rence of slow pulse and low temperature in intracranial otitic abscess, and on the untrustworthiness of local tenderness as a guide to its localisation. Three years later appeared the now classical lectures of Mr. Barker, in which the pathology of the cerebral complications attendant on ear disease is exhaustively dealt with. It would be impossible for me adequately to dilate upon the many excellences of those lectures, which in the topographical anatomy and the details they furnish for the diagnosis of intracranial pathological conditions, indicating, for example, the favourite sites of extradural and the limits of temporo-sphenoidal abscesses, in defining the symptoms of thrombosis of the lateral sinus, further in advocating the use of small trephines, and in giving particulars of a series of successful operations, including the first successful one for cerebral abscess by Macewen, have proved simply invaluable to the surgeon.

Next Mr. Victor Horsley,² in his observations on a case of thrombosis of the lateral sinus in which recovery took place after septic embolism, suggested the opening of the sinus and the removal of clot after ligature and section of the internal jugular in the neck. This idea was subsequently carried into practice with complete success by Mr. Arbuthnot Lane.³ In 1890 Mr. Ballance⁴ further added to the surgeon's resources by his useful essay on thrombosis of the lateral sinus, with a tabulation of symptoms.

Early in the present year (February 18th) my colleague, Mr. A. J. Pepper, read before this Society an important paper on the graver complications of middle ear disease. Among the contributions to the literature of cerebral abscess, the statistics given by Dr. Newton Pitt, in the *Guy's Hospital Reports*, are of great value.

The anatomical work accomplished with reference to the bearing of aural upon cerebral disease during the past few years by Horsley, Symington, Cunningham, Anderson, Makins, and Müller, have enlarged our acquaintance with cerebral topography. Professor Alec Fraser, in his *Atlas* showing the results of the superposition of photographs has afforded the surgeon weighty assistance in the carrying out of operations on the brain. To the aural surgeon, however, the work done by Professor Ambrose Birmingham is of the highest moment. In his paper on the mastoid region of the skull,⁵ he has dispelled illusions, disproved many old assertions, and established new truths as regards the anatomy of the lateral sinus. Thanks to him, surgeons will now be more than ever careful, when opening the mastoid antrum, to keep close to the border of the meatus.

COMPLICATIONS OF SUPPURATION OF MIDDLE EAR.

The graver complications of middle ear suppuration comprise:

1. Facial paralysis from central or peripheral implication, meningitis or abscess.
2. Suppuration of the mastoid cells and sometimes also of the deeper tissues of the neck, more commonly subperiosteal abscess, and necrosis and sclerosis of the mastoid.
3. Extradural abscess, that is, suppuration between the skull and the dura.
4. Meningitis.
5. Cerebral abscess from direct extension of septic inflammation along veins or lymphatics from the roof of the tympanum, mastoid antrum, or lateral sinus.
6. Thrombosis of the lateral sinus and internal jugular vein.
7. General pyæmia.
8. Marasmus.

The intracranial lesion is usually associated with at least one other complication. In most instances aural suppuration does not early eventuate in intracranial complications. The patient's general health may be excellent provided there is free exit for pus. Caries and necrosis of the temporal bone may go on to an extraordinary extent without causing cerebral mischief.

SYMPTOMS OF CEREBRAL IMPLICATION.

The pathology of brain disease due to otorrhœa is usually quite simple. An outbreak of head symptoms is usually only the final stage in a long series of insidious morbid changes of which the patient himself has been unaware. The mere accumulation of pus within the tympanum may give rise to symptoms simulating those of basal meningitis. The special sign

of actively inflammatory brain lesion from aural suppuration is pain. Almost universally there is dull aching over and radiating from the mastoid region and racking headache. Oscillations between subnormal and high temperature with rigors indicate pyæmia. The temperature in uncomplicated meningitis is continuously high, as also in simple subdural abscess, in which, however, there are modifications according to the complications present. An abrupt fall in temperature after an initial rise signalises the completion of the development of an abscess; the temperature then, as a rule, remains at or below the normal.

Other diagnostic signs of cerebral abscess are slow, full, and even pulse, constipation (as in meningitis), sluggish cerebation, respirations which are rhythmical but less in number and extent, and localised paralysis and convulsive movements. In meningitis there is usually delirium and the pulse is quick, small, and irregular.

Even when the diagnosis is almost certainly that of diffuse meningitis, the penetration of the mastoid cells and tympanic wall and exploratory trephining by Wheeler's operation may be the means of saving life.

CASE OF HYDROPHOBIA FIVE YEARS AFTER HAVING BEEN BITTEN.

By JOHN IRVING, M.B., C.M.GLAS.,
Surgeon to the Huddersfield Infirmary.

H. L., aged 17, was admitted to the Huddersfield Infirmary with symptoms of hydrophobia about midday on September 5th, 1892. He had been bitten near Shepley, a country district, by a mad dog (fox terrier) on August 1st, 1887. A brother and neighbour were bitten at the same time. The bite was on his left hand, his brother being bitten on the wrist and arm more severely. All three went to Paris on August 5th, and stayed there until August 19th, being treated by Pasteur. On October 4th his brother was admitted into the infirmary suffering from hydrophobia, and died on October 5th. His age was 26.

Since the bite H. L. had been perfectly well with the exception of lymphangitis of his left arm two years ago, for which he was treated by Dr. Alister Macgregor, who traced the mischief on that occasion to a deep scratch from a rusty nail; the inflammation soon subsided, although at the time the lad's mother was deeply concerned, thinking it was the result of the dog bite three years previously. His parents stated that he first complained on August 31st of pain about the neck and the back of the head; he thought it was a cold, but as Dr. A. Macgregor, the family medical attendant, has kindly supplied me with an account of the illness previous to his admission to the infirmary, I give it in his own words. He writes:—

I was called to see H. L. on September 4th. On arriving at his house I met his father outside the door, and he said to me that his son was sleeping on the hearthrug, and that I should not waken him, as he had been very violent. He went on to explain that on September 1st H. had complained of a pain in his left shoulder and in his back, which pain steadily grew worse, much to his father's alarm, as his other son F. (who died of hydrophobia five years ago) had been taken the same way. They bathed the shoulder, and the pain in it disappeared on the morning of September 4th, although the pain in the back (where M. Pasteur had inoculated) still remained, though not so severe. During this time the lad seemed out of sorts, but took his food fairly well, and showed no aversion to fluids. On September 4th, at dinner time, he had taken some food, when, on beginning to drink some beer, he was seized with a spasm, and said the beer stuck in his throat, and he could not swallow it. Immediately afterwards he began to look very wild, as the father expressed it. The poor parents were very alarmed, and sent for me. After hearing this history, I entered the house and found the patient lying on the hearthrug, apparently sleeping. I had hardly sat down when he wakened and spoke quite sensibly to me—so sensibly, in fact, that I formed the impression that the parents (who had never really got over the death of the other son) had got needlessly alarmed about him. H. told me that the pain in his shoulder and back had kept him from sleeping for three nights, but that he thought he could sleep a bit now that the pain was rather better. He said he "didn't all so much now." On questioning him about his appetite, he said that some beer he had had at dinner had upset him, so that he could eat no more, but on my asking him if he could drink some milk he said "Oh, yes, I think I can." He had by this time got up and sat in a chair, and when the milk was handed to him he took the mug quite calmly and proceeded to drink it. Hardly, however, had it touched his lips when a violent convulsion seized him in his arms, and with a groan he flung the mug away. I said to him: "Come, now, H., try again and see if you can't drink some." He said "I'll try." Then he braced his shoulders back and made a convulsive grasp at the mug handed to him, but directly he tried to swallow it his eyes gleamed fire

² *Clin. Soc. Trans.*, vol. xix, p. 255.

³ *Ibid.*, vol. xxii, p. 262.

⁴ *Lancet*, vol. i, p. 1116.

⁵ Dublin, 1891.

(exactly like a cat's in the dark); his features worked convulsively; he tried to gulp the milk down, but it all came back; he let the mug drop, and, with a peculiar weird cry, sank back in the chair. He immediately afterwards showed signs of becoming very violent, so I injected half a grain of morphine subcutaneously, and got him put to bed. At midnight (that is, about 8½ hours afterwards) he was sleeping calmly, but about 6 A.M. on September 5th he was seized with convulsions. On my visit at 8 A.M. I again injected half a grain of morphine, which quieted him until he reached the Huddersfield Infirmary. On the journey he seemed quite easy; in fact, on arriving at Huddersfield he said he had had a very pleasant ride. While waiting in the cab at the infirmary gates he said he felt very hungry; but when some beef-tea was offered to him he became violently convulsed. I shall never forget the scene when the poor lad was saying "Goodbye" to his parents, brothers and sisters; it was really most heartrending. He realised fully on Monday morning (September 5th) what was to be his fate; but always previously, his father told me, he had made very light of his dog bite, and said it did not bother him much, so that, in his case at least, morbid glooming was not the exciting cause of the hydrophobia symptoms. The other lad who was bitten by the same mad dog is up to now, I believe, quite well, but, as you may imagine, in a fearful state of mind.

On admission he looked fairly well but rather flushed, and his lips were very dry. He talked rationally, and said that two minutes before the severe attack, on attempting to drink at dinner on the previous day, he had been able to drink quite easily. He was offered some beef-tea, but was seized with convulsions, complained of some oppression in his respiration, and a feeling as if there were a lump in his throat. About half an hour after admission he managed to drink a little milk without much trouble, and to eat a slice of bread and butter, but about four hours afterwards he became restless and nervous, and sat up on the side of his bed. He was still quite rational, but seemed afraid of the spasm coming on. His tongue was dry in the middle, with a little frothy deposit at the edges. Pulse 120, respirations 36. After a hypodermic injection of half a grain of morphine he became quite calm. There was, however, an occasional sudden respiratory spasm, apparently caused by attempting to swallow some saliva. He was ordered to have chloral by the bowel alternately with the hypodermic injections of morphine, and to be fed by enemata of peptonised milk and beef-tea. These were given every four hours. Up till midnight he was fairly quiet but with slight respiratory spasm. At 2 A.M. he became restless, and started up in bed several times, and the spasms became more severe. At 4 A.M. he grew worse, and was shouting, foaming at the mouth, and jumping out of bed. He remained pretty much in this condition until 6 A.M., when he quieted down a little, and an hour and a half later took about a teaspoonful of tea, and liked it, although he drank it with great difficulty. A few minutes later he again became much worse, and shouted, and threw himself about in bed. Spasm became more severe; there was much foaming at the mouth; he was quite unable to drink, and had a severe attack of spasm when the nurse wanted to wash his face. By 9 A.M. the nurse and his two brothers, who were with him during the night, could not control him. He had to be held down in bed, and struggled very violently, at the same time spitting out large quantities of saliva, and rambling and shouting. He was put under the influence of chloroform, and half a grain of morphine was injected, and his hands, shoulders, and feet were secured to the bedstead.

He was kept more or less under the influence of chloroform, and whenever he commenced to come out of it he began struggling and shouting. There were frothing at the mouth, spitting all over the bed and floor, widely dilated pupils, great restlessness, profuse perspiration, violent efforts to get out of bed, vomiting of greenish watery fluid, and some priapism. The saliva was very abundant, and was every now and then sucked into the windpipe, and then violently ejected. At times he would suddenly stiffen the whole body, throw his head backwards and to one side, put his features into a half grin, and scream loudly. His pulse was 136, very feeble, and there were abundant coarse *râles* in the chest. Shortly after 11 A.M., on coming round from the chloroform, he was fairly quiet, but was foaming at the mouth, and vomiting mouthfuls of saliva and mucus continuously, which he spat all round his bed. The respiratory spasm became worse, and caused more sucking in of the saliva into his trachea, and somewhat cyanosed him. The pulse rose to 180. An hour and a-half later he was still very restless. He had intervals of apnoea lasting sometimes about half a minute, when he became rather cyanosed, and then the respiratory spasm came on. He was still vomiting and spitting out large quantities of saliva. He continued to ramble, and threw himself about as much as

the straps would allow him, and became gradually weaker. The respiratory spasm also became worse, and the cyanosis also increased, whilst the vomiting became more frequent. Shortly after 2 P.M. the cyanosis was more or less continuous. The pulse was only just perceptible at the wrist. He was still bringing up mouthfuls of saliva, but did not spit it out, but allowed it to run out of his mouth. He still rambled a little, and moved his head about. At 2.30 he died, little more than forty-eight hours after the commencement of the serious symptoms.

The temperature reached 101.2° F. The specific gravity of the urine was 1025, and it contained a fair quantity of albumen. At the *post-mortem* examination the brain and lungs were found very much congested.

I am indebted to Mr. Cholmeley, Assistant House-Surgeon, for notes of the patient's condition whilst in the infirmary.

MICROSCOPICAL EXAMINATION.

Dr. Sims Woodhead, Director of the Laboratory of the Royal Colleges of Physicians and Surgeons, has made an examination of portions of the central nervous system. Unfortunately, owing to some postal misunderstanding, the specimens only reached his hands after a somewhat long interval. Dr. Woodhead's report is as follows:—

The cord, medulla oblongata, and portion of cerebral cortex was somewhat softened, at one or two points almost creamy; the surface of each piece was blanched, but on making sections at various levels there appeared some evidence of congestion, especially of the grey matter of the piece of the cortex, of the medulla, and of the anterior horns of grey matter of the cord in which the small vessels, especially those running from the pia mater, are considerably engorged. In the floor of the fourth ventricle, just at the lower angle at the entrance of the central canal, there is a very minute hæmorrhage, or what may be mistaken for one, for it is possible that this appearance may be due to the diffusion of pigment from a distended blood vessel into a softened area. The same remark applies to several similar patches found on either side of the raphe in the motor area higher up in the pons. On account of the almost diffident condition of some parts of the specimens, it is difficult to make out anything further. The tissues have since been kept in strong methylated spirit, changed repeatedly in order to harden them as much as possible. Pieces were then embedded in celloidin, and with difficulty a few fairly good but thick specimens of the medulla and pons at various levels and of the cortex cerebri have been obtained. These stained in Ehrlich's hæmatoxylin and then with benzo-purpurin, cleared up with clove oil and mounted in xylol balsam, show the following appearances.

In the medulla and pons, beyond extreme granularity of the cells of the olive and of the ganglion cells of the nuclei, some congestion and dilatation of the smaller vessels, slight distension of the perivascular spaces in which a few leucocytes could be found, there is nothing abnormal. There is no evidence of hæmorrhage near the median line (mentioned above), though there are localised patches in which the congestion appears to be much more marked than on the general surface. In the piece of the cortex there are far more evident changes. The vessels of the pia mater are distended, though here and there the spirit seems to have caused them to contract and so to drive out the blood. In and around the larger vessels, both those near the surface and those in the deeper layer, there are marked evidences of acute inflammation. Attention is at once drawn to large accumulations of leucocytes which occur just at the points of bifurcation, especially of the veins. The perivascular lymph spaces are considerably distended, even when they are not filled with leucocytes. At one or two points where putrefaction changes have set in, it appears as though some of the vessels had actually given way, but it is impossible to determine this with any degree of accuracy. Here, as in so many cases of hydrophobia, the histological changes in the nerve centres are little characteristic of the disease. I have seen much the same appearances as above described in cases of acute mania, in "acute congestion" of the brain, and in a case of acute tetanus, so that I am afraid there is little here to help to make a more accurate diagnosis than has already been able to be made from the clinical history of the case.

MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, Etc.

FOREIGN BODIES IN THE LUNG.

MR. W. J. MIDDLETON, in the BRITISH MEDICAL JOURNAL of November 26th, describes two very interesting cases of foreign body in the lung where the foreign body was expelled, in one case nine months, and in the other twelve months, after its introduction, and without resort to any surgical procedure.

When a foreign body has entered the trachea or has become impacted in a bronchus, whether the resulting symptoms be trivial and occasional only, or whether they be severe and persistent, that patient is liable at any moment to suffocation by impaction of the body in the larynx during a strong expiratory effort, as in coughing. Where such an accident does not occur, can we say that the lung will remain healthy? The re-