

Clinical Cases.

FIVE CASES OF DISEASE OF THE BRAIN, STUDIED CHIEFLY WITH REFERENCE TO LOCALISA- TION.

BY CHARLES K. MILLS, M.D.

Neurologist to the Philadelphia Hospital.

- I. Softening of the Pons Varolii.
- II. Tumour of the Pons Varolii.
- III. Localised Tubercular Meningitis.
- IV. Localised Tubercular Meningitis; Internal Pachymeningitis.
- V. Multiple Cerebral Embolism.

THE cases contained in this paper are, I think, of value from a number of stand-points, but my remarks upon them will be brief, and will chiefly refer to their bearing upon the question of cerebral localisation. Much of pathological interest is taught by them in regard to such subjects as thrombosis and embolism, necrotic and inflammatory softening, meningeal tuberculosis and pachymeningitis, and primary and secondary disease of the cerebral vessels; but too elaborate a discussion of separate features would have extended the article to unwieldy limits. Even with reference to localisation, the cases are, as far as possible, allowed to speak for themselves. Cases of multiple or diffused lesions, such as three of the five here presented, are not, as some suppose, without value in the study of the problem of the cerebral functions. If, for instance, we have a symptom-picture, which points with great positiveness to lesions of the cortical motor zone, and if, after death, such lesions are found, we should not disregard them altogether because of complicating conditions. In every field of medicine we are compelled to resort to processes of

comparison and exclusion, in order to arrive at a satisfactory explanation of special manifestations. The all-important point is that we shall be honest and thorough in our investigations.

CASE I.—Softening of the Pons Varolii—Right Hemiplegia followed by General Paralysis—Rapid Elevation of Temperature before Death.

B—, aged 40, was admitted to the Philadelphia Hospital on May 20, 1879. He stated that three weeks before, while lying down, he suddenly felt as if his head was spinning round. He tried to get on his feet, but could not do so on account of the vertigo and a general loss of strength. He did not lose consciousness during the seizure. His speech became so "thick" that he could not be understood, and he found, on rallying from the first effects of the attack, that he was partially paralysed in the right arm and leg, and that his face was a little drawn to the left.

For several weeks previous to this seizure he had suffered with headache. He had abused alcohol, and also had a history of syphilis.

During the three weeks previous to his admission he had been slowly improving. He was hemiplegic on the right side, and was weak in all his limbs, but he could sit up and could walk around, holding on to the beds and chairs with his left hand. The paralysis was more decided in the right upper extremity than in the lower, but it was well marked in both. He had a slight left convergent strabismus, and his mouth was drawn a little to the left, but his face was not otherwise affected; no ptosis, external strabismus, lagophthalmus, or other symptoms, except those noted, being present. He could converse without difficulty, although his speech was not perfectly distinct, owing apparently to slight lingual and oral weakness. He had full control of rectum and bladder. Sensation was well preserved. He was pale and haggard.

The above was his condition during the first twenty-four hours after coming under observation on the 20th of May. On the 21st, he called the attention of the nurse to the fact that he had suddenly become paralysed in the left arm; during the same day his left leg was attacked; he became much weaker in every respect, and had to take to his bed. He did not change notably in his symptoms until the 23rd, when he began to complain of severe frontal headache. On this day his urine was examined, and was found to contain neither albumen, tube-casts, nor sugar.

On the 24th he had a peculiar seizure, which, as I chanced

to be present in the ward at the time, I was fortunate enough to witness. His face was pale and anxious, and he broke out into a profuse general perspiration. The left internal strabismus was observed to be much more marked than it was on admission. Respiration became difficult, but it was not stertorous; he breathed in a jerky manner, and was almost constantly making a puffing sound with his mouth, at which also he frothed a little. He could not speak, and could not thrust his tongue beyond his lips. On talking to him, he seemed to understand all that was said to him, and would make futile efforts to reply; he would become highly excited, looking at those around him with a most beseeching expression, while the tears suffused his cheeks, as if appreciating the helplessness and hopelessness of his situation. He could only open his mouth enough to separate his teeth from a quarter to half an inch. Liquid food and medicine could be taken, but he had some difficulty in swallowing, owing to a tendency to regurgitation. He was helpless in all his limbs, but the loss of power seemed still more prominent on the right. Slight flexions were present at the wrist, in the fingers of the right side, and at both knees. At intervals he had considerable convulsive tremor, affecting especially the right side, and the leg more than the arm. His pulse was 88 and weak; his temperature, taken in the rectum, was 100.4° F. Involuntary passages did not occur; nausea and vomiting were not present. His eyes and neck, at this time, showed no tendency to deviate either to the right or left.

On the 25th, 26th, and 27th, he continued in nearly the same condition, having improved slightly, under counter-irritation to the back of the neck and extremities, and the use of stimulants. He still perspired freely nearly all the time; he remained anxious-looking and emotional, his breathing became a little better, but continued to be of the same puffing, jerky character; he did not regain the power of speaking, thrusting out his tongue, or swallowing without difficulty; the strabismus and helplessness of his limbs did not change. His pulse was weak, and ranged between 100 and 112. His temperature, taken in the rectum, varied but little during these three days. The record was as follows:—

Morning.				Evening.			
May 25	.	.	100° F.	.	.	Not taken.	
" 26	.	.	100° F.	.	.	100.4° F.	
" 27	.	.	99.8° F.	.	.	100.3° F.	

On the morning of the 28th he became much worse. Sweating became more profuse; his paralytic symptoms deepened;

about midday involuntary discharges took place; his head and eyes, for the first time, showed a constant tendency to turn to the *right*; his breathing became more and more irregular and difficult. His temperature shot up rapidly; at 7 A.M. it was 101.3° F.; at 3 P.M., 105.5°; at 6½ P.M., 108°; after this it was not taken. He died at 11½ P.M.

An autopsy was held fourteen hours after death. The pia mater exhibited slight general cloudiness, with scattered patches of extreme redness. On examining the base the basilar artery was found to be enlarged to nearly twice its usual diameter, its walls being thickened and rigid, and rough on the outside. Some of the small vessels, which go down from it into the pons, could be seen to be obliterated; the basilar trunk itself, however, was quite free and of good calibre, and was readily opened with the scissors. The other main arteries, and their primary and secondary branches, showed wide-spread evidences of atheroma.

The pons varolii was the seat of an extensive and interesting lesion, the following description of which is partly from notes made at the time of the post-mortem examination, and partly from careful subsequent investigation of the specimen. This lesion was an irregular area of softening, the centre of which was just below the centre of the pons. Superficially, the softening was an inch in greatest length, which was from above downwards and to the left of the median line, and three-fourths of an inch in width at its widest part, which was near its lower boundary. Vertically, the softening extended about one-fourth of an inch higher to the left of the median line; while horizontally, and below the centre, it reached about one-eighth of an inch farther to the right than to the left. It presented four spots of excavation, the intervening spaces being filled up with softened and broken-down tissue; one of these was a comparatively large central space, the second was to the left and above, the third to the left and below, and the fourth to the right and below the centre. The entire surface-softening was included in an irregular triangle, formed by joining these outer points of depression or excavation. On subsequent close examination, the central region of the pons was found to be scooped out to the depth of half an inch, and it was also irregularly invaded in all directions by softened tissue; but its upper and lower fifths, and lateral bands of from one-fifth to one-sixth of its width, were unaffected. The cranial nerves, superficially, were not involved. The floor of the fourth ventricle presented a healthy appearance.

An extravasation as large as the palm of the hand was present in the cardiac end of the stomach. The aorta was

atheromatous. Both kidneys were fatty, the degeneration being more advanced in the right than in the left.

Remarks.—Summing up this case, the chief points are found to be as follows: headache; a vertiginous seizure, which left partial right hemiplegia; left convergent strabismus; defective articulation; paralysis of the left arm coming on suddenly three weeks after the first attack, but without any special head or general symptoms; a third seizure, accompanied by pallor, anxiety of expression, great emotionality, profuse perspiration, difficulty in breathing and swallowing; inability to speak, to thrust out the tongue, or open the mouth widely; paralysis of both arms and both legs, but still more pronounced on the right; convulsive tremor, most marked in right leg; weak and frequent pulse; slight elevation of rectal temperature for four days. On the day of death, deepening of all the symptoms just noted, and, in addition, paralysis of bladder and bowels, conjugate deviation to the right, and rapid elevation of temperature to 108° F. Some interesting negative facts are, absence of anæsthesia, of nausea and vomiting, of true convulsions, and of changes in the urine.

At first sight this case might be looked upon as contradictory of the views of those who hold to the decussation at the anterior pyramids of voluntary motor fibres, but I doubt whether it should be so regarded. Leaving out the question of some possible peculiarity in the decussation, in regard to which I have not yet had the opportunity to determine positively, we have in the history of the case, conjoined with the results of the autopsy, a satisfactory explanation of all the manifestations. The paralysis following the first seizure was of the face and limbs of the right side, and it continued until the day of death to be most decided on the right. The autopsy revealed an area of softening in the pons, occupying the centre and both sides of the median line, and this was evidently due to closure by a thrombotic process of some of the little nutrient arteries given off from the basilar. The different vessels probably became obstructed at intervals corresponding to the several successive seizures which the patient experienced. The softening in the centre and to the left may, in this way, have taken place earliest, an order of events which would best account for the right hemiplegia. The spots of excavation, spoken of in the notes of the autopsy, possibly represented the point at which the successive attacks of softening occurred. Paralysis of both sides of the body did occur before the death of the patient, as might be expected from a lesion of the kind and in the situation described.

Convergent strabismus, inability to articulate, to thrust out

the tongue or to open the mouth widely, and difficulty in swallowing and breathing, as the cranial nerves were not affected superficially, point to the probable involvement of the nuclei or deep tracks of the abducens, facial, hypoglossal, motor-trigeminal, glossopharyngeal, pneumogastric and spinal accessory. The escape of the posterior columns and the floor of the fourth ventricle are of interest in connection with the absence of anæsthesia, of nausea and vomiting, and of true convulsions.

The temperature observations made upon this case, although limited, are sufficient to be of some value in reference to the question of a heat-centre in the pons. Supposing the existence of such a centre, it is probable that the first foci of softening did not implicate it directly, but as the necrotic area enlarged, this centre became eventually involved.

CASE II.—Tumour of the Pons Varolii—Convulsions, Vomiting, Rotatory Movements towards the Left—Final Hæmorrhage into the Medulla Oblongata.

J—, 35 years of age, seven years before coming under observation, began occasionally to have headaches, and a year later became subject also to temporal and orbital neuralgias. The headaches and neuralgic attacks gradually grew worse and worse, both as regards frequency and severity. Five years after his first headaches he began to have "spells," in which he would be unconscious for a moment or two, and would have twitchings about the mouth and in the hands. These seizures returned at intervals of from one to two months for about a year, when they were supplanted by general convulsions. Sometimes he would have several convulsive paroxysms in the same day. Before the attacks and during these days of spasmodic storm he suffered with terrible headache and uncontrollable vomiting. Often at other times, without convulsions, he had spells of nausea and headache. Ophthalmoscopic examination showed atrophy of both optic nerves. His memory had failed, and he was usually in poor spirits. No positive paralysis was ever observed, although he was never closely examined for slight deficiencies in strength. His bowels were obstinately constipated, and he suffered nearly all the time from fecal accumulations. He occasionally had "cramps" in the stomach and legs. He denied venereal disease of any kind, but his wife had had several stillborn children. The bromides, potassium-iodide, mercurials, and measures to relieve special symptoms, such as constipation, were employed, but without doing more than slight temporary good.

Six weeks before his death I saw him in one of a series of terrible convulsions. Although the convulsion was general, his right side seemed to be more affected than the left. Lying on his back, the spasms had the effect of lifting up the right side of his body and causing him to work over towards the left, as if trying to get on his face in this direction; but before getting altogether on his left side, he would fall backwards again, when the same curious lifting and rotary movement would be repeated. Frothy saliva escaped from his mouth; his face was much contorted, and was pale and livid by turns. The seizure lasted in all three minutes; when he relaxed he was bathed in perspiration and went off into a stertorous sleep. He died at the close of a series of similar convulsions, his death being preceded by general paralysis and involuntary evacuations.

The post-mortem examination, made twenty-three hours after death, revealed a round white tumour, a quarter of an inch in diameter, in the pia mater just to the left of the centre of the anterior surface of the pons varolii. The entire anterior central portion of the pons was softer and more doughy than usual. Just at the junction of the pons with the medulla oblongata was a recent hæmorrhage, which had spread downwards to about the middle of the latter. No other lesions of the brain were discovered, and this was the only organ examined.

Microscopic examination showed the tumour found in the pia mater to be a granulation tissue, probably of syphilitic origin. Nothing but mere detritus masses could be detected in the softened portion of the pons, and it could not therefore be determined positively whether this was a broken-down gumma.

Remarks.—The important symptoms in this case were headache, neuralgia in a portion of the trigeminal distribution; *petit mal*, supplanted after a time by fully formed convulsions; convulsions general, but the movements more marked on the right half of the body; during the convulsions, execution of a rotatory movement towards the left; failure of sight and optic atrophy; loss of memory and depression of spirits; frequent spells of nausea and uncontrollable vomiting at the time of the seizures; obstinate constipation, and occasional cramps or spasms in the stomach and legs. The above are points carefully studied and noted when the observations were made. The case, however, was under my care several years since, when I was not so fully impressed as I am now with the importance of investigating minutely. Sensation was not

closely studied, although I remember distinctly that on several occasions the patient complained of numbness of one side of the face. No definite paralysis of face, arms, or legs, of muscles or muscular groups, was present, although the man showed general and gradually progressing weakness of all the limbs. The trigeminal neuralgia, with probable anaesthesia, the peculiar epileptiform seizures, the local cramps or spasms, and the excessive vomiting, were symptoms which pointed to tumour of the pons—to a strongly irritative lesion in this region. The headache, optic atrophy and mental failure are the accompaniments of cerebral tumours in various locations. General weakness of all the limbs is the condition found in a number of cases of central lesion of the pons. Even supposing the central softened mass on the anterior surface of the pons to be a gummatous tumour, some of the fibres constituting the motor tracks on both sides were probably uninjured. The peculiar rotatory movements towards the left, recall the experiments in which Schiff divided the middle cerebellar peduncles at the side of the pons, the result being the performance by the animal of a rotatory movement towards the side of the lesion. The pial tumour was a little to the left of the median line, although the actual destruction of the pons was central. The fibres which go to form the middle cerebellar peduncle were, no doubt, affected by pressure and directly, the irritation being greatest to the left. The lesions were so situated as not to have involved directly the great sensory tracts. The hæmorrhage which spread over the medulla oblongata was doubtless the immediate cause of death; and this was probably brought about by the impairment of the vessels by disease of their walls and by proximity to the growth, and by the rise in blood-pressure during the terrible epileptiform paroxysms.

CASE III.—*Localised Tubercular Meningitis, with Cortical Softening—Involvement of the Centres for the Limbs and Face.*

M. H.—, 30 years of age, could not give a very clear and full previous history; but stated that one year before coming under observation she had been ill for several weeks; that she had had high fever, with great pain in her limbs, particularly in the right arm and leg. During and after this attack she suffered almost constantly from headache, and occasionally from dizziness. She had also been troubled with cough. Four months before her admission to the hospital, while standing over a fire cooking, her right arm suddenly fell helpless

to her side. About ten days later, the right side of her face became partially paralysed; and still later, but exactly when she did not remember, her right leg became weak. Two months before admission she had an inflammatory affection of the right eye, from which she had just recovered. She had kept on her feet most of the time, but says that she was feverish and weak, and was much worried with pain in her head.

On examination I found that the right arm and right leg, which were about equally affected, were markedly weaker than the limbs of the left side; they were decidedly paretic, but presented no contractures, and only slight wasting. The lower part of the right side of the face was also paretic, as was shown by slight dropping of the nostril and corner of the mouth, and twisting to the left of the mouth. She could not draw up the right angle of the mouth when directed to do so, as she could the left. The tongue was not deflected, but its movements were performed in a weakly, tremulous manner. The right eyelid drooped slightly. No strabismus was present. The condition of the pupil could not be determined, on account of an opacity which covered the right cornea almost entirely, and was the result of the recent keratitis from which the patient had been suffering. Both eyelids could be opened and closed, and frontal furrows were present on both sides. Her manner of speaking was hesitating and unsteady, as if from weakness of the oral and lingual muscles. She was not aphasic, in the sense of having any partial or complete loss of speech. She did not drop letters, syllables, or words; but talked along, without stopping, in a drawling, uncertain, slipshod sort of way. Sensation was unimpaired. Hearing was good. Smell was defective, but not lost on either side. She could not, of course, see with the right eye, with its opaque cornea; the sight of the left was poor. Ophthalmoscopic examination, by Dr. E. O. Shakespeare, revealed a beginning neuroretinitis. Her mind acted torpidly, and, judging from the results of questioning her in regard to her past history, her memory was of the poorest kind. She had full control of her evacuations. She had no history of vomiting or convulsions. Her appetite was poor, and she was constipated. No examination of pulse or temperature was at this time made. She had probably had venereal disease, but I could not determine this certainly. Percussion and auscultation showed partial consolidation of the apices of both lungs.

She was ordered potassium iodide and cod-liver oil. Sina-pisms were also applied to the neck, and a calomel purge was given occasionally. She improved a little in every respect;

but particularly as regards her headache, which almost entirely left her. After remaining under treatment for five weeks, during which time she was never confined to her bed, she asked for her discharge, and left to return to her family; but twelve days later she was again brought to the hospital, but now in a helpless and semi-conscious condition, from which she never rallied, dying one week after her re-admission. A few points were again noted. The loss of power in the right arm and leg had become a marked paralysis. The leg could be moved up and down over a space of a few inches, as she lay in bed. The paresis of the lower facial fibres was more positive; and both ptosis and rotation outwards of the eye were present and well marked. At intervals the right leg, and the arm and leg of the *left* side were the seat of twitching or convulsive tremor. She had fever, with paroxysms of sweating; she was delirious at times; her pulse was frequent and irregular.

The autopsy was held sixteen hours after death. The dura mater of the left hemisphere was slightly adherent in its inner surface at two points along the parietal margin of the fissure of Rolando, one at the median end of the fissure and the other just above its centre. On pulling the membrane away, which could be easily done, a mass of soft yellow exudation was found at each place of adhesion. The pia mater, both anterior and posterior to the fissure of Rolando, presented evidences of inflammatory action, being more or less opaque, streaked, and hyperæmic. On attempting to remove it, at several points its under-surface was found to be adherent to the convolutions, and to be the seat of what appeared to be tubercular masses, with purulent exudation, the cortex being superficially softened at some of the points. The peculiar character and localisation of the pathological process in the pia mater and cortex may be understood from the following description: (1) The pia mater was opaque, thickened slightly, and adherent at points over the paracentral lobule and the upper extremities of both ascending convolutions, the cortex being softened over a spot one-fourth of an inch in diameter in the ascending parietal convolution, at its upper limit. (2) Deposits, yellowish exudation, and a space of superficial softening, half an inch in diameter, were present about the centre of the ascending parietal convolution. (3) Two foci of cortical softening were found in the ascending convolutions, on each side of the lower fourth of Rolando's fissure, with pial changes at corresponding points.

The sides of the fissure of Rolando, at its middle third, were bound together by inflammatory products.

The pia mater of the interpeduncular space was very opaque,

much thickened, and of a gelatinous consistence; and the changed condition of the membrane, but with less opacity and thickening, continued into the left fissure of Sylvius, and upwards from the horizontal branch of this fissure along the convexity of the hemisphere in the general region of the distribution of the Sylvian artery. Greyish nodules were found here and there along the vessels, and a few purulent streaks and hyperæmic spots, besides the special areas along the fissure of Rolando already described. The pia around the optic chiasm, and particularly on its left side, was markedly thickened and changed in appearance. All the vessels, nerves, and bodies, at the base, from the pons to the chiasm, were more or less involved in an inflammatory process of the membrane. The microscopic appearances pointed to a tubercular inflammation of the pia mater, confined chiefly to the central portion of the base of the brain, and to the territory of the left middle cerebral artery. The tubercular process had been most extensive, and its effects most severe, at the points along the fissure of Rolando, and over the paracentral lobule, which I have specifically described. Some development of miliary tubercles had probably occurred at points in the cortex itself. Small vessels had certainly been obliterated. The pia mater over the pons and medulla, over both occipital lobes, and along the edges of the longitudinal fissure, was much more injected than it is usually found, but no tubercular appearances were discovered outside of the territories detailed. The ganglia, and all portions of the brain, were examined, but no other abnormal conditions were found. I was not able to secure an examination of any other organs.

Several specimens of the diseased pia mater, with blood-vessels, and the softened cortex, and the eyes with parts of the optic nerves attached, were submitted to Dr. E. O. Shakespeare for microscopical examination, and to him I owe the following valuable report, and also the opportunity of examining the mounted specimens:—

“The specimens of the membranes and cortex of the brain, and of some of the larger cerebral vessels, were found in good condition for microscopical examination. Thin sections were made from the softened cerebral convolutions, with its attached pial covering, were stained with carmine, and were temporarily mounted in oil of cloves. The larger vessels were treated in a similar manner; and transverse sections of them, when placed beneath the microscope, showed the walls of many to be diseased. The lesion was limited almost exclusively to the adventitia and surrounding connective tissue. In general, only the outer part of the muscular tunic was affected. The

tunica intima showed scarcely any appreciable alteration. The lesion located in the outer coat of the vessel was characterised by a dense accumulation of embryonal cells in the meshes of its reticulum. Seen in longitudinal section, the vessels often presented a fusiform enlargement from point to point, an alteration which was restricted to the outer coat, and which rarely ever affected the calibre of the vessels. It was almost exclusively the arteries which were thus diseased.

"The sections of the cerebral cortex were made vertical to the surface of the softened convolution. They showed a variable number of caseous nodules, some of which were situated in the cortical substance near the surface, others in the thickened and inflamed pia mater. Upon minute examination, these caseous nodules were found to be composed of numbers of smaller caseous foci, massed together and united by an embryonal tissue, which presented the following peculiarities: in the portions at some distance from the minute caseous foci, embryonal cells were crowded together so thickly that it was impossible to discover any trace of the original structure. Scattered here and there in this embryonal mass were to be seen a number of what many authors figure and describe as 'giant cells.' On first view, and under a moderate magnification, these large multinucleated bodies certainly presented many of the characteristics of myélopaxes; indeed, at the very edge of the minute caseous foci, where the embryonal tissue was on the border-land of caseous degeneration, it was altogether impossible from an examination of these bodies at this location alone to say that they were not genuine 'giant cells.' Recurring, however, to the more remote portions of the embryonal mass, where the cellular tissue was not nearly so much altered, by a very careful study, under a very high power given by an excellent lens, there was no difficulty in recognising peculiarities in these large multinuclear bodies, which demonstrated their nature and construction to be different from those of true 'giant cells.' In the loose altered spots of the embryonal mass could very readily be recognised the lumen, wall, and peri-vascular lymph-space of small arteriole and other blood-vessels. Many of these vessels presented an unobstructed lumen; and yet the walls of all of them were altered. The lesion of the patulous vessels was again mainly limited to the external coat, which, together with the outer layers of the middle coat, was represented by a mass of embryonal cells, constituting a cylinder. The endothelia of the tunica intima were swollen, and presented enlarged nuclei, but did not appear to be proliferating. Sometimes the peri-vascular lymph-space would be filled more or less completely with large

embryonal cells, but it was usually almost empty. The swollen vessels presented corresponding alterations. In some less altered spots, many of the vessels, in the same condition above described as to their walls, were plugged with a granular, fibrinous clot, which entirely filled the lumen, and which enclosed some white blood-corpuscles, a few detached swollen endothelia, and sometimes small numbers of visible red blood disks. In these cases a granular fibrinous coagulation more or less completely obstructed the perivascular lymph space also. In the intermediate region, between the above-mentioned altered portions of the embryonal mass and a narrow zone immediately surrounding the minute caseous foci, were many multinuclear bodies, in which the outlines of the vessels could still be faintly recognised by the arrangement of the nuclei. In this region were some also, in which, without the aid of a previous study, no suspicion of a similar origin and mode of formation could be suggested by their appearance, every semblance of a transverse cut of a vessel having been entirely lost; and yet, throughout the different portions of the embryonal mass, multinuclear bodies were so numerous, and of such varied appearance, that every gradation could be traced between the sections of undoubted blood-vessels, the external parts of which were in a state of proliferation, and the multinuclear bodies in the neighbourhood of a caseous focus, which gave no hint of such an origin.

"In the brain substance, and in the pia mater at the border of the embryonal masses which surrounded the caseous foci, the arterioles and the capillaries were altered in the following manner. Their external portions showed a cellular irritation throughout their entire length; at points the irritation had gone so far as to result in a nodular, or fusiform, or cylindrical thickening of the outer wall of the vessel. In the immediate vicinity of these altered vessels the tissue was in a state of inflammatory hyperplasia or inflammatory softening.

"From the foregoing study of the microscopical characters of the various lesions, it must be concluded that the specific morbid process in this case was that of tuberculosis.

"Longitudinal sections of one optic nerve, at its entrance into the eye, were made and prepared for study. Examination of these showed the nerve to be in the early stages of a descending optic neuritis."

This report speaks for itself, showing clearly the existence of tuberculosis explaining satisfactorily the occurrence of the softening, and containing several points of special interest, such, for instance, as those relating to supposed "giant cells."

Remarks.—Although the autopsy revealed a somewhat diffuse meningeal inflammation, the only definite destructive lesions were those of the convolutions surrounding the fissure of Rolando. The inflammation, deposit, exudation and destruction of the cortex were greatest at the middle region of the left ascending convolution, where are situated the motor centres for the movements of the upper extremity. Motor paralysis of the right arm was the most marked of the paralytic manifestations. To explain the paralysis of the leg, we have thickening and adhesions of the pia mater over the paracentral lobule, with a small area of softening high up in the ascending parietal convolution. The nasal, oral, and lingual paresis have their explanation in the foci of softening and the tubercular granulations low down in the ascending convolutions. The lesions of the cortex were carefully and closely studied, a pencil-sketch, with measurements, indicating their intensity and exact location, being made at the time of autopsy, under my directions, by one of my resident physicians, Dr. Bell. Her disturbance of speech was an oro-lingual paresis rather than a true aphasia. The ptosis and late external strabismus, were probably due to the involvement of the oculo-motor nerve in the meningeal inflammation at the base. Both ophthalmoscopic examination and the microscope showed the association of optic neuritis with the meningitis.

CASE IV.—Localised Tubercular Meningitis with Cortical Softening—Involvement of Oro-Lingual Centres—Internal Pachymeningitis of the Opposite Hemisphere.

Y—, aged 65, was sent from the Blind Ward to the ward for nervous diseases, because he had had a slight apoplectiform seizure. I could only learn in regard to him that he had been weak and thin for a long time; that he had been troubled with cough and diarrhoea; and that he had complained frequently of headache and giddiness. His mind acted very feebly; he seemed to understand what was said to him, but he was somewhat obtuse and slow in answering. His speech was peculiar, it might be described as slightly “staggering:” a little tremor was present in the tongue and the muscles about the mouth; he sometimes dropped a letter or syllable. He was drowsy, dosing the larger part of his time. The only evidence of loss of power in the face, which was carefully studied, was about the mouth, which was drawn slightly to the left, and the right angle of which drooped slightly. His tongue did not deflect to either side, but its movements showed weakness and some want of control. The uvula and soft

palate were not paralysed. Neither ptosis nor strabismus was present. Cataract operations had been performed on both eyes, and he was nearly blind, having only quantitative vision. His limbs were all very weak, but no distinct motor paralysis, more marked in one than in the other, could be made out, except that the grip of the right hand was poorer than that of the left. His right leg, however, had been amputated a few inches below the knee, about five years before coming under observation, which prevented a full determination as to paralysis on this side. Physical examination of both lungs showed that wide-spread and advanced tuberculous disease was probably present. He was troubled with an obstinate diarrhoea, and died, in ten days after admission, of exhaustion, which was largely due to this diarrhoea.

An autopsy was held twelve hours after death. The skull was normal. The brain weighed forty-three ounces. The inner surface of the dura mater of the convex portion of the right hemisphere, from the middle of the parietal lobe forward to the anterior extremity of the brain, was covered with a layer, from half a line to a line in thickness, of a red, yellowish-red, and reddish-brown colour at different points. This stratum could be readily scraped from the dura, leaving this membrane intact beneath. The same formation, but of less thickness, extended down the right side of the falx. The pia mater, beneath the changed dura, presented a slightly opaque, bluish-white appearance. After stripping up this membrane, it was found that all the convolutions of the convex surface of the hemisphere, within the area covered by the diseased dura, were much flattened, and of a reddish-brown or light rusty colour.

The dura mater of the left hemisphere was unchanged. The pia mater of this side was hyperæmic, and here and there through it were seen peculiar-looking straight streaks of red. On removing the membrane from the substance of the brain it was adherent at several points, particularly along the fissure of Rolando. Miliary tubercles were made out at scattered points over the postero-frontal and parietal lobes.

On the surface of the left hemisphere were four foci of superficial softening, corresponding to points where the pia mater was more or less adherent to the cortex, and presented on its under-surface spots of yellowish exudation. The softening was only from one to two lines in depth, and was of a whitish-yellow colour; it had probably resulted from the obliteration of small vessels by the tuberculous process, which was more intense in the pia mater at these positions than elsewhere. These softened areas were situated as follows: (1) An

irregular area, five-eighths of an inch wide at its upper boundary, and narrowing below to one-fourth of an inch, at the level of the middle third of the fissure of Rolando, its posterior margin being eleven-sixteenths of an inch in front of this fissure; it included small portions of the second, third, and ascending frontal convolutions. (2) A spot, about the size of a half-dime, in the ascending parietal convolution, about the junction of the middle and lower thirds of the fissure of Rolando. (3) A spot, two-thirds of an inch vertically, and one-fifth of an inch wide, extending on both sides of and one-third of an inch below the lower extremity of the same fissure. (4) An area, five-eighths of an inch in width, and extending across the second temporal convolution, an inch backwards from the Sylvian fossa.

A very slight depression was found about the centre of the ventricular surface of the left corpus striatum.

Both lungs were infiltrated with miliary tubercles, the upper lobes being most affected. Cheesy metamorphosis had taken place in several localities, and an abscess had formed in the right apex. The cardiac valves were all-sufficient, and nothing abnormal was found in the examination of the heart except a single calcareous plate on the outer side of one of the aortic crescents. The right kidney contained a brown encysted stone, and two similar stones were found in the substance of the left kidney. Both small and large intestines presented a remarkable condition of ulceration; fifty-two distinct ulcerative foci being counted in the former, and twenty in the latter.

Microscopical examination confirmed the macroscopic evidences of the existence of tuberculosis of the pia mater.

Remarks.—In this case we have a tuberculosis of the pia mater of the convexity of the left hemisphere. The tubercles discovered were fewer in number, and the signs of inflammation were less marked than in the last case; speaking generally, the same portion of the convexity was attacked in both, but in the present case the base escaped. Limited tuberculosis of the pia mater is not so rare as many neurologists suppose.

In regard to localisation, it will be seen from a study of the areas of softened cortex that portions of the arm, face, and speech-centres were implicated. The softening was very superficial, and the paresis of the right upper extremity may or may not have been the result of the lesions; but I am inclined to attribute the peculiar speech and the paresis of mouth and tongue to the cortical destruction, as the oro-lingual centres of Ferrier were so distinctly involved. In regard to the softening of the second temporal convolution, I have nothing to say in

connection with the symptomatology of the case. The special senses could not be studied to any purpose.

The internal hæmorrhagic pachymeningitis of the right hemisphere was in itself a most interesting lesion, but a lengthy discussion of it is foreign to my purpose in the present paper. Although the case was one complicated by many conditions, some of the patient's symptoms were doubtless due to the cerebral compression, which the autopsy showed had been exerted by the chronic pachymeningeal disease. Drowsiness was one of these symptoms; and the headache, vertigo, and weakness of the left half of the body may also have been partly, at least, due to the dural affection. Pachymeningitis has not unfrequently been observed in cases of tuberculosis of the pia mater, lungs, and other parts of the body, and also in connection with atheroma and affections of the kidneys.

CASE V.—Multiple Cerebral Embolism—Softening of the Corpus Striatum and Internal Capsule, and widely distributed Cortical Softening—Hemiplegia with Aphasia.

C—, aged 45 years, when she came under my care, was not in a condition to give any information herself, and the few facts in regard to her previous history were obtained from her relatives and friends. During three years she had suffered with pains in her limbs. She was brought to the hospital on the 18th of July. Late in the preceding winter she had a stroke, which paralysed the right half of the body and affected her speech. Afterwards she never used more than three expressions, namely, "Yes," "No," and "Do you know?" She became irritable and emotional. On the 6th of July she had a second apoplectic seizure while lying down. She could not move from the position she was in, but was not unconscious. From this time she was helpless, was unable to feed herself, had involuntary passages, and had some fever with delirium at night. She often carried her left hand to the head, as if it was the seat of pain.

She appeared to understand much that was said to her when she was first seen, but she could not answer questions correctly. Her vocabulary now consisted only of the two words "Yes" and "No," and she used one of these for the other indifferently. The mouth showed a scarcely perceptible twist towards the left, so slight that I could not feel sure that it was not a deviation such as is sometimes seen in those in health. She could not get her tongue beyond her lips. She could swallow, but with some difficulty. She could open and

shut both eyes, and no noticeable paresis was present in the upper part of the face. The right upper extremity was powerless, and was wasted from the shoulder to the finger-ends; the fingers were slightly clawed. The right leg was also helpless, but not so absolutely as the arm; the limb was wasted, the thigh much more than the leg below the knee. She had no appetite; she did not seem to care for food at all. Sensation, roughly tested, seemed to be retained; she shrank from the pricking of the æsthesiometer, but this may have been partly a reflex manifestation. Patellar reflex on the right side was decidedly exaggerated, and a slight clonus could be developed from the right ankle. The above notes on her condition were made July 20th. She lived until the 30th, gradually growing worse, although she sometimes rallied a little for a few hours. She had more or less fever all the time, her night delirium continued, her pulse became frequent and irregular, and profuse sweating often occurred. Her temperature was taken both morning and evening, and in both axillæ, from July 24th to the 29th. The temperatures registered in the right axilla, the side paralysed, were higher than those in the left. The evening temperatures were usually considerably higher than those of the morning. The averages of the observations were as follows:—

	Right Axilla.	Left Axilla.
Morning . . .	99° 7' F.	99° F.
Evening . . .	101° .	100° 2.

A post-mortem examination was made twelve hours after death. The dura mater was paler than usual, and the pia mater was everywhere so anæmic and transparent that the convolutions could be readily traced before its removal. The brain weighed forty-one ounces. No less than seven areas of true cortical softening were found, as follows: (1) A spot, about two-thirds of an inch in diameter, on the left side, where the first frontal, second frontal, and ascending frontal convolutions come together. (2) One, a third of an inch in diameter, in the left third frontal convolution at the bottom of a secondary fissure. (3) A similar focus on the outer edge of the left island of Reil. (4) An irregular area, two-thirds of an inch in its greatest length, about the middle of the first temporal convolution of the right side. (5) A large foyer, which began an inch to the right of the longitudinal fissure, and just anterior to the transverse occipital fissure of Ecker, and extended backwards, expanding like a fan to the posterior limit of the upper surface of the right occipital lobe. (6) A similar foyer in the left occipital lobe, which began at a corresponding point, and spread in the same general direction, but only for about half

the distance of the softening on the right side. (7) A small spot, one-third of an inch in diameter, on the right posterior aspect of the cerebellum.

On cutting into the left lateral ventricle, a small yellow spot was visible on the surface of the corpus striatum at the beginning of its cue or tail. On incising this, the ganglion beneath was found to be softened and partly diffuent, the destruction being deep enough to involve a portion of the internal capsule. No other lesions of convolutions, tracts, or ganglia were discovered, after diligent search. The blood-vessels, particularly those of medium and small calibre, showed at intervals evidences of inflammatory changes in their walls. I succeeded in finding emboli lodged in vessels leading directly into the softened occipital areas. At a number of points in the branches of the middle and posterior cerebrals, at more on the left side, were seen what appeared to be plugs, or minute blood-clots, some loosely and some firmly lodged.

Pleuritic adhesions, which were particularly firm and dense on the posterior aspect of the left lung, were found on both sides. The lungs were oedematous, but contained neither deposits nor cavities. The heart was dilated, and its walls were in a condition of fatty degeneration. No valvular lesions, however, were present. Both kidneys were atrophied, granular, and tough.

The vessels were carefully stripped from the brain, and these, with specimens from the softened areas, were examined microscopically by Dr. E. O. Shakespeare, who reported as follows: "The walls of the vessels were in a state of cellular hyperplasia. The endothelial cells covering the trabeculae of the finer fibrous network of the pia mater were swollen, granular, and sometimes contained more than one very distinct nucleus. In the meshes of the network in many places could be seen a coagulum of granular fibrine, enclosing white blood corpuscles, a few red ones, and some detached and swollen endothelia. In a few of the largest vessels an embolus could be distinctly recognised; and at the location of the plug, the inner, middle, and exterior coats of the vessels were much irritated and sometimes inflamed. The diseased cerebral substance presented at different points the usual appearances of inflammatory and necrotic softening. The pia mater attached to the softened cerebral tissue showed inflammatory thickening, and the appearances commonly present in subacute meningitis."

Remarks.—How shall we associate the symptoms presented by this case during life with the numerous lesions found after death? Such a multiplicity of diseased areas might at first

tend to throw us into confusion. The major portion of the dissociated hemiplegia was probably due to the softening of the left corpus striatum and adjacent internal capsule; the aphasia to this lesion, or conjointly to it, and to those of the third left frontal convolution and the island of Reil. The softening (1), at the junction of the first, second, and ascending frontal convolution, included the anterior upper portion of the general region for the superior extremity. It will be remembered that in the history given the paralysis and wasting were stated to be much more marked in the upper than in the lower limb. This fact was carefully determined, measurements of the limbs having been made both before and after death. The greater completeness of the brachial paralysis and wasting may have been attributable to the partial involvement of the general arm centre in one of the lesions, both the voluntary-motor centres in the cortex, and the sub-voluntary or automatic centres in the striate body probably being destroyed. The cortical areas of softening were not high enough to implicate the supposed centres for the leg; and the paralysis and atrophy of this member must, I think, be attributed alone to the lesion of the caudate nucleus and adjoining tracts. The wasting of the right thigh out of proportion to that below the knee, which strongly attracted my attention, is an observation not to be disregarded, although I have no special explanation to offer. I am satisfied that, with all the advances that have been made in this direction, we do not study our cases minutely enough. Many facts of value may yet be learned from the commonest examples of hemiplegia, by a closer study of symptomatology. Differences in the amount of wasting in various parts of the same limb, the greater preponderance of paralysis or spasm in certain muscles or muscular groups, and many other similar points, need to be more thoroughly investigated with reference to the effect and position of the lesions found post-mortem. The exact limitations, superficial and deep, of lesions of the basal ganglia, should be determined. In this case the head of the striate body escaped, but the softening extended below the caudate nucleus. Decided facial paralysis was absent, although a very slight paresis of the lower right facial region seemed to be present. The cortical facial zone, as usually given, just escaped. The tongue and the muscles concerned in swallowing were undoubtedly affected. Owing to the condition of the patient, hearing and the special senses generally could not be thoroughly investigated, so that I am not able to say whether any defect of hearing resulted from the temporo-sphenoidal lesion. In regard to the large foyers of softening in both occipital lobes, and the cerebellar

focus, if any inferences in regard to the lesions in other regions have been correct, the effects produced by these were latent, or were not of a positive character. Ferrier regards the occipital lobes as specially related to the visceral or organic sensibilities, but is doubtful whether I would be justified in referring such symptoms as impaired appetite and involuntary discharges, which occur in so many conditions, to any localised cerebral lesion. Sensation, so far as it could be tested, seemed to be unimpaired.

It may be worth while to give a passing notice to such symptoms as patella reflex, temperature, and fever and delirium. Very commonly, as in this case, in hemiplegics and monoplegics, tendon reflex is exaggerated on the paralysed side, owing probably, in part, at least, to the fact that the parts below the lesion are more or less cut off, or from the restraining influence of the higher brain. The temperature was slightly elevated, as in many instances of localised cerebral disease; it was also higher on the paralysed side than on the other. The case being one of demonstrable multiple embolism of vessels of appreciable size, Bastian's hypothesis in regard to fever and delirium being due to capillary occlusions becomes worthy of consideration. The conditions were such as to bring about such occlusions.

Pathologically, this case is a most interesting illustration of multiple cerebral embolism. It is probable that on several occasions particles from a partially-organised blood-clot were carried into the circulation, and lodged in the cerebral vessels. Some of these became permanent fixtures at their points of lodgment; others were immediately swept out again. Necrotic softening took place in some instances in the districts supplied by the occluded vessels; in others this process was probably prevented by collateral circulation. As has been noted, plugs were discovered in a number of places in branches of the middle and posterior cerebral arteries, these in some cases apparently not having caused necrobiosis. In some recent experiments made in conjunction with Dr. A. J. Parker, of Philadelphia, we found the communications between the three chief vascular territories of the brain freer even than is held by Heubner, and far freer than Duret would have us believe. Our inspections so far have been made only on two brains, and great individual differences may and probably do exist; but the fact that in these two sets of experiments we found communicating vessels at least two millimetres in diameter, shows that parts of the brain may frequently be saved from softening by anastomosing vessels. This is contrary to the views advanced by me in a Review of the subject of *Cerebral Localisation*

(‘American Journal of the Medical Sciences,’ July 1879), in which I state that up to the time of writing, my clinico-pathological observations were corroborative of the views of Duret, Cohnheim, and Charcot. Subsequent pathological experience, and the experiments just referred to, have, however, led me to a somewhat different conclusion, although I do not yet feel satisfied in regard to the question. Multiple embolism is probably more frequent than is usually supposed, occluded vessels which do *not* occasion softening often escaping attention.

The inflammation of the pia mater was confined to that portion of the membrane in immediate relation with the necrotic districts.

The inflammation of the walls of the vessels, which was so marked, may have been partly aroused by the irritation produced by the numerous embolic foci, partly it was due to these vessels coming from the zone of inflammation around the softened areas.

The co-existence of fatty degeneration and dilatation of the heart with contracted kidneys affords a plausible explanation of the formation of the supposed clot. A tendency to the production of cardiac and other thrombi is well known to exist in diseases such as fatty heart, which cause marked impairment of the power of this organ.

The areas of softening observed were more numerous in the left than on the right half of the brain, which is in accordance with the well-established and easily explained fact that emboli are much more frequently carried into the left carotid than into the right. The occurrence of bilateral softening of the occipital lobes is deserving of passing attention. A tendency to bilateral embolism has been noted by several observers.