foundations of the subject were necessarily hypothetical, the ideas of the registration of speech-memories in separate centres of the cerebral cortex admitted of reconsideration, while the pathological basis of the subject demanded fresh data obtained by incontrovertible methods of research.

Pierre Marie's [44] attitude is that of a destroyer and reformer. His desire is to sweep away the majority of previously existing ideas and theories upon the subject of aphasia. He challenges the conjecture that speech-memories are registered in the cerebral cortex as "visual word"-memories, "auditory word"-memories, and "kinæsthetic word"-memories. He denies the existence of a visual word-centre, and indeed of any centre for visual speech situated in the angular gyrus or supra-marginal convolution. He denies the narrow localization of Wernicke's centre in the posterior two-thirds of the left second temporal convolution. He denies that the left third frontal gyrus has any function whatever in connexion with the speech-mechanism. He admits of no distinction between the so-called "motor" and "sensory" cortical aphasias, and he rejects these terms as being unwarrantable and useless. He argues that there is only one speech-centre diffusely localized in the left temporo-parietal lobe, that this centre is a region of intelligence specialized for language, not a centre of sensory images ("Une zone

intellectuelle spécialisée, pas un centre sensoriel d'images"). The so-called "word-blindness" and "word-deafness" are defects of the special intelligence of speech, and are not due to the loss of function in centres in which sensory images of words are represented. He contends that there is but one variety of aphasia from a lesion of the cerebral cortex, and that this corresponds with the aphasia of Wernicke which results from destruction of the region of intelligence specialized for language and situated in Wernicke's zone; and that, according to the degree of depression of function produced by the lesion, the special functions of the region are lost in the reverse order of their depth of impression. This aphasia must be sharply distinguished from the conditions having their origin in subcortical lesions—namely, "anarthria" and "pure alexia." The classical aphasia of Broca is a combination of aphasia with anarthria, and is the result of a double lesion, the one situated in the region of Wernicke, determining the aphasia, and the second lesion within a quadrilateral area around the lenticular nucleus determining the anarthria. The limits of the quadrilateral region, a lesion of which is productive of anarthria, are, anteriorly and posteriorly, vertical frontal planes level with the anterior and posterior limiting sulci of the insula respectively. Its outer limit is the surface of the insula, its inner limit the wall of the lateral ventricle. Above this the quadrilateral is prolonged towards the overlying gyri of the convexity, while below it is lost in the subthalamic region. The lesions productive of anarthria are usually situated in the upper two-thirds of this quadrilateral. The quadrilateral is in connexion with the third frontal convolution in front, but contains no part of the latter, whilst behind it is in connexion with Wernicke's zone by means of the temporo-parietal isthmus, a compact band of white matter rounding the posterior marginal sulcus of the insula. Lesions situated in front of this isthmus are productive of anarthria; those situated behind the isthmus are productive of aphasia.

These views of Marie have given rise to much criticism and controversy. Dejerine [14-18] came forward as the champion of the older ideas, and with his school has fought hard, especially for the retention of the left third frontal convolution as the motor speech-centre. This controversy has given birth to much excellent literature upon the subject, among which stand out the delightful monographs of Dejerine for his side, and of François Moutier [61] as the exponent of Marie's views.

On the one hand, certain critics have advised the rejection without

hesitation or further consideration of Marie's doctrine. Among these are Mahaim [42], Debray [13], and Van der Hoeven [61], and a perusal of their comments gives the impression that these are both hasty and without a sound basis. On the other hand, certain authorities have accepted entirely Marie's doctrine, notably Dercum [20], Collins [61], Lasalle-Archambault [33], Dieulafoy [21], and others of the French school. Dercum has recently declared that he has never found Marie's doctrine at fault in practical application. Between these extremes the majority of those who have criticized Marie's position admit the necessity for the revision of the subject, and most of them, while inclining to the conception of the chief speech-centre as a centre of intellect specialized for language, seem unwilling to exclude Broca's area as an unessential part of the mechanism of speech. But within the last few years evidence has been accumulating from another aspect of cerebral function which has the most important bearing upon the interpretation of speech-defects and which is opposed to a part of Marie's doctrine. Liepmann [35] in 1900 brought into prominent notice a state of inability to perform skilled movements with the limbs in the absence of paralysis, loss of coördination, sensory loss or gross intellectual defect. This condition of apraxia has been worked at by many observers since Liepmann, and a review of the subject by S. A. K. Wilson [78] has recently appeared in *Brain*. The term "apraxia" is here confined to the motor apraxia of the classifications, and in the writer's opinion it should always be so confined. The evidence at present available suggests very strongly that apraxia is dependent upon a lesion situated in the region of the first and second frontal convolutions of the left side. That is to say, that the ability to perform certain subjectively purposive movements or movement-complexes on either side of the body is dependent upon the integrity of the posterior parts of the first and second frontal convolutions of the left side. There is reason, too, for thinking that the connexion of these centres with the left side of the body is by means of the anterior fibres of the corpus callosum and the opposite hemisphere, since a localized subcortical lesion involving the callosal fibres from the posterior extremities of the first two frontal gyri on the left side has been associated with left hemiapraxia as the sole symptom. The bearing of this evidence upon the localization of a motor speech-centre in the left third frontal convolution is obvious and striking, for motor aphasia bears the same relation to movements of the muscles concerned in speech as does apraxia to the movements of the limbs. Motor aphasia, pure or cortical, is clearly synonymous with apraxia, pure or cortical, of the movements

concerned in speech, and motor agraphia is apraxia pure and simple. According to this view a series of higher motor centres concerned with certain subjectively purposive movements are situated in the posterior parts of the three frontal gyri and immediately in front of the Rolandic centres (lower motor centres) for the corresponding regions of the body. Lesion of these centres is productive of phenomena of a similar order in the corresponding region of the body—namely, motor aphasia, motor agraphia and apraxia.

At a time, then, when the most determined efforts are being made, and upon grounds which are at least logical, to dismiss the left third frontal gyrus from our conception of the essential speech-mechanism, evidence of a very strong nature is increasingly coming to hand which calls for the retention of Broca's area as a speech-centre, entirely apart from the consideration of local lesions of the brain causing speech-defect. We shall see later on that there is other important evidence which forbids us to accept at the present time the doctrine of Marie in so far as the left third frontal gyrus is concerned. In the writer's opinion it is this part of Marie's scheme that is the weakest. Against this part of his contention alone have substantial arguments been brought, and around it has the controversy waged most hotly.

The writings of von Monakow [59] upon this subject of "diaschisis" are important in connexion with this question. He would make an absolute distinction between those defects of speech which are only seen soon after a lesion has occurred, which are slight in degree and which soon pass off, and those which are well marked and permanent. The early and transitory symptoms he explains by a lowering of functional activity in a more or less distant part of the speech-mechanism, due to the upsetting of balance between the several parts of this mechanism produced by the destruction of one of the integral parts by the lesion. This phenomenon he calls "diaschisis." It must not be forgotten that the nervous system acts as a whole, and not in terms of its individual elements. Similarly the speech-mechanism acts as a whole, and not in terms of its hypothetical centres. For example, von Monakow would explain the slight and transient alexia which so commonly occurs in cases of Broca's aphasia as the result of damage to the speech-mechanism as a whole, causing lowering of function and temporary loss of the least impressed function—namely, visual speech. Marie [44] takes an opposite view, insisting with truth that the alexia, agraphia and amnesia occurring in cases which have been claimed and used as classical cases of Broca's type have been of

considerable degree and often not transient. Every gradation from this to Wernicke's type occurs, and for Marie such symptoms are proof positive of the existence of lesions of Wernicke's zone which he insists are always present in cases of Broca's aphasia. In connexion with another part of the subject, however, Marie seems to make use of von Monakow's explanation when he says that alexia is best explained by a lowering of function in the general centres of vision.

The theory of Bernheim, of Nancy [3], put forward in 1907 as a general conception of the whole subject of aphasia, stands alone and widely apart both from the older hypotheses and from the doctrines of Marie. This theory recalls the ancient hypothesis of Bouillard, who assumed that the prefrontal lobes of both hemispheres were the all-important centres for speech. Space does not admit of the discussion of this peculiar theory, the principles of which are as follows: All aphasias are subcortical in origin and result from lesions of transmission paths. The brain, as the organ of thought, produces internal speech. There are no centres for auditory word-memories nor for visual word-memories: cortical sensory centres for primary perception of impressions alone exist. The memory-images are called up in the psychic sphere (frontal lobe), as are all the phenomena of consciousness. There are no kinæsthetic centres for articulation or for writing. The coördination of movements for articulation and for writing occurs in the nuclei of the medulla and of the cervical region of the spinal cord respectively, which have become automatically associated by habit. Broca's aphasia may be the result of the severance of the prefrontal projection. "In reality," he says, "Broca's aphasia has not been localized, for the good reason that it is not localizable." It is important to notice in connexion with Bernheim's theory that he is the only writer who has laid stress upon the possibility of isolation of the prefrontal region from its connexions accounting for the phenomena of defective intelligence, &c., which are shown by cases of Broca's aphasia. It appears to the writer of the present article that this possibly important point has not been sufficiently considered by authors in connexion with the symptomatology of lesions of the left third frontal gyrus and of the posterior parts of the other frontal gyri.

Following this brief survey of the question as it stands at present, it is necessary to consider in greater detail the chief points in Marie's doctrine, with the arguments for and against, and to discover whether it is possible at the present time to arrive at legitimate conclusions concerning his chief contentions. Whilst considering this subject it is

important to keep certain anatomical facts in view. The lesions chiefly concerned in the production of aphasia are vascular in origin, and as such rarely, if ever, are strictly limited to, and involve the whole of, a region of which the functional localization is attempted. Again, vascular disease is a general disease affecting many of the cerebral arteries, and its result is, in most cases, multiple lesions; isolated lesions rarely. There is hardly a case in the whole records of the subject against which these arguments cannot be raised: "the lesion extended beyond the region in question," or "other lesions were present in other regions," and therefore the cases are inconclusive and arguments cannot be logically based upon them. Such cases, which form the bulk of the literature of aphasia, though individually inconclusive, are collectively of such weight as cannot be lightly disregarded.

The term "cortical lesion," as applied to the local lesions productive of aphasia, means a lesion of the convolution as a whole, and the limit of a cortical lesion is a line drawn tangential to the deepest grey matter of the sulcus bordering the convolution; everything deep to this line is subcortical. It has already been pointed out that lesions reaching the surface of the brain are almost always both cortical and subcortical, and usually much more subcortical than cortical.

The Question of the Left Third Frontal Convolution.

Destruction of a limited area of the brain does not always produce the usual symptoms, and these may be completely absent. For example, cases are on record in which wide destruction of the Rolandic gyri, of the Rolandic projection, and of the internal capsule occurred, and in which there was no hemiplegia. However inadequate the theory of transference and of "gaucherie" may be, and however inexplicable the occurrence is, yet the fact remains that such events do occur in rare instances, and they do not absolutely prove that the region destroyed has not the function usually attributed to it. It must be admitted, however, that an unusual number of cases in which the left third frontal convolution has been destroyed, without the appearance of aphasia, have been recorded, considering the rarity of similar occurrences in other regions of the brain.

The grounds upon which Marie bases his exclusion of the left third frontal gyrus from the essential speech-mechanism are (1) that the collective evidence of all the cases which have been reported in the literature of the subject is strongly against the hitherto accepted

theory of a motor centre for speech in Broca's region; (2) that in his own very wide experience he has never seen a case which, on careful examination, supported this view; and (3) he has another explanation and localization for the "motor" speech-defect.

The collective evidence of the literature is remarkable, and is summarized thus by Moutier [61]: Between the years 1861 and 1906, 304 cases with autopsy have been published bearing upon this subject. Of these cases 201 are not really of value, since in twenty-six cases the clinical or the pathological records were so incomplete as to give the reader no conviction as to the nature of the case, and in the remaining 175 cases the lesions were too vast for accurate localization.

There remain, then, 108 cases in which the lesions were localized, and the records sufficient for purposes of argument. In nineteen only of these cases did the facts appear to support Broca's theory, and in eleven of these the lesion was confined to the subcortex; in eight only was the cortex of the left third frontal gyrus affected.

The remaining eighty-four cases were entirely against the classical theory; in fifty-seven the left third frontal gyrus was intact, and motor aphasia was present. In twenty-seven cases this convolution was destroyed and there was no aphasia present, and in two of the latter cases the left third frontal gyrus was destroyed upon both sides.

According to Moutier, a careful examination of the nineteen apparently favourable cases shows that even these cases are the reverse of conclusive; in some there were multiple lesions, in one there was an extensive lesion of the right temporal lobe. Complete alexia was present, and there is no mention as to whether the patient was left-handed or not. He sums up the analysis with the words "there does not exist in medical literature one observation of Broca's aphasia in which a single lesion, strictly localized to the foot of the left third frontal gyrus, has been proved to exist upon autopsy."

This evidence would seem to be overwhelming in favour of Marie's doctrine; but it is to be remembered that it is the summary and conclusion of a worker convinced of the truth of the new doctrine, and who gives no quarter to possibly fallacious cases that stand against him. He has, however, excluded no case without logical and detailed reasons.

It is interesting in this connexion to refer to the analysis of 104 cases of aphasia, with autopsy made by Frankel and Onuf in 1899. They met with four cases only which seemed to confirm Broca's localization, but they examined five cases of Broca's aphasia in which the left third frontal convolution was absolutely intact, and in which the afferent and efferent fibres connected therewith showed no traces of degeneration.

Two cases of Burckhardt's [11] are peculiar in the literature, and are worthy of note. In the one case this surgeon removed 5 gm. of the grey matter from the foot of the first and of the second left temporal gyri, no word-deafness resulting. Eight months later he resected the cap and foot of the left third frontal gyrus, and no aphasia resulted. In the second case, Burckhardt removed part of the left supra-marginal gyrus; subsequently he performed two destructive operations upon Wernicke's zone, and finally resected the cap of the left third frontal gyrus. At no period did this patient present any speech-defect. These patients were dements, and the operations were undertaken with the idea of relieving verbal hallucinations and logorrhœa.

The fact that an observer so eminent and of so wide experience as Pierre Marie states without reserve that no case has come under his personal observation which confirms the localization of a motor speech-centre in the left third frontal gyrus is in itself of great weight, and it necessitates a careful reconsideration of opinions and the recording of fresh observations, with the exclusion of every possible fallacy on the part of all workers upon this subject. Marie was by no means the first to call the classical localization in question. Twenty years ago Allen Starr [76] found it impossible to come to any determination upon the localization of Broca's centre, since no one in America had observed a case with a strictly localized lesion. From this time onwards many authors have drawn attention to the poverty of the evidence for this localization, though none has formally opposed it. Authorities upon cerebral surgery seem to be in universal accord in regarding Broca's aphasia as a most untrustworthy localizing sign.

The localization of the "motor" phenomena of Broca's aphasia or of "anarthria" in a new region, that of the quadrilateral, is admittedly the weakest part of Marie's argument. He is unable to express a definite opinion as to which are the essential structures in the quadrilateral, of which disturbance gives rise to "anarthria," nor does he attempt any physiological explanation. And further, it has been proved beyond doubt by Madame Dejerine that the projection-fibres from the cap and foot of the third frontal convolution enter the quadrilateral, and are contained in that part of the quadrilateral, lesion of which most frequently produces "anarthria." Madame Dejerine [19] further shows that a lesion of the quadrilateral opposite the upper end of, or above the anterior convolution of the insula, must isolate the third frontal gyrus by section of its projection-fibres, and also that the foot of the corona radiata enters the quadrilateral. The question of the quadrilateral and its localization will be discussed in detail later.

Dejerine [16], who leads the opposition against the doctrines of Marie, commences his argument in the following words: "It is at the present time undeniable that it is difficult to defend with anatomical proofs the localization of articulate speech strictly and uniquely in the posterior third of the third frontal convolution. This localization is possible, but it has not been proved beyond doubt. But though future researches may necessitate the extension of the localization of articulate speech to parts immediately neighbouring upon the foot of the third frontal gyrus, one thing is certain, and it is that there is in this zone a region, a lesion of which determines motor aphasia, and it does so in the absence of any alteration of the temporal lobe." He then puts forward two new cases which he has investigated by the method of serial microscopic sections. In the one case the lesion involved the anterior two-thirds of Broca's convolution, the orbital part, the cap and to a slight extent the foot of the convolution being affected. It implicated the underlying white substance of F3 and F2 and the foot of the anterior segment of the corona radiata. It involved slightly the cortex of the anterior marginal sulcus of the insula and the anterior fifth of the external capsule. The clinical aspect in this case was typical of Broca's aphasia. Most of the speech-defects had disappeared six months after the occurrence of the lesion, and this recovery Dejerine attributes to the relative escape of the foot of the third frontal convolution.

In the second case the lesion occupied the cap of F3 and the adjacent portion of F2. The "quadrilateral" and Wernicke's region were intact. Lesions, however, were present in the right hemisphere: (1) In the paracentral region, (2) in the middle portion of the first limbic gyrus, (3) in the second frontal gyrus, and (4) in the knee of the corpus callosum. The clinical aspect of this case was again typical of Broca's aphasia, and the condition remained permanent.

Dejerine considers that these cases are conclusive of the presence of a motor speech-centre in the region of F3. Marie contends that the first case was one of temporary anarthria from lesion of the anterior part of the quadrilateral (the anterior gyrus of insula and anterior fifth of the external capsule were involved). In his experience, cases of true Broca's aphasia never recover as did this case. To the second case he objects on the ground of multiple lesions involving general defect of intelligence. Against both cases he urges that he has never seen a case of true Broca's aphasia without accompanying hemiplegia.

Dejerine points out that in all cases which have been brought forward by Marie [47], Moutier [51-55], Souques [71-75] and others of this

school, as cases of Broca's aphasia from combined lesion of Wernicke's zone and of the quadrilateral, the degree of word-blindness and of word-deafness that has been present has been so great as is only met with in cases of sensory aphasia or total aphasia, and that from the severity of word-blindness and word-deafness present their cases must be excluded as arguments bearing upon the classical aphasia of Broca. Every case that these authors have cited in which there has been no lesion of the left third frontal gyrus with motor aphasia and in which lesions have been present in Wernicke's zone and in the quadrilateral, according to Dejerine are cases either of sensory aphasia from the lesion of Wernicke's zone, or cases of total aphasia from an additional destruction of the projection and connexions of the left third frontal gyrus which are contained in the anterior and upper part of the quadrilateral.

Dejerine [16] explains the cases in which Broca's convolution has been destroyed and in which no aphasia has been present on the ground of possible dominance of the right hemisphere, ambidexterity, compensation by surrounding regions and compensation by the right hemisphere.

Von Monakow [60] firmly supports the classical localization of Broca, and in conjunction with Ladame [30] has recently published an important case confirming his opinion.

A careful and open-minded consideration of the evidence and arguments that have been brought forward upon both sides of this question and of the evidence gathered from recent studies upon apraxia leave us with a firm conviction in favour of the retention of the left third frontal gyrus as an important speech-centre upon the outgoing side.

For the motor speech-centre it is a question of choice between Broca's centre, our conceptions of which are at least intelligible, and an unlocalized and functionally unexplained motor mechanism situated somewhere in the vast area of the quadrilateral. And since the quadrilateral has been proved beyond doubt to contain the projection of F3 and also the foot of the anterior part of the corona radiata, it may be that the "anarthria" ascribed to the lesion of the quadrilateral results in reality from section of the projection and connexions of the cap and foot of the third frontal gyrus.

The Question of the "Quadrilateral" and of "Anarthria."

It matters not that Marie uses the term "anarthria" in a different sense to that in which it has been hitherto employed, for his definition

of it is clear and involves no confusion. It means "loss of, or difficulties with, articulate speech, with complete preservation of internal speech and with the absence of articulatory troubles of a paralytic nature." It is identical then with Broca's "aphemia" and with "pure" or "subcortical" motor aphasia. He uses the term "dysarthria" for articulatory troubles of a paralytic nature. It is, however, difficult to follow Marie when he suggests that anarthria may be the result of a lesion of the quadrilateral in either hemisphere, and that a lesion of the quadrilaterals of both hemispheres is productive of dysarthria (pseudobulbar paralysis), for the latter is surely dependent exclusively upon bilateral lesion of Rolandic centres and their projection-fibres.

The quadrilateral of Marie is a vast region taking up a good proportion of the middle third of the hemisphere. It is bounded externally by the surface of the insula, and internally by the lateral wall of the third ventricle. In front and behind it is bounded by vertical frontal planes at the anterior and posterior limits of the insula respectively. Below it merges into the subthalamic region, and above it is limited by the convolutions of the convexity. It is connected with Wernicke's zone by the temporo-parietal isthmus, the two making up the only localizable speech-region. Lesions of this region behind the isthmus are associated with aphasia, lesions in front of the isthmus with anarthria.

It seems clear that the limits of the quadrilateral laid down by Marie [44] are rather for purposes of forming a convenient working plan in the unsatisfactory state of our knowledge concerning the functions of this region, than to insist upon a dogmatic localization. The argument, therefore, that has been brought against him that his localization is no localization at all seems to be beside the point, for he candidly admits that he is unable to point out which are the essential structures in the quadrilateral lesion of which produces anarthria, and he is unwilling, moreover, to set up any hypothesis as to the function of many of the structures contained in the quadrilateral, nor will he theorize as to the way in which anarthria is produced.

It is quite beside the question to ask Marie (as has been frequently done in the recent controversy) whether he thinks that anarthria can be produced by a lesion of the optic radiation, of the thalamus, of the caudate nucleus and of the internal capsule, though all these structures are contained in the quadrilateral. The real question is concerned with the functions of the cortex of the insula, of the extreme capsule and claustrum, of the external capsule, of the projection of F3 and the foot of the corona radiata, in so much as these occupy the quadrilateral, and of the corpus striatum.

Concerning the function of the insular cortex, extreme capsule and claustrum, little is known. Some authorities who hold by Broca's localization would extend the motor speech-centre on to the anterior gyrus of the insula. The external capsule contains the forward connexions of Wernicke's zone, and if Broca's localization be correct it must contain the connecting path between Wernicke's centre and Broca's centre, and a lesion of it therefore must entail anarthria by separation of the two centres. A lesion of the anterior and upper part of the quadrilateral would be likely to involve the projection-fibres of the third frontal convolution and the foot of the corona radiata, and the resulting anarthria is easily explicable, according to Broca's theory, as pure motor aphasia from isolation of the third frontal gyrus from its downward path.

There remains to be considered, then, the corpus striatum, and to the importance of this structure in the production of anarthria Marie commits himself. He says: "I should say that the lenticulo-striate body represents, either by itself or with its afferent and efferent paths, a mechanism far more important from a motor point of view than does the left third frontal convolution in the production of speech."

Now it is well known that the anatomical connexions of the lenticulo-striate body are with the thalamus, and with Luys' nucleus, that it sends no fibres into the cerebral peduncle, and that it has little or no connexion by fibres to the cerebral cortex directly. The cortical projection-system, though many of its fibres traverse the corpus striatum, sends no fibres which end in, or connect with, the corpus striatum. From these facts the lenticulo-striate as a motor mechanism for one of the highest cerebral functions—that of speech—is in the highest degree improbable.

There are no clinical and pathological records which give any support to the theory that the lenticulo-striate body is intimately concerned with the speech-mechanism, and the same may be said, though less confidently, of the cortex of the insula and the immediately underlying structures—the extreme capsule and the claustrum.

The very arguments that Marie has used against Broca's localization and the supposed function of the left third frontal convolution are applicable, and with greater force, against his localization in the quadrilateral and against the presumed function of those structures in the quadrilateral which possibly may be concerned in the speech-mechanism. It must be remembered that Marie has not committed himself to any

localization of speech-function within the quadrilateral beyond the reference to the lenticulo-striate body given above, nor does he define the lesion within the quadrilateral productive of anarthria more definitely than by stating that it is most common in the upper two-thirds of this region.

Our conclusion must be that Marie has not proved his case for the shifting of the motor speech-function from Broca's region to the quadrilateral, yet the question is one that needs much further investigation before a satisfactory solution can be arrived at.

Is the so-called Broca's Aphasia in reality Wernicke's Aphasia (Word-blindness and Word-deafness) plus Anarthria?

Accepting Marie's definition of anarthria and placing the lesion responsible for this in the region of or beneath the left third frontal convolution, it seems quite clear that Marie is correct in claiming the presence of the double lesion in a great many of the cases that have been included as cases of Broca's aphasia. Dejerine's contention that Marie's cases are examples of total aphasia is identically the same proposition as is Marie's argument that Dejerine's cases are cases of aphasia plus anarthria. The more severe degrees of word-blindness and word-deafness occurring in cases of Broca's aphasia are much more easily explicable, and do not require the stretch of imagination if we accept Marie's teaching that they are the result of a second lesion in Wernicke's zone, than is necessary when we revert to diaschisis and general depression of the speech-functions to aid our conceptions.

It remains for subsequent investigators to show how far a lesion limited to Broca's region is associated with word-blindness and word-deafness. Dejerine [16] admits it to his conception of the results of a lesion of the left third frontal gyrus in slight degree and as a transitory symptom only. Marie [44] excludes it altogether from his definition of anarthria. Recorded cases show every degree, both of severity and permanence, and they give no means of clinical distinction between cases claimed as examples of Broca's aphasia and of Wernicke's aphasia respectively. If we admit a cortical motor centre for speech, forming an integral part of the physiological basis of internal speech, from our knowledge that the majority of individuals are strong "visuals" as regards general intelligence, and strong "auditives" as regards the special intelligence of speech, even allowing for

individual variation and different degrees of education, we can only attribute a very minor importance to the motor speech-centre in internal speech. Therefore we can only admit of slight and transient disturbances, either of intelligence or of receptive speech, as the result of the upset of balance and lowering of the functional capacity of the speech-mechanism as a whole from the damage to its motor centre, and we must agree with Marie that anything beyond such slight and transient disturbances is strong evidence that Wernicke's zone is also involved.

The Nature of the Speech-function of the Cerebral Cortex.

By far the most important part of Marie's doctrine is his conception of the speech-function of the cerebral cortex, and in the opinion of the writer of this paper this conception will receive general acceptance as the working hypothesis of the future. He argues with great weight against the hitherto accepted theory that the elements of speech are registered as sensory images—"visual word" images, "auditory word" images and "kinæsthetic word" images—in the cerebral cortex. The separation of the visual part of speech from the auditory part of speech is erroneous, while the separate localization of these functions in the cerebral cortex rests upon no foundation in fact whatsoever. The forms of the so-called "transcortical aphasia," that have been supposed to result from lesions isolating these centres, exist in theory only and have never been demonstrated by clinico-anatomical methods. The existence of a separate visual speech-centre and an auditory speech-centre rests upon the slenderest facts, and is controverted by every-day experience. The nervous system does not act in terms of its individual elements, but as a whole. The individual elements of the speech-region of the brain have no isolated function; consequently there can be no mapping out of the localization of isolated parts of the speech-function in the cerebral cortex. The speech-function is diffusely localized as one centre in the left temporo-parietal region, and this centre is an intellectual centre specialized for speech, not a sensory centre for the storing of sensory images. Destruction of some of the individual elements of this centre is productive not of loss of isolated parts of the speech-function, but of a general depression of speech-intelligence and of general intelligence which results in the loss of the speech-functions, in reverse order of their depth of impression, according to the severity of the destruction in the

speech-region. For example, a foreign language is lost first, and most by the aphasic, and is regained last and least. The visual speech-function, being less deeply impressed than the auditory speech-function, is much more affected by a slight lesion than is the latter.

The whole of the phenomena of aphasia are much more easily explained on the ground of varying degrees of crippling of a single speech intelligence centre than upon the old hypotheses.

The consideration of the phenomena of aphasia as defects of intelligence is a very old idea, and it was held by Trousseau. It has never been eradicated from current ideas on the subject, as the introduction of ideational centres into many of the "schemes" of the speech-functions shows. It has been held by Marie for many years, and his experience has convicted him of its truth. At the present time many authorities accept this view. Grasset [25] accepts it, von Monakow [60] inclines to it, and Dieulafoy [21], in the fifteenth edition of his "Manuel de Pathologie Interne," has adopted the entire doctrine of Marie in his description of aphasia.

The origin of the theory of "sensory" aphasia rests with Wernicke. Following upon the work of Meynert, Wernicke [44] traced what he considered to be the central acoustic path to the region of the first temporal gyrus and the insula. This anatomical discovery led him to theorize the existence of a centre for the comprehension of spoken language in the first temporal gyrus, and by an extraordinary coincidence almost at the same moment he made the clinical discovery of that form of aphasia which has ever since borne his name. Within a short time he was able to examine the brains of two patients with this defect, and in both of them there was a lesion of the left temporo-parietal region. This apparently emphatic confirmation of his idea led Wernicke to put forward his theory of "sensory" aphasia. An anatomical investigation upon a sensory path, a postulated centre for the comprehension of spoken language, a brilliant clinical discovery, and two autopsies founded "sensory" aphasia. This doctrine was universally accepted within a short time, and it was subsequently amplified and extended as the result of reasoning and of psycho-physiological methods of introspection, and not from clinico-anatomical observations. It was thus that the conceptions of separate centres registering separate sensory images, with complicated inter-connections, arose, and from these were deduced in theory the nature of the speech-defects which ought to result from destruction of the various separate parts of this complicated speech mechanism.

The theory of sensory images is contradicted by clinical facts. Let us take, for example, a case of partial word-deafness. The patient is able to comprehend perfectly, and to respond to simple sentences and commands. Make these sentences and commands more and more complicated, and a point will be reached when he neither understands nor responds. Put these same complicated sentences and commands to him in a dissociated form as a series of simple phrases and orders, and he again understands perfectly and obeys. Here there is obviously no loss of auditory word-memories, for he understands perfectly when the sentences and commands are simple. But he does not comprehend when the same commands are mixed together. This is obviously due to a lowering of the function of the speech-centre as a whole, and not to the loss of one of its parts endowed with a specific and localized function. Tests of this nature can be applied to every part of the speech-function with the same result—that of controverting the theory of sensory images.

The vocabulary in aphasia, both upon the receptive and upon the outgoing side, shows no gap compatible with the destruction of regions in which definite word-memories are registered. Such patients retain words in the order of the depth with which they are impressed and the extent to which they are used subconsciously.

If we abandon the theory of sensory images, and accept Marie's conception of a single centre of intellect specialized for language, situated, broadly speaking, in the region of the angular gyrus, supramarginal gyrus, and the posterior parts of the two upper temporal gyri of the left side, a multitude of the phenomena presented by patients with aphasia become easily intelligible, which were inexplicable or admitted only of highly forced explanations upon the old theory. The absence of a single recorded case of isolated cortical alexia, previously explained on the grounds that the visual word-centre and the auditory word-centre were so close together that a lesion of the former necessarily involved the latter, is now explained by the non-existence of a separate visual word-centre. The constant association of alexia with word-deafness and its persistence over the word-deafness when partial recovery occurs, which is really inexplicable by the old theory, is a strict consequence of the new theory; for the visual speech-function of the single centre is a less impressed function than the auditory speech-function, and is therefore the first to be lost and the last to be regained when the speech-centre is damaged. Again, the patient whose voluntary speech is reduced to the production of two or three words only, and who,

when started either with the voice or by accompanying music, can sing a song of many verses with perfect articulation, is absolutely contradictory to the theory of sensory images. Such a case is a proof that the images are not lost, but that the speech-function as a whole is depressed, yet can be temporarily roused into a condition of higher activity by the emotional elements of sound and rhythm. Did space permit, it would be easy to put forward many phenomena occurring in these patients which can only be adequately explained by the new theory, but the above are sufficient in this place.

Anatomical proofs, either of the separate existence of a "visual word-" and an "auditory word"-centre, or of the local registration of sensory images in the speech-centre, or, indeed, of any functional localization in the general speech-centre, may be said to be absolutely wanting if the single case of Dejerine, already referred to, be excepted.

It is many years since Dejerine [8] suggested the localization of a visual word-centre in the angular gyrus, and there has been no anatomical confirmation of his idea, and there have been many records which disprove it. It may, with justice, be said that narrow localization within the speech-area by the combination of clinical and anatomical methods is impossible from the nature of the lesions which we have to interpret, since these are vascular lesions, and as such are both cortical and subcortical, and generally much more subcortical than cortical. The subcortical part of the lesion isolates much more of the cortex of the speech-centre than the lesion appears to involve. For this reason we must discount, to a certain extent, the value of recorded cases of local lesions in the speech-centre (second temporal gyrus).

Strict and narrow localization of function in the cerebral cortex is, in all probability, an erroneous idea. Perhaps it is relatively true as regards the cuneus, and, to a less extent, as regards the Rolandic cortex. In the cuneus there is strictly local representation of the visual field as regards the quadrants, but while an extensive lesion of the region representing the quadrant produces a complete quadrantic hemianopia, a small lesion does not cause complete loss of part of the quadrant, but produces a partial loss over the whole of the quadrant. That is to say, each quadrant is represented as a whole, and partial damage impairs the function of the quadrant as a whole. The absence of strict localization in the Rolandic region was pointed out by Hughlings Jackson many years ago in his description of "representation in compound proportion." The paralysis that results from a local lesion of the motor cortex is never a strict monoplegia, but it is in reality a partial hemiplegia.

Marie and Guillain [50], in their work upon the pyramidal tract, pointed out, in antagonism to existing beliefs, that the result of small local lesions in the motor region of the internal capsule was not the production of various monoplegias as was usually taught, but the production of a widely-spread hemiplegia of slight intensity, and the larger the lesion the greater was the intensity of the hemiplegia. Marie deduces from this fact a law which he would extend to the whole of the cerebral cortex. He says: "This law of the production of cerebral hemisyndromes from a lesion of a small portion only of the region which gives rise to such syndromes is applicable throughout the brain. It is thus that a local lesion of that region of the brain which has to do with the perception of common sensation is productive of a hemianæsthesia of all one half of the body. It is thus that a lesion of the cuneus, however small, gives rise always to a hemianopia, and, finally, it is according to this same law that a local lesion of any and of every part of Wernicke's zone gives rise to an aphasia involving every part of the speech-function."

Dejerine is entirely opposed to this "Loi globale" of Marie, and he insists that the nature of the symptoms that result from a local lesion are entirely dependent upon the position of the lesion. In the opinion of the writer of this paper the evidence is strongly upon Marie's side as regards Wernicke's zone and the higher regions of the cerebral cortex, but that at present Dejerine's opinion is justified, so far as the motor centres, and probably also the centres in the posterior parts of frontal gyri, are concerned.

Marie in his doctrine lays great stress upon the intellectual defects shown by patients with aphasia. Aphasia is to him essentially an intellectual defect entailing defects both of the specialized intelligence of speech and of general intelligence in every case. In this opinion he is supported by many eminent authorities, including Grasset and von Monakow. He uses the fact of the presence of marked intellectual defects in all these patients as important evidence of the unvarying presence of a lesion of Wernicke's zone, and as a proof of the unity of aphasia. Cases of aphasia in which intelligence has been said to be perfect have been brought forward by the supporters of Broca's localization, but Marie clearly shows that in these cases the evidence of complete intellectual capacity is quite inadequate, since not even the simplest tests were applied. He has been able to prove beyond doubt the presence of obvious reduction of intelligence among all those under his care. He points out their inability to tell the time by the clock and

to put the hands of the clock at a given hour, and their ineptitude for the simplest mathematical calculations. He instances the common experience of the aphasic musician who is unable to play the music that he formerly knew by heart. He cites the case of a highly skilled cook who became aphasic and who was said to have no defect of intelligence. This man when taken to the kitchen was unable to prepare even the simplest dish. Such defects are not defects of speech-intelligence; they are defects of general intelligence and can be found in every person with aphasia.

The speech-functions of the right hemisphere were held by Bastian to be important, for he considered that speech centres existed in the right hemisphere in similar positions to those of the left hemisphere and with similar function but of lower functional value. Marie is of opinion that Bastian's view has not received sufficient attention. With Moutier he would place a second Wernicke's zone upon the left side, and the function of such a centre as forming part of the general region speech-intelligence and the results of local lesions of this region are readily intelligible upon his doctrine. He states that "anarthria" may result from a lesion of the right quadrilateral in a right-handed person. Marie agrees with the usually accepted ideas regarding compensation and re-education both in surrounding regions and in the opposite hemisphere as explaining the recovery which occurs in aphasia. Such recovery is of slow occurrence. He quite rightly rejects altogether the theory of sudden or rapid transference of the speech-centres as a pure invention which has been made to account for absence of aphasia with a lesion of the left Broca's gyrus in right-handed subjects.

For this same reason he condemns the theory of "gaucherie" in the right-handed person, and he points out the disproportionate number of the cases of lesions of Broca's convolution into which this explanation has been drawn. No such forced explanation, he says, will save Broca's localization. Marie has not recently expressed his opinions as to the bearing of our increasing knowledge of apraxia upon the subject of aphasia, and Moutier, beyond a criticism against the conclusiveness of recorded cases, does not discuss the subject in his monograph. Marie [44] in 1906 explained apraxia as an intellectual defect. He considered that the seat of the lesion responsible for apraxia was somewhere in the speech-region, and that this region was perhaps also a centre for the ideas of highly skilled and conventional acts. From the defect of intelligence produced by the lesion the apraxic patient was unable to perform certain acts. The apraxic subject was, in fact, suffering from

aphasia of low intensity. On the one hand the apraxic was unable to understand the command, and on the other hand the idea of the exact nature of the act demanded of him escaped him. At the present time such an explanation as the above is entirely untenable in the light of the cases of apraxia confined to the left side, and without right hemiplegia, that have been reported.

The doctrine of Marie, in so far as his conceptions of the chief speech-centre are concerned, marks a step in advance in our knowledge of the subject of aphasia, and will probably receive general acceptance. It is more in accord with facts, it is less beset with theory, it is a far better working hypothesis, and it is a sounder basis for future research than are the classical theories.

The retention of Broca's localization at the present time seems imperative from the evidence both on the side of aphasia and apraxia, and it is quite compatible with the rest of Marie's doctrine. In forming our opinion we have to choose, according to either doctrine, a motor speech-mechanism subsidiary to the chief speech-centre. We are offered the left third frontal gyrus and the classical ideas of its function by Dejerine. We are shown the quadrilateral by Marie, and we are asked to accept some structure as yet not definitely pointed out within this area. At the same time we are not sure that these alternative localizations are distinct, for it is possible that the same structures are involved in the lesions in either localization which are productive of aphasia.

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A Text-book of Psychiatry for Physicians and Students. By LEONARDO BIANCHI, M.D. Authorized translation from the Italian by JAMES H. MACDONALD, M.B., Ch.B. Glasg. London: Baillière, Tindall and Cox, 1906.

AT a time like the present, when the study of psychiatry is being prosecuted both in Europe and in America with a vigour hitherto unprecedented, the appearance of an English translation of the work of the famous Italian neurologist and psychiatrist should be especially welcome.

The volume, which contains upwards of 900 pages, is admirably translated, and, even in its most abstruse passages, is as intelligible as if it had been originally composed in the English language. The translator, in fact, cannot be too warmly congratulated on the successful performance of an exceptionally difficult task.

That the book is already several years out of date is of little import to English psychiatrists, as its chief value depends on the fact that it contains the reasoned opinions of an eminent representative of the Italian school of psychiatry. It is, however, to be regretted from the aspect of the student that no attempt has been made by the author and the translator to refer to, if not to incorporate, the important results of recent research.

The volume is divided into three parts, which deal respectively with the anatomy and physiology of the cerebrum, the physiopathology of mind, and the subject of mental disease.

The first part calls for little remark. The anatomy of the cerebrum is lucidly described, but the account contains no reference to the important histological researches of the past eight or ten years. The localization of the visual area, for example, dates from the work of Henschen, Vialet, Dejerine, Brissaud, &c.; and the account of the differential structure of the cortex generally from the publications of Hammarberg and Schlapp, and the earlier researches of Cajal and Flechsig. It is therefore hardly necessary to remark that the description of the physiology of the cerebral mantle, which, considering the date at which it was evidently written, is excellent, contains no reference to the experimental work of Sherrington and Grünbaum.

The section on the physiology of language is exceptionally good, and is worthy of careful study. The views of the author as to existence of centres for percepts and even for abstract conceptions, which are distinct from those for the images of words, are clearly expressed and ably discussed. The chief argument adduced in favour of a concept centre is sufficiently expressed in the following sentence: "If, then, the word can be learned and pronounced completely void of conceptive significance, as in the case of infants and idiots, and if it be true that even the dements, when their intelligence is resolved into its elements, can pronounce many words deprived of their signification and devoid of any conceptive content whatsoever, inasmuch as the concepts themselves are resolved into their imaginative elements, many of which have been lost with the destruction of the mind, one can only conclude that the words which clothe the conceptions have a seat of distinct formation, and that whilst in one case the conceptions may disappear and the words remain, in another the latter may disappear and the former remain in a state of latency." It may, however, be remarked that the truth that concepts may disappear and the words remain does not make it necessary to predicate a centre for concepts. It can equally be asserted that words serve merely as symbols on which, with a normally acting cerebrum, it is possible to integrate voluntarily conceptions by complex processes of association, and that a repetition of these processes, similar in fact but different in detail, is necessary whenever an individual conception is evolved. Indeed, it may be argued with regard to both percepts and concepts that just as a percept is symbolized by a word which is meaningless, apart from the arousing of sensory memorial images of various kinds, so a concept is symbolized by a word, but is only integrated by the voluntary performance of still more complex processes of cerebral association.

The first part of the volume concludes with a lengthy criticism of the views of Flechsig, with especial reference to the functions of the anterior and posterior centres of association; and the author repeats and elaborates his well-known views on the functions of the frontal lobes. The following paragraph, which summarizes Bianchi's conception of the functions of the cerebral mantle, is worthy of quotation: "In concluding this first part of the work, one can symbolize the cerebral mantle as a state with a representative system—a parliament and government. The mantellar parliament would be constituted by the perceptive zones, each of which furnishes to the central government the product of its own labour, accomplished with material prepared by the force of Nature from the primary receptive and ganglionic neurones throughout the course of

successive years. The central government would be represented by the frontal lobes, which, utilizing the products elaborated by all the members of parliament, direct in their turn the legislative work to the advantage of the people, who, in the nervous system, are represented by all those millions of neurones, scattered or grouped together in the sensory periphery of the body, in the ganglia, in the spinal medulla, in the posterior, mid and intermediate brain, their products being transmitted to the perceptive centres, which merely transform them and transmit them in their turn to the government, whose synthetic, directive, and regulative influence displays itself in the adaptation of the individual to the new conditions of existence and in the extension of the individual and social environment."

The first chapter of Part II. deals with the physiopathology of perception. The author holds a rather broad view of sensation, defining it as "the notice of the modification induced by external agents (stimulus)." He lays emphasis on the progressive nature of the process of perception, in the sense that it consists "in the perception of differences—that is to say, in the capacity to gather new features and new relations in the external world, as well as to furnish new and useful combinations with the perceptions and the secondary products of these that have accumulated in the mind." It is, however, difficult to see in what way this conception of perception differs from the Continental notion of apperception, and the author evidently finds a difficulty in making the distinction, as he somewhat obscurely defines apperception as "a more acute perception, rendered possible by a more concentrated attention, and promising results of much greater advantage for the evolution of the mind (*vide* the following chapter on attention). It is accordingly the highest expression of the perceptive progressiveness." After a reference to and discussion of the simpler disorders of perception, illusion and hallucination are clearly described and fully illustrated. When considering the question of the source of hallucinations, he supports the hypothesis of Tamburini. "Hallucinations are phenomena whose organic substratum is in every case the respective sensory area of the cortex. The more or less evident alterations that we find there, when we have the opportunity of making sections of the brain of a subject of hallucination, are an indisputable proof of the truth of this doctrine." As an example, he describes in full a case of meningo-cerebral gumma, and remarks: "Such a finding explains all the symptoms; the nodules on the Rolandic zone were the cause of the Jacksonian attacks; the diffusion over all the somæsthetic zone gives the reason of the profound

disturbances of kinæsthesis and of the hypochondriacal condition; the diffusion backwards on the occipital lobe and the projection upon the first temporal convolution are to be regarded as the immediate cause of the auditory and visual hallucinations." It may be remarked here, in view of the author's opinions on "sensory insanity," that he applies the term "cortical sensory centres" to almost the whole mantle of the cerebrum behind and below the precentral sulcus, and not simply to the several sensory or projection spheres which are now generally recognized. In other words, Bianchi employs the word "sensory" as the equivalent of "perceptive and sensory."

The next chapter deals lucidly and fully with the subject of attention. "Attention is a psychic fact interposed between perception or its ideative equivalent and the reaction which that tends to provoke." "It is not the will, as some have believed, nor is it apperception. It contributes to the process of apperception and to the direction of the will in the mechanism of conscious reaction." Voluntary and involuntary attention are discussed and the motor phenomena associated with the act of attention are referred to. The author then expresses his views, which are in general agreement with those of Bastian, Ferrier, and Wundt, as to the cerebral seat of attention. "The frontal lobe is the organ of attention, in so far as it is the one on which we bring together the whole intellectual and emotional patrimony of the personality, and in so far as it is the centre for summoning up separate images, of which the equivalents or duplicates are sent from the sensory zones to the frontal lobes for physiological fusion with others and for the formation of mental products of a more elevated order." The chapter concludes with an account of the various disorders of attention.

Memory is next discussed. The author defends his view that concrete images are the products of the combination of elementary images, and that they are conserved in the various perceptive spheres or cortical sensory centres, and not in a special organ of apperception (Wundt) situated in the frontal lobes. When discussing the reproduction of images, after he has distinguished between evocation and reproduction, he describes the former function as voluntary and involuntary, and he concludes that involuntary evocation occurs in each of the respective perceptive (and memorial) spheres. With regard to voluntary evocation he remarks: "Every time, therefore, that a thought arises there comes an order from the frontal lobe to the separate organs that form and conserve the concrete images of objects and of words to bring forward all their products of concrete images for the development of the concept, of

which these are elementary components." As, however, the author states that whilst reproduction succeeds evocation, it sometimes arises independently of it, one can only conclude that he wishes it to be inferred that concrete images may arise spontaneously, that they may be evoked by any other local psychic components, and finally, that they may be evoked by the act of volition through the agency of the frontal lobes.

This is not the place for a discussion as to whether Bianchi's views with regard to the existence of definite centres for perceptions and their conservation are correct; but it may be remarked that he gives these centres a location distinct from that of their respective word-images. In this case a word-image could directly evoke its fully developed percept in the respective centre of conservation of this, and would not, as is the opinion of the writer of this review, serve simply as a symbol on which a percept might be integrated from simple sensory memorial images by a complex process of association. Whilst the writer would regard both the concept and the percept as psychological generalizations which indicate different grades of cerebral association, Bianchi appears to consider them distinct entities with cerebral centres of conservation, distinct from the regions which serve as the physical bases of word-images.

The chapter concludes with a lengthy and interesting description of the various disorders of memory.

A chapter on ideation follows. This contains an interesting account of the evolution of language and of the laws of association. It may be remarked, with regard to the author's views on this subject, that he includes under the anatomical substratum of thought—(1) centres for the conservation of elementary images and of concrete images (percepts) which occupy the "sensory areas of the cerebral mantle"; (2) the region of central government and the centre for concepts, which occupy the frontal lobes, and (3) all the zone of language.

Disorders of thought are then referred to, and "deliria" (delusions) are elaborately discussed, classified and illustrated. The chapter ends with a short account, under the doubtfully well-chosen title of "mental confusion," of such symptoms as dyslogia, incoherence, echolalia, coprolalia, &c.

The second part of the volume concludes with chapters on the emotions and sentiments, the will and consciousness.

The different theories with regard to the emotions are discussed at great length, and the author strongly supports the view that emotion is

a psychic phenomenon accompanied by, but distinct from, the various somatic phenomena which constitute its physical expression. The will, on the other hand, is rapidly dismissed. It is "nothing more than the conscious resolution of the motor tendencies of the intellectual emotional syntheses, and we may figure it to ourselves as a conscious motor potential tending to discharge itself through circuits of various orders from the lowest, which much resemble the inferior and instinctive reflexes, to the highest, which are represented in the actions of the heroes of humanity." Consciousness is defined as "the *ego* differentiated from the world," and is divided into the illuminated and the non-illuminated, or "the unconscious." The author considers that consciousness has its anatomical substratum in the whole cerebral mantle, but its chief centre in the frontal lobes.

Part III., which deals with the subject of mental disease, occupies the remaining 500 pages of the volume.

The first chapter contains an extremely full and lucid description of methods of clinical examination. The classification of mental affections is then considered, and the various possible bases of classification are critically discussed. The author divides mental affections into three great groups: (1) Those represented essentially by evolutionary psycho-cerebral defect; (2) all the affections of infective, autotoxic and toxic origin developing in individuals regularly evolved; and (3) all the affections with an organic substratum, localized or diffuse, in the central organ of mind.

Whilst the author does not consider the groups to be necessarily mutually exclusive, *e.g.*, the incompletely evolved may be subject to the disorders of the second group, it is quite clear that he really intends the mental affections of the second group to be regarded as of infective, autotoxic or toxic origin.

As regards the question of dementia, he remarks: "All the mental affections of the second group and many of the first, after a long duration, and when they do not recover, give rise to phenomena characteristic of mental decadence, which frequently is slowly progressive." A short description of secondary dementia is inserted as an appendix to the account of the second group.

In group I. are included phrenasthenias (*i.e.*, idiocy and imbecility), paraphrenias (*i.e.*, eccentricity), delinquency (*i.e.*, criminality), epileptic insanity, hysterical insanity, paranoia, fixed ideas and obsessions, neurasthenia, and sexual psychopathies. The description of this group occupies 240 pages, or nearly one-half of the section on mental diseases.

The subject of idiocy and imbecility is treated solely from the aspect of general symptomatology, though the author accepts groups of non-epileptic and non-paralytic, epileptic and paralytic (and choreic), and myxœdematous cases respectively, and gives a short separate description of cretinism. The clinical account is good, but the anatomico-pathological portion is less satisfactory.

A short but interesting chapter on eccentricity is followed by a more lengthy one on delinquency. The author divides delinquents into four classes—moral imbeciles, born criminals, occasional and passional delinquents, and cases of criminal or moral insanity. The first class he includes under the phrenasthenias; and the third and fourth are referred to in the succeeding chapters on the first group of mental affections. The description of the second class is very good, but does not call for remark.

The account of epileptic insanity is interesting, but the chief detail gathered from its perusal is the general resemblance which exists between the descriptions of the epileptic and of the criminal.

An excellent chapter on hysterical insanity follows. It contains a number of interesting cases. The author sums up his conception of hysterical insanity in the remark: "I would say that the hysterical subject has an infantile mind, but sexually is adult and anomalous."

The chapter on paranoia is one of the best in the volume. The author defines this psychosis as "constituted essentially of a disturbance that is mainly intellectual, and through which the personality undergoes a slow transformation in its relations with the outside world.' His views will perhaps be most clearly expressed in his own words: "It is certain that when a man accepts as a reality a false product of his own thought, the very fact of his doing so compels us to admit that there is a great anomaly in the formative process of thought and a serious defect of judgment; the mechanism of thought is subjugated by the emotions and the sentiments that are morbidly predominant. The abnormal emotive state, for reasons that are quite individual, either alters the process of synthesis in the formation of thought, so that the delirium appears to be primary, and not assisted, at least in the beginning, by sensory disorders, or it alters the perceptive process, giving rise to illusions and hallucinations, and through the abnormal products of the perceptive sphere deliria are formed." . . . "In all the better known and better studied forms of paranoia the emotions are the fundamental and primary fact." . . . "Paranoic deliria are therefore not autochthonous ideas, but of emotional origin." . . . "These three emotions—suspicion, ambition, and love—give the three classic

forms of paranoia: the persecutory, the ambitious or proud, and the erotic." The descriptions of the several types, and also the examples which are given, are excellent; and the psychoses excluded from the group of primary paranoias are mentioned, namely, systematized deliria consequent on other psychoses, all states of confusion and hallucination, and secondary paranoia. It will thus be seen that the author's conception of paranoia accords more with that of foreign than with that of English alienists. In describing this psychosis under mental affections due to cerebral subevolution, Bianchi differs from the majority of alienists, as does the writer of this review. It may be remarked that amongst the types of paranoia are included such forms as religious paranoia, paranoia erotica, and mania for litigation, which the writer regards as symptomatological varieties, due to the special mental configuration of the subjects, rather than as distinct kinds of paranoia. The number of such types might, in fact, be increased almost indefinitely, although all in general symptomatology conform to a standard description.

The chapter on fixed ideas and obsessions is clear and well written, and is followed by a short account of neurasthenia. The latter is defined as "a particular state of the nervous system in which the nervous energy, under whatever form it be displayed, is below the normal minimum relatively to each individual, and below the average of the race to which he belongs," and the author locates it, as regards its physical basis, in the somæsthetic zone of the cortex. The description of the first group concludes with an account of the anomalies of the sexual instinct.

The second group "comprises all the mental affections of infective, autotoxic, and toxic origin developing in individuals regularly evolved." It includes mania, melancholia, maniacal-depressive insanity, sensory insanity, mental confusion, acute and late paranoias, neurasthenic, choreic and luetic insanities, acute delirium, and pellagrous, alcoholic, morphinic, cocainic, chloralic and saturnine insanities. Such critical remarks as appear desirable will be inserted under the several headings.

The author limits the term "mania" to the simple form. As would therefore be expected, he remarks that it is rare, occurring in his experience to the extent of 2—3 per cent. of admissions, and is more common in women than in men. He describes it as mild, typical and grave. He excludes hallucinations from the symptomatology. The description is excellent, but it is difficult to understand why this psychosis is included under the second rather than under the first group.

The description of "melancholia" agrees generally with that of Kraepelin, and in spite of its position in his classification the author does not adduce any new reasons against its being the most common type of presenile involution of the cerebrum.

"Maniacal-depressive insanity" is dismissed in less than three pages, one of which is occupied by excellent diagrams of periodic mania, periodic melancholia, and the various mixed and "circular" types of recurrent insanity. The author states: "We have nothing to add to the pathology of this form, except that it is rooted in grave psychopathic heredity and in anomalies of the character. The prognosis is grave; mental enfeeblement follows much more slowly than in the pure form." As was remarked in the case of "mania," it is difficult to understand why this general type is placed in the second rather than in the first group.

The chapter on "sensory insanity" requires more lengthy consideration. The term "sensory insanity" is defined as follows: "Under this name are included all those psychopathic states which begin with hallucinations and illusions, no matter whether these sensory disturbances dominate the scene during the whole course of the disease or disappear at the beginning of it, leaving syndromes which it would be a mistake to consider as particular forms of disease, since, in point of fact, they form, with the sensory disturbances, one single whole, clinically and psychologically inseparable." The word sensory, as employed by the author, covers both the sensory and the perceptive centres, which approximately occupy, in his opinion, the somæsthetic area (as described prior to recent researches) and nearly the whole of the cerebral cortex behind and below it. "The almost constant fact" in sensory insanity "is the hallucinatory explosion of the disease. . . ." "In the classification I have separated mental confusion from sensory insanity because, in some cases of the former, in retracing the history of the disease, I have not succeeded in satisfying myself as to the hallucinatory beginning."

The author divides sensory insanity into three classes:—

(1) "Illusional or perceptive insanity." This consists "in an illusory perception of one's own physical person or that of others." It is difficult, from the examples cited, to see why these cases are not included under fixed ideas and obsessions.

(2) "Subjects of hallucination of good mental constitution, in whom the hallucinations, being neither intense nor terrifying, but varied, may be repeated for a very long time without disturbing their mental organization and conscious personality." The cases cited would be described by many as examples of delusional insanity, with vivid and varied hallucinations of a persecutory nature.

(3) This includes such syndromes as acute dementia, amentia (Meynert), stupidity, and dementia præcox. The author considers that whatever form mental confusion in such cases may assume, hallucinations, incoherence and deliria (delusions) occur; and, further, that the hallucinations are the cause of the incoherence and deliria.

In the single page devoted to mental confusion the author states that he does not feel warranted in absolutely denying that this may sometimes occur without being preceded by hallucinations and illusions, and this is apparently the reason why it is separately mentioned. It may be presumed that "polyneuritic psychosis" is included by Bianchi under sensory insanity and primary mental confusion, though this is not explicitly stated; whether or not, it is not included, either by name or symptomatology, under "alcoholic insanity."

Space does not permit of any detailed criticism of the author's conception of "sensory insanity," and isolated remarks on it would readily convey an erroneous impression. It may, however, be noted that no satisfactory etiological or pathological basis is presented by the author for the grouping under one heading of such entirely separate types as recoverable intoxication confusions, premature and mature dementias, and the like, all of which commonly, but not necessarily, exhibit illusions and hallucinations as part of their early or acute symptomatology, and some of which, at any rate, can hardly be considered to be always of infective, autotoxic, or toxic origin.

The remaining chapters dealing with the second group, the contents of which have already been enumerated, are short and unimportant. The description of this group concludes with a short appendix on secondary dementia.

In the third group "are included all the affections with an organic substratum, localized or diffuse, in the central organ of mind." It contains chapters on dementia paralytica, and on luetic, senile, post-apoplectic, aphasic and traumatic dementias.

The account of dementia paralytica is similar to that commonly given in text-books and calls for little remark. There is no reference to the age at which the disease occurs, and the juvenile form is not even mentioned. The author considers that dementia paralytica may be provoked by a large number of causes, and that "there is no clinical difference between syphilitic, alcoholic and other non-syphilitic paralytics." Whilst Robertson's researches, and even his employment of serotherapy, are mentioned, there is no reference in the chapter to the work of Mott, which omission in itself is sufficient to demonstrate how much the account is out of date.

A short chapter follows on luetic dementia (syphilitic brain disease).

The chapter on senile dementia excludes "the progressive paralyses of the aged and the psychic alterations consequent upon destructive foci," and is practically a description of second childhood.

The account of post-apoplectic dementia contains nothing of note, but that on aphasic dementia (secondary to cerebral lesions) is excellent. Several good cases of lesion of the visual and auditory word-centres are described. The author's views are summarized in the following sentences: "Thought, in so far as it is a product of synthesis and works logically according to the laws of association, does not exist except in the sensible form of language. If language be suppressed, perception and immediate judgments on images are still possible; not so a train of thoughts or an extended synthesis. . . ." "In ordinary dementia the degenerative process, which involves the textural structure of the whole brain, tends to destroy the elementary components of thought. In aphasic dementia, on the other hand, these last exist integrally, but the processes of synthesis are impeded in so far as these result from the psychological fusion of the elementary components of the word."

It will be noted that, in conformity with the author's acceptance of centres of perception apart from words and their cortical centres, he considers perception possible in the absence of language. It is difficult, however, in view of the above quotation, to understand his postulation of a concept centre apart from language. The writer would rather regard both conception and perception as complex processes of cerebral association which occur through the medium of certain words or word groupings, these being symbolic of concepts and percepts which require integration before such words acquire meaning. From this point of view centres for concepts and percepts do not require postulation, except in so far as the various word-centres serve as physical bases for the different symbols through the medium of which concepts and percepts may be integrated from sensory memorial images by processes of cerebral association.

The book concludes with a short chapter on traumatic dementia.

It is unnecessary to express an opinion concerning a volume which in its several parts exhibits its author as respectively a neurologist, a psychologist and a psychiatrist of the first rank. The book requires to be read to be appreciated, and, although the views expressed are widely different from those accepted by non-Italian schools of psychiatry, the erudition, experience and reputation of the author entitle them to both respect and consideration.

J. S. Bolton.

Publications Recently Received.

[*Notes on a book under this heading do not preclude a subsequent review.*]

Saggio di Anatomia Segmentale. La Metameria Somatica, Nervosa, Cutanea e Muscolare dei Vertebrati. Memoria di G. van Rynberk. Profusely illustrated. Pp. 318. Roma: Tipographia della R Accademia dei Lincei, 1908.

In this monograph the author treats of segmentation in all its aspects. After an introduction, in which he deals with the subject in general, he passes to the evidences of myomeric, neural and skin segmentation as discovered by direct examination. The third part of the work is devoted to a consideration of the results yielded by the morphological, experimental and clinical methods with regard to the segmentation of the nervous system. Every scheme that has been put forward is reproduced and duly considered. The book is a wonderful record of the work that has been done on many different lines towards the elucidation of the question. It will be of value, even to those who are unfamiliar with Italian, on account of its profuse illustrations.

The Origin of Vertebrates. By Walter Holbrook Gaskell, M.D., F.R.S. Pp. 537. London: Longmans, Green and Co., 1908.

Throughout the last ten years Dr. Gaskell has published a series of papers bearing on his view that the verebrates have arisen from an arthropod ancestor. The whole of this work has been put together in the present volume in a most attractive form. Each chapter deals with the consideration of some definite aspect of the question. Should a reader be unfamiliar with the detailed investigations of any part of the subject, he can nevertheless follow the general thread of the argument with the help of the excellent summary which follows each chapter. The book is written in a fascinating manner, clear, simple and concise. The innumerable original observations are marshalled with such skill that, although the book is pre-eminently one for specialists, the unlearned are carried on from point to point without fatigue. The stress laid on the dominance of the nervous system in the upward path of development makes the work peculiarly attractive to all neurologists.

An Introduction to Social Psychology. By WILLIAM MCDOUGALL, M.A., M.Sc., Wilde Reader in Mental Philosophy in the University of Oxford. Pp. 355. London: Methuen, 1908.

Psychology is the recognized basis of all social sciences. But as ethics, economics, political science, and the philosophy of history were studied before the birth of modern psychology, they are based on a priori dogmas now recognized to be false. In this interesting book the author reviews modern knowledge on primary instincts, emotions, sentiments and volition. The second half of the book is devoted to the consideration of the part played by primary instincts in the life of societies. It is written in an admirable style, and is full of happy suggestions for thought.

The Functional Inertia of Living Matter: a Contribution to the Physiological Theory of Life. By DAVID FRASER HARRIS, M.D., Lecturer on Physiology in the University of St. Andrews. Pp. 136. London: Churchill, 1908.

The author believes that, in addition to excitability, protoplasm possesses the fundamental property of functional inertia. This is expressed in latent periods, insusceptibilities to stimulation and rhythms. It also lies at the bottom of some of the facts of hereditary disposition, maintenance of type and psychic inertia, and is not merely a diminution of affectability, but a positive property of all living matter.

Mind and its Disorders: a Text-book for Students and Practitioners. By W. H. B. STODDART, M.D., F.R.C.S. With many illustrations. Pp. 488. London: H. K. Lewis, 1908.

This book is divided into three parts, devoted to normal psychology, the psychology of the insane, and a detailed consideration of the forms of mental disease. So large a scheme leads to the somewhat superficial treatment of some of the subjects touched on in the more abstract parts of the work, but more than two-thirds of the book are devoted to a useful and practical account of the phenomena of insanity. It will be useful to the general practitioner or to the student preparing for one of the higher examinations.

The Sexual Life of our Time in its Relations to Modern Civilization. By IWAN BLOCH, M.D., translated from the Sixth German Edition by M. EDEN PAUL, M.D. Pp. 790. London: Rebman, 1908.

This book deals with sexual life, normal and abnormal, with the forensic aspect of offences against morality, with sexual education and many other similar questions. It belongs to a series of works of which Forel's "Sexual Question"

is the best known, written apparently for the intelligent layman rather than the specialist. The pathological portion will add nothing to his knowledge of psycopathia sexualis. The frequency with which words and sentences are printed in heavy type is offensive to the eye of the reader.

Transactions of the Ophthalmological Society of the United Kingdom. Vol. 28. Fasc. iii. London: Churchill, 1908.

This number contains three papers by Mr. Nettleship bearing on heredity—senile cataract in husband and wife: conditions of the lenses in their children and grandchildren, lamellar cataract, discoid cataract, and retinitis pigmentosa, affecting different members of the same pedigree; and, lastly, a colour-blind family. The remaining papers have no general interest.

Handbook for Attendants on the Insane. Fifth Edition revised and enlarged. Published by the Authority of the Medico-Psychological Association.

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