about 3 ounces of blood-stained urine, and complained of a good deal of pain. During the night he suffered from a desire to micturate about every half hour.

December 9th. In his attempts to micturate he managed to pull his catheter out, which was not replaced, and he has been more comfortable ever since.

December 10th. Slept much better and feels easier. Says he has not passed water so easily for years.

December 11th. A large slough, measuring nearly $\frac{1}{2}$ inch in length, has passed with his urine, which, with the exception of the sloughs, is quite clear, acid, 1020, no albumen.

December 17th. Catheter passed after he had micturated, and there was no residual urine whatever.

December 18th. The prolapsed rectum and piles were operated on, a cautery being used to the mucous membrane.

December 26th. Had to have his water drawn off.

January 7th. Now perfectly well. Passes his water without any difficulty, and has no residual urine. Piles and prolapsus are also well.

November 9th, 1892. Remains well.

From the notes which have just been read it will be seen that the operation in question is one which has, so far, yielded most excellent results in a class of case which is usually amongst the most intractable in surgery so far as cure is concerned. The only one of the four cases in which the temperature after the operation rose as high as 100.5° , or gave one the slightest momentary anxiety, was the first, in which case also it will be remembered that there was an abscess formed in connection with the testicle, thus unduly prolonging the period of convalescence. To a large extent, if not entirely, this was, in my opinion, due to the retention of the catheter for a longer period than forty-eight hours. It is the practice of Bottini to insist on this plan of procedure, but such retention is, I am convinced, both unnecessary and undesirable, as is shown by the more favourable course which the other cases have taken. As far as the results are concerned, three out of the four cases now pass the whole of their urine without catheter, and experience no difficulty whatever in its passage, whilst the fourth, which was the most severe case of them all, is so far improved that he can urinate when standing up, which he had been unable to accomplish for some year and more, as well as having much less residual urine in his bladder than was formerly the case.

RETINAL AFFECTIONS OF CHRONIC BRIGHT'S DISEASE AND DIABETES.

Being an Abstract of the Middlemore Lectures delivered at the Birmingham and Midland Counties Eye Hospital.

By ROBERT SAUNDY, M.D., F.R.C.P.,

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LECTURE I.

THE following classification of the retinal affections of Bright's disease formed the basis for the descriptions given by the lecturer:—

1. Diffused opacity.
2. White patches: (a) Rounded soft-edged areas generally situated near the disc, and (b) smaller, brighter, often radiated specks generally near the yellow spot.
3. Hæmorrhages.
4. Optic papillitis.
5. Diffuse retinitis.
6. Secondary atrophic changes.
7. General retinal periarteritis.

General retinal periarteritis was included in this classification because although not solely, it was usually associated with Bright's disease, and had therefore as much claim to be regarded as one of the retinal affections met with in Bright's disease as any of the forms more usually recognised.

The pathological explanation of all these changes was sought in the dyscrasia, which set up, first nutritive disturbances and, secondly, inflammatory and degenerative changes in the tissues. The minute histological details of each were fully described. The course of the inflammatory lesions was shown to be not necessarily hopeless of cure, though more or less permanent structural change might remain; even the degenerative spots sometimes disappeared; cases illustrating these facts were quoted. The difficulty of assigning any absolute diagnostic value to these appearances was pointed out and illustrated by drawings and cases.

On the other hand the prognostic meaning of the degenerative white specks (b), of diffuse neuro-retinitis, and of general periarteritis in otherwise well-assured cases of Bright's disease, was well defined and of the most serious character. The lecture was illustrated by a number of diagrams and by microscopical preparations kindly lent by Mr. E. Treacher Collins, Mr. Lawford, and Mr. Priestley Smith.

LECTURE II.

After a brief historical introduction the lecturer described the retinal affections of diabetes in accordance with Hirschberg's recent classification.

1. Retinitis centralis punctata diabetica, in which there is a characteristic inflammation of the central portion of the retina, giving rise to the appearance of bright spots often coalescing, and frequently accompanied by hæmorrhages.

2. Retinitis hæmorrhagica diabetica, in which the essential process is hæmorrhage followed by inflammatory and degenerative changes.

3. Mixed cases.

The disease in all its forms occurs only in persons of middle or advanced life, and in cases of diabetes of some standing. It is generally bilateral and causes disturbance of vision, the patients complaining of a mist before their eyes. In the first form the ophthalmoscope reveals groups of small, clear, bright specks situated in the structure of the retina, in and around the disc, occurring on the nasal as well as the temporal side, never radiated in arrangement but often occupying large areas, never pigmented but accompanied by fine punctiform hæmorrhages. It is never attended by optic papillitis or diffuse retinitis. Newly formed blood vessels have been observed, and there is a decided tendency to the occurrence of large hæmorrhages into the vitreous. This description was illustrated by a case, and the fundus oculi shown by means of Mr. Priestley Smith's demonstrating ophthalmoscope.

The pathology of the affection was held to be dependent on the diabetic dyscrasia which leads to nutritive changes in the walls of the vessels, and to the effusion of albuminous material into and between the layers of the retina. In the second form the hæmorrhages are usually punctiform, but may be striated, and are situated all over the retina; their source is not the superficial retinal vessels, and their rounded shape indicates that they are situated below the nerve fibre layer. There may be haziness of the retina, depending on some degree of œdema. The hæmorrhages are due to vascular degeneration caused doubtless by the dyscrasia, but the histological details have not been worked out. The course of both is progressive, and no instance of recovery has been recorded, although temporary improvement in vision occasionally takes place.

The first form is highly characteristic when well marked, but the diagnosis should always be confirmed by examining the urine. Little is known definitely as to the prognostic importance of either, but on general grounds it is probable that they indicate an advanced degree of those nutritive derangements which herald a fatal termination.

This lecture was illustrated by drawings and microscopical sections, the latter being kindly lent for the purpose by Dr. Stephen Mackenzie.

THE PATHOLOGY AND TREATMENT

OF

SUPPURATIVE DISEASES OF THE EAR.

Being an abstract of the Harveian Lectures delivered before the Harveian Society of London.

By G. P. FIELD, M.R.C.S.,

Aural Surgeon to St. Mary's Hospital and Dean of the Medical School.

LECTURE III.—INTRACRANIAL ABSCESS.

A NEW epoch in brain surgery was inaugurated by the publication of Mr. Hulke's Remarks on Trephining for Evacuation of Intracranial Abscess in Connection with Suppurations in the Ear.¹ His account therein of three typical cases will ever be prominent in the literature of middle ear disease. It was Mr. Hulke who first laid stress on the frequent occur-

¹ *Lancet*, July 3rd, 1886.

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.