THE
GOULSTONIAN LECTURES
ON
SOME CEREBRAL LESIONS.

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LECTURE I.

AN ANALYSIS OF FIFTY-SEVEN FATAL CASES OF EAR DISEASE,
AND OF THE COMPLICATIONS WHICH LED TO DEATH.

OTORRHOEA is but too frequently looked upon as a troublesome complaint that must be endured, and when patients have had a discharge which has persisted for many years in spite of all treatment, they, at any rate, if not their medical attendant as well, are apt to consider that they have done with it, that it is hopeless for them to trouble any more about it, and that it will never alter. Yet there can be no greater mistake, for occasionally, under some exciting condition, the inflammation spreads to the cranial cavity, the patient rapidly becomes mortally ill, and in many instances dies after an illness of a few days, or it may be some weeks', duration.

The unexpected onset of grave symptoms, the agonising headache which too often forms one of them, the state of mental torpor or coma into which so many of the patients pass, and the utter uselessness of medical treatment in the more severe cases, must on various occasions have impressed themselves deeply on everyone here.

Of late years improved methods of surgery, and especially the more effective means that are adopted to sterilise foul and septic centres of infection, have led, in a few instances, to more successful results. Now that attention is being directed to the matter, we may look for a much greater measure of success; and here, as has so frequently been the case, the pathologist is able to indicate to the surgeon the lines on which the treatment should be laid down.

For my purpose I have examined the *post-mortem* records at Guy's Hospital for the years 1869 to 1888, a period of twenty years, and have found 57 inspections in which ear trouble has set up disease in the cranial cavity, which ultimately proved fatal. I desire to express my appreciation of the kindness of my colleagues in placing their cases at my disposal, and thus providing me with a large number of valuable records, the majority of which have not been previously published. I am more particularly indebted to Dr. Goodhart, who made a large number of the *post-mortem* examinations. During this period there were nearly 9,000 inspections. Death was, therefore, traceable to ear disease in 0.66 per cent. of the cases. Further examination shows that 27 of the cases had occurred during the last four years, which would raise the ratio for that period to 2 per cent. I am quite unable to offer any explanation of this very greatly increased frequency. An examination of the reports fails to throw any light on the matter, and there is no reason to suppose that it was due to any great difference in the treatment.

No case of simple otitis media, with or without disease of the mastoid cells, was fatal during the whole of this period; and only twice was the complication that led to the fatal result outside the cranial cavity, and even then it was in contiguity with it.

In one there was a retropharyngeal abscess, and in the other the patient bled to death from rupture of the internal carotid artery. Death from ulceration of the carotid is a very rare event, only fourteen cases having been recorded, according to Politzer.

It is worth remembering that when pyæmia occurred in ear disease, the onset was preceded in all cases by thrombosis of the lateral sinus. The difficulties of diagnosis in ear disease are much increased by the fact that, not only may a patient die without any otorrhœa having been noticed (this occurred in nine, that is, in one-sixth of the cases), but in two instances the membrana tympani was found intact at the inspection. These patients died with meningitis and lateral sinus thrombosis respectively. The possibility of the otorrhœa being secondary to the meningitis may be the explanation of these cases.

Of the 57 cases, 34 were males and 23 females. The right ear was the source of the mischief rather less frequently than the left. Seventeen of the patients were under 10, the same number between that age and 20; 14 in the next decennial period, and only 9 over 30. The only patients over 50 were the man aged 65, who died from hæmorrhage from ulceration of the carotid, and a woman aged 66, who died with meningitis. Three of the 4 babies who died under 3 years of age succumbed from meningitis.

The acute symptoms in ear disease appear sometimes to come on spontaneously (24); at other times they have followed exposure to cold (4), a blow on the ear (6), mastoid suppuration (13), the introduction of foreign bodies into the external meatus (3), or the removal of a polypus (1). Any of these causes may start acute inflammation in the mastoid cells, or antrum, or the middle ear; especially if the ear be already diseased. Rapid swelling of the mucous membrane takes place, and a discharge of sero-pus or pus ensues from the surface. Should there be a sufficient outlet, this is evidenced by a great increase in the otorrhœa; but when the swollen mucous membrane blocks up the outlet, the chronic external discharge will cease entirely. This cessation is really a symptom of the acute inflammation, and not a cause thereof, as has been sometimes assumed. Every accession of fresh symptoms is due to the imperfect drainage of the contents of the middle ear, the mastoid antrum, or of the mastoid cells. The discharge was increased in 13 cases, and absent in 10.

As long as no pus is pent up under tension no evil symptoms ensue, but with an increase of tension, earache and a more or less severe headache will develop. With the retention of pus in the petrous bone, or in the mastoid cells or antrum, patients may suffer intense pain, their temperature may be high, and they appear extremely ill, but only those cases proved fatal in which the inflammation spread outside the petrous bone. The majority of these cases, if they are freely drained by opening up the mastoid cells, recover and lose their severe symptoms, and many of them ultimately discharge their pus externally without surgical aid; but the risk is diminished by skilful surgical interference.

Toynbee, in his papers in 1851 and again in 1855, attempted to prove that the seat of the complications could be determined to a great extent by the spot in which the pus was collected in or about the petrous bone. His careful papers did much to elucidate the matter, but it is generally held that further facts have not corroborated his conclusions. He thought that (1) affections of the external meatus and mastoid cells produce disease in the lateral sinus and cerebellum, (2) affections of the tympanic cavity produce disease in the cerebrum, (3) affections of the vestibule and cochlea produce disease in the medulla oblongata. As is shown later on, thrombosis of the lateral sinus often has originated from caries of the posterior wall of the tympanic cavity, and mastoid disease sometimes spreads to the middle fossa of the skull; still the usual sequence is that indicated by Toynbee. Disease of the internal ear appears usually to set up meningitis in the posterior fossa of the skull.

In an adult the mastoid cells may be divided into two groups, a set arranged vertically, which form the mastoid process and which do not exist during the first years of life, and another lying

between the mastoid and the tympanum, arranged horizontally. During the two first years of life even these horizontal cells are but little developed, and the external meatus is separated from the middle fossa of the skull by a lamina of bone, continuous with the roof of the tympanum, which is often extremely thin, and is liable at this age to share in any attack of inflammation affecting the mastoid cells. At a later age the layer of bone separating these parts from the middle fossa is often more than a twentieth of an inch thick. Toynbee, therefore, thought that disease of the mastoid cells at an early age produced disease in the middle fossa, while later in life the disease spread to the lateral sinus. In the cases of mastoid disease which were examined, it was found that the lateral sinus was thrombosed in two children under 10 years of age, whereas in eleven patients above 10 the lateral sinus was thrombosed in 6, there was a temporo-sphenoidal abscess in 4, and a cerebellar abscess in 2.

The roof of the tympanum is formed by an elongated plate of bone, the thickness of which varies so much even in health that it may be dense, and a line in thickness, or a mere shell of translucent bone, parts of which are often deficient. The experience of the *post-mortem* room shows that the situation of the groove for the lateral sinus is liable to great variation, and that it may excavate the bone deeply, so as to be separated from the mastoid cells and the cavity of the middle ear by a thin layer of bone, in which lacunæ may be found when there has been old-standing caries, but rarely otherwise. When, in trephining, the lateral sinus is found more anterior than usual, it suggests that the posterior wall of the middle ear and the inner wall of the mastoid cells are thinner than normal, and that therefore disease is more likely to have spread into the posterior fossa rather than forwards. The development of the mastoid cells and the density of the bone round them are equally variable.

The anatomical and *post-mortem* evidence therefore lead me to the conclusion that the condition of the mastoid cells and of the roof of the tympanum, and the situation of the lateral sinus, play the most important part in determining the direction in which the disease shall spread; and that too great stress should not be laid on the presence or absence of disease of the mastoid, although it may be somewhat of a guide to the seat of the mischief.

As far as can be judged from our records, nothing can be learnt which will be of assistance in diagnosis, from the condition of the wall of the middle ear. There was no complication which was not in some instances accompanied by caries or necrosis. The most convenient arrangement of the complications to be discussed will be according to their site: 1, dura mater; 2, cerebral tissue; 3, sinuses; 4, pia-arachnoid.

Owing to the thinness of the roof of the tympanum, the dura mater over the anterior surface of the petrous bone is rather more often inflamed (in 16) than that over the posterior wall of the middle ear, but less often if we include the part bounding the mastoid cells as well (18). The parts may be discoloured merely, or they may be sloughing and fetid, or pus may collect beneath the membrane, whence it may rupture into the arachnoid cavity, setting up a meningitis (3), or it may be let out by operation.

In these cases the otorrhœa is of old standing, and generally the bone beneath is inflamed, discoloured, carious or necrosed, and in some of the cases caries had produced fair-sized apertures in the bone, through which the infection had spread directly.

I wish to emphasise the occurrence of inflammation or sloughing of the dura mater over the roof of the tympanum, which was present in 10 out of the 12 cases of temporo-sphenoidal abscess, and probably 7 of these could not have recovered unless the dura mater as well as the abscess could have been allowed to drain. In extradural abscesses the inflammation has probably spread along the lymphatics round the veins. An extradural abscess may produce optic neuritis, as is proved by the following case which was under Mr. Lane's care:

Optic Neuritis due to Extradural Abscess.—E. T., aged 12, under Mr. Lane. October 4th, 1888. Received a blow on her ear five years ago; no history of otorrhœa till four weeks ago, after a cold. Headache on September 28th. Drowsy on October 2nd. On admission, October 4th, double optic neuritis, headache, retraction of head, vomiting. Trephined at once; on right side half an inch of bone was removed above posterior root of zygoma, the anterior surface of the petrous bone was exposed, and some drops of offensive pus let out. Temporo-sphenoidal lobe was explored with a needle, without result. On gouging further back the lateral sinus was exposed, the dura mater was found separated from the bone, and $1\frac{1}{2}$ drachm of foul pus welled out. The mastoid process which contained pus had been previously gouged so as to communicate with the middle ear, and a few drops of pus near the sinus let out. The wound was frequently syringed out and dressed antiseptically. October 8th. Temperature 105° ; pulse 165; drowsy. She improved very much in every way; at first the optic neuritis diminished; still headache and drowsiness increased till her death, on October 14th. *Post mortem* there was abundant pus in the posterior fossa bathing the cerebellum. The brain, etc., normal. No pus between the dura mater and petrous bone, which was necrosed. The softened dura mater behind the petrous bone was perforated, and the abscess had discharged on to the surface of the brain.

The neuritis cleared up considerably after the operation, and the meningitis from which the child died only came on at the end.

Besides this case, which was verified *post mortem*, we have had other cases of otorrhœa with optic neuritis which have recovered; one under Dr. Moxon's care some years ago recovered without operation, the three following recovered after trephining.

Mastoid Suppuration; Optic Neuritis; Trephining; Recovery.—A. K., aged 13, under Mr. Bryant in 1872. Two years' right otorrhœa; two weeks' earache; one week mastoid pain; failure of sight in the right eye. Admitted with bleeding from the right ear, with right optic neuritis, drowsiness, and drawing speech. The mastoid was trephined. Four days later intense optic neuritis, blind with the right eye, perception of light with the left. The right pupil was dilated and insensitve. After a fortnight an abscess broke at the back of the ear. Almost blind. Two weeks later she could see objects, and after a time the sight improved somewhat. The local trouble disappearing.

Mastoid Suppuration; Optic Neuritis; Trephining; Recovery.—E. B., aged 20, under Dr. Pavy. October 10th, 1885. For the last month she has had earache, for two weeks severe pain in her neck; she often screams with pain. She has lost flesh considerably. On admission she was constantly moaning; takes but little notice of anything; answers questions slowly. On the 13th the mastoid was trephined; $\frac{5}{16}$ of foul pus let out. The pain at once decreased. The temperature for the next three weeks was 98° to 103° . Well-marked optic neuritis. On November 14th an abscess was opened below the mastoid. On the 19th another abscess was opened, and again on December 2nd. She left on January 8th, after an attack of erysipelas, feeling quite well.

Mastoid Suppuration; Optic Neuritis; Recovery.—G. W., aged 13, under Dr. Taylor. February 3rd, 1887. After exposure to cold sixteen days ago he had earache, followed by otorrhœa in four days' time. He has been laid up for the last ten days with pain in the head and neck, with rigors. The discharge is offensive, and there is slight œdema over the mastoid. Temperature 104.6° . Early optic neuritis, best marked in the left eye. Mr. Jacobson trephined, and let out some offensive pus from the mastoid cells. By March 2nd the discharge was sweet, and the operation opening was almost healed. Temperature normal. Soon after the operation the optic discs had reached a stage of pronounced neuritis, and they have not varied materially since. He went home again feeling quite well. In May, 1888, the discs were still swollen, but in June they had cleared up; the outline was sharply defined, and the veins were not tortuous. It is difficult to be certain what was the lesion; possibly a pachymeningitis, or even a thrombosis of the sinus, with certainly retention of pus in the bone. It is improbable that the sinus was thrombosed, as with so offensive a discharge it is unlikely that the mischief would have cleared up at once as soon as the ear was drained. I incline to the view that the optic neuritis was due to the condition of the dura mater. This was certainly the explanation in the first, and very probably in the other two.

Of 18 cases of cerebral abscess, 9 occurred on each side of the brain, and the preponderance which Gull and Sutton found in their

24 cases in favour of the right side of the brain was probably accidental. In the cases under consideration, 11 of the patients were men, and 13 of the cases occurred between the ages of 10 and 29; the only case over 40 being one of pontic abscess. Only twice had the otorrhœa lasted less than a year, and in these the duration was seven and four months respectively. This agrees with the general experience that otorrhœa does not set up cerebral abscess until it has lasted months or years.

Three of the abscesses were in the cerebellum, one in the pons, two in the centrum ovale, and the remaining twelve in the temporo-sphenoidal lobes, within a very short distance of the roof of the tympanum.

In only 2 of these 12 cases of temporo-sphenoidal abscess was the dura mater over the anterior surface of the petrous bone healthy, in 8 it was sloughing, in 2 inflamed, and in 1 there was a localised extradural abscess. On two occasions the dura mater on the posterior surface was also diseased.

The exact mode in which the brain becomes infected in these temporo-sphenoidal cases has long been a moot point. Where there is healthy brain tissue between the abscess and the bone it is probable that the infection has been spread by the veins which empty into the superior petrosal sinus, from the temporo-sphenoidal lobe on the one hand, and the tympanum on the other, by means of a septic phlebitis, or more probably by means of the perivascular lymphatics. Other veins run direct from the brain into the dura mater over the petrous bone. It is worthy of note, however, that in not a single case is there a statement that the superior petrosal sinus was thrombosed; it was certainly examined in many of the cases, though possibly not in all. If the mischief had usually spread by the veins and not by the lymphatics, we should have expected thrombosis to have been occasionally noticed. Frequently the brain adheres to the anterior surface of the petrous bone, over which the dura mater is inflamed, and thence infection spreads by contact to the cerebral tissue.

The two abscesses in the centrum ovale originated the one from thrombosis of a vein from the Sylvian fissure to the dura mater, and the other from pyæmic infection due to an old septic thrombus in the lateral sinus on the opposite side. An extradural abscess behind the petrous bone started one of the cerebellar abscesses, and another had originated near the torcular Herophili from an old thrombus in the lateral sinus.

Only five of the abscesses were less than an inch in diameter, while ten exceeded two inches. Death was preceded by coma in the majority, and in seven cases the duration of the coma varied from a few hours to three days, and in only three instances was this limit exceeded.

The symptoms of cerebral abscess are often very vague for a considerable time, which may be called the latent period, the duration of which is very variable. In one instance the symptoms had lasted over a year; in three over two months; in nine over one month, and in five under a month. During this period headache (15), vomiting (10), and a slow, dull, mental condition are usually present. Among the other symptoms which were noted were restlessness (4), lethargy, drowsiness (5), irritability, delirium (2), convulsions (4), earache (4), emaciation (2), noisiness (2), and slow cerebration, the patients often taking a long time before they replied to questions. The headache is usually only of moderate severity, and the vomiting is occasional and causeless. In only four cases were rigors noticed; in two they were probably due to an accompanying lateral sinus thrombosis. The other cases were complicated by broncho-pneumonia and an extradural abscess. It would therefore appear that rigors are infrequent with cerebral abscesses, and are rarely directly due to them. Among the symptoms which are of great significance when present with old disease of the left ear are failure of memory and aphasia (2); and with disease of either ear, paresis of the face (2), or of the face and arm (2), or tremors and twitchings of the hand. The aphasic condition, when present, was characterised by the loss of some words, and was probably directly due to the destruction of the temporo-sphenoidal auditory centre, producing word-blindness.

In only five (28 per cent.) of the cases was there an accompanying meningitis, and in each instance it was secondary apparently to the abscess. One abscess had invaded the cortex, two had burst on to the surface (one of them the day after an exploratory operation), in a fourth the meningitis followed the drainage of a fetid abscess, and in the last case it was limited to the base and was probably secondary to the discharge of the contents of the abscess into the ventricle. Hence in none of the cases did the meningitis appear to have spread directly from the petrous bone. Only three

times was there more than one abscess; in two of these instances there were one and two adjacent secondary abscesses respectively; and the remaining case, which alone of all these five abscesses lay on the opposite side to the affected ear, consisted of two adjacent abscesses, which appeared to be secondary to a thrombus of some date in the opposite lateral sinus. Four times (22 per cent.) there was suppuration about the mastoid. In none were there any pulmonary abscesses or a general pyæmia. Once there was a recent sinus thrombosis, and twice old clots of seven and twelve months' date, in the lateral sinus. When the abscess was situated rather higher in the centrum ovale (twice) there were attacks of tremors, with loss of power in the right arm, without loss of consciousness, but associated with facial paralysis in the one case, and with paresis of the face and arm in the other. Facial paralysis; was caused in one patient by an extradural abscess round the internal auditory meatus, and associated with crossed hemiplegia, in another patient it was due to an abscess in the pons.

In seven cases, in which a note of the condition of the optic discs was made, they were found swollen in two, in which the abscesses were large; there was optic neuritis in two (both having also old sinus thrombosis, which was probably the cause of it), and in three they were normal. In all but two the patients were fully examined, and probably therefore in the majority of the remaining cases, in which no note is made of the condition of the discs, they were normal. It would appear that changes in the optic discs are not frequent, and that when present they are often not advanced.

Pflüger has once recorded optic neuritis in connection with a cerebellar abscess. Neuritis was present in one case, but was very probably due to the sinus thrombosis.

Lateral Sinus Thrombosis; Cerebellar Abscess; Optic Neuritis.—J. S., aged 23; under Dr. Fagge, 1878. A year ago the patient was weak and ill, giddy, and had occipital headaches; otorrhœa since childhood, dragging left leg in walking; rigors for six months. Five weeks ago he lost the use of his legs; he sweats profusely. October 2nd. Right otorrhœa. Incisions made over the right mastoid. Double optic neuritis. Temperature 98° to 105° up to November 1st; normal later; relieved by quinine. Rigors, vomiting for last fortnight of life, but feeling fairly well. On December 22nd he suddenly woke up with a scream, became collapsed, and died four hours later. Severe pains at the back of the neck if moved. *Post mortem:* Healed scar over occipital bone external to the lateral sinus; the dura mater and bone for an inch and a half near the torcular Herophili were thickened and inflamed; a dense decolorised adherent clot extended from torcular in right sinus for half way; a second less dense decolorised clot extended in outer part, leaving a space between; the external surface of the skull was healthy. The right tympanum contained pus; the left very indurated roof, but free from pus. An abscess three-quarters of an inch in diameter, filled with greenish pus, was situated in the upper part of the right posterior or semilunar lobe of the cerebellum; the surface tore on removal where it was adherent to the right side of the torcular Herophili. The cerebellar abscess was probably secondary to the lateral sinus thrombosis.

After a variable period, the acute stage is entered upon, which may last less than a week. At this period an agonising headache was the most marked symptom in nine of the patients, but was not noticed when the patient was very lethargic. The patient may scream with agony, may rock himself to and fro, holding his head between his hands; he may be considered hysterical, or he may bury his head in the pillow and pay no attention to what is going on. A state of stupor passing into coma is not infrequent, and is generally fatal in less than three days, but in some the sudden onset of coma may be the first indication that the illness is a serious one.

In some of the cases in which the pain was agonising during life, it was found at the inspection that the abscess had burst into the ventricle (5); in two others the lateral ventricles contained turbid fluid, but the pus was separated by a thin layer of cerebral tissue from the cavity.

The temperature is rarely high with uncomplicated cerebral abscess. It was not above the normal in six cases; in eight it was high; three of these had meningitis, two thrombosis of the lateral sinus, and in one of the others it was noted that the dura mater was thickened and greenish, and in another it was black and sloughing, with a collection of cheesy pus outside it.

In only one or two, therefore, was the pyrexia due to the cerebral abscess. The pulse is often increased in rate, but when the

abscess is large the pulse may become slow and irregular. The dura mater, especially in the region of the fifth nerve, is acutely sensitive when inflamed; hence the intense severity of the headaches associated with abscess in connection with ear disease. Tenderness of the scalp was not especially noted in these cases. Death was due either to coma, from the pressure of the abscess; to meningitis, from the pus reaching the surface of the brain; or to sudden respiratory failure.

The three cerebellar abscesses presented no very characteristic symptoms; one patient died the day he came up; another man having had thrombosis of a sinus for a year, developed further clotting in the vessel, and after ten weeks died suddenly; but the symptoms all through were referable rather to this mischief than to the abscess; in the third case the symptoms were those of abscess, but there were no symptoms which indicated the locality of the pus, except that there was a partial facial paralysis (5).

One Case of Abscess of the Pons.—M. T., 46, under Dr. Taylor. June 7th. Partial left hemiplegia. Three weeks ago her face was drawn to the left. She suddenly lost the use of her hands. June 12th. Paralysis of the right side of the face, weakness of the left leg, gait drunken, right otorrhoea. The left hemiplegia gradually increased till her death, on July 2nd. Optic disc normal. *Post mortem:* Foul greenish pus in the right tympanum. An abscess was found one inch by three-quarters of an inch on the right side of the pons, towards its upper part, occupying the motor tract; it had thick greenish walls, with acute softening round it; the inflammation was diffuse.

There is no evidence, pathological or other, that these cases ever recover without the aid of a surgeon; and although a few successful cases have been drained, even now almost every case is fatal.

The following conclusions may be drawn from the present series of cases:—

- (1). Abscesses in the temporo-sphenoidal lobe, which is by far the most common situation, are often associated with an inflamed or sloughing dura mater over the anterior surface of the petrous bone, or with a collection of pus beneath it.
- (2). Other complications are infrequent, except meningitis, which is generally due to the extension or to the rupture of the abscess.
- (3). These abscesses are almost always situated very close to the roof of the tympanum.
- (4). A foul discharge is often a source of danger, and frequently, if not invariably, the spread of the mischief is due to imperfect drainage of the middle ear.
- (5). Mastoid suppuration often infects the posterior surface of the petrous bone, but it may be associated with disease limited to the middle fossa of the skull.
- (6). Cerebral abscesses only occur when the otorrhoea has lasted for months or years.
- (7). The symptoms usually come on insidiously.
- (8). Rigors, pyrexia, and optic neuritis are all infrequent in uncomplicated cases, but they all occur occasionally.
- (9). A headache of intense severity, and a dull, sluggish mental state are the two most characteristic symptoms.
- (10). Cerebellar abscesses are less common, and will probably be associated with disease of the dura mater behind the petrous bone, or with thrombosis of the sinus.

The objects to be aimed at in treatment are:

a. In every case to improve the drainage of the ear, by gouging a way or trephining the mastoid sufficiently to open up the horizontal cells or antrum, where pus is often found, and to break a hole through the deeper part of the posterior wall of the external meatus, so as to allow no secretion to be retained. The cavity should be rendered sweet and aseptic as soon as possible. In a case of otitis media it is often desirable to carry out this treatment as soon as there is evidence of a fresh accession of severe mischief; should further exploration be necessary later on, the risk of infection from the septic otorrhoea will be very much reduced. It is always desirable that the external ear should be dressed apart from the other openings, if any are made.

b. To expose the anterior surface of the petrous bone, so as to allow free drainage for any pus or debris which may have formed in connection with the dura mater, which is often inflamed or gangrenous. This is best reached at a point half an inch above the anterior margin of the external meatus. Should there be any pus retained, some will often be found in the diploë of the bone removed, in which case the bone should be broken away to a quarter of an inch above, and just in front of, the meatus, so as to expose the most dependent part of the anterior surface.

c. To drain the abscess from below when possible. Messrs. Horsley, Macewen, Barker, Bergmann, and others, have discussed the best methods of attaining this result. In the case of a temporo-sphenoidal abscess, the area beneath which the pus will almost universally be found may be said to be bounded anteriorly and posteriorly by curved lines drawn through the temporo-maxillary joint and the middle of the mastoid, running at right angles to the sagittal suture, and to extend from half an inch to 2 inches above the meatus. The lower part of this area should, therefore, be explored with trocar and cannula, after breaking the bone away or trephining a fresh hole, and opening the dura mater, unless special symptoms indicate that the abscess is higher up. If the attempt to find pus is unsuccessful, the lateral sinus should be exposed and examined half an inch directly behind the meatus; if necessary the bone may be further broken away, and the outer and under part of the cerebellum explored for abscess.

By this method all the seats where pus is likely to accumulate can be systematically examined, and we give the patient the best chance. It is necessary to examine all these seats in doubtful cases, because, although in some uncomplicated cases we may be able to determine the lesion fairly definitely, yet, where two or more lesions are combined, the uncertainty in the diagnosis is so great that the best method is to explore all probable spots.

Thrombosis of the Lateral Sinus.—This occurred twenty-two times. The condition both of the wall of the vein and of its contents varied. In some there was well-marked phlebitis, but not in all. In considerably more than half the thrombus was suppurating; but several of those in which the clot showed no evidence of breaking down had set up a pulmonary pyæmia, demonstrating that they were none the less septic, and would probably have broken down if the patient had lived longer. The thrombosis had spread in nine cases directly from inflammation or necrosis of the posterior wall of the petrous bone, the dura mater over it being inflamed or sloughing; in three there was a collection of pus outside. In the remainder infection had spread from disease of the mastoid cells or of the posterior wall of the tympanum by means of the veins which empty into the sinus—for example, the vein from the cochlea—without having infected the dura mater.

This fact that in almost half of the cases of thrombosed sinus the disease had spread directly from caries or necrosis of the posterior wall of the tympanum is of the greatest importance; in one case there was an aperture bringing the middle ear directly into contact with the wall of the lateral sinus; in others suppuration had already occurred on the surface of the bone. If any treatment is to be successful it must deal with the condition of the bone and dura mater as well as with the sinus.

It is obvious that in these cases the thickness and density of the lamina of bone separating the middle ear and the mastoid cells from the sinus is a main factor in determining whether the disease shall spread backwards or not. In only one-third of the cases of sinus thrombosis did we find that the disease had originated in the mastoid cells; and, on the other hand, in some cases pus was found in the mastoid cells, but the cerebral disease was limited to the middle fossa (six times). Still, the rule holds good that whenever the mastoid vein—which perforates an inch and a quarter behind the meatus, on a level with it—is found thrombosed, the sinus should always be explored.

The thrombus may be a small one consisting of only a few clots, or it may occupy the whole of the sinus, and in 11 cases it extended into the internal jugular vein, in all of which there was also pulmonary pyæmia, and in only 4 did this occur with a healthy jugular vein. The clotting four times extended into the longitudinal and three times into the opposite lateral sinus. In one instance very extensive clotting was found throughout the lateral, both the petrosal, the cavernous, and the circular sinuses, and the jugular vein; in another, in the ophthalmic vein. Once clotting occurred in the cerebral veins, and another time in a vein to the tentorium, which set up meningitis. Four times suppuration took place round the jugular vein.

In over 70 per cent. of these cases the mode of death was by pulmonary pyæmia (abscesses in 15, empyema alone in 1). Two of these had also general pyæmia, 3 meningitis, and 1 a cerebellar abscess. The causes of death in the remaining 6 were: general pyæmia, 2; meningitis, 2; retropharyngeal abscess, 1; and cerebellar abscess, 1.

All these complications were directly due to the thrombosis in the sinus, with the exception of the retropharyngeal abscess and four of the cases of meningitis, of which two had spread from the dura

mater and the third through the internal auditory meatus, and probably the fourth also. Thrombosis is, therefore, a very fatal lesion, but there is some evidence that patients with the typical symptoms appear to recover, at any rate for a time; and among these cases are 3 which showed that the patients had recovered—only, however, to succumb to the secondary effects after intervals of a year, six and two months.

In the first a man, aged 23, with old otorrhoea, had been ill, giddy, and had had occipital headaches a year previously, his left leg had dragged in walking, and for the last six months he had rigors. He sweated profusely. He died with a cerebellar abscess, which was secondary to an old thrombus, which was adherent and decolourised, occupying the inner part of the sinus. A second clot of more recent date was in the outer part of the sinus.

The second was a girl, aged 8, who six months previously had sickness, vomiting, headache, and inflammation of the brain, following a blow on the head. Mastoid suppuration followed. She died with three cerebral abscesses in the centrum ovale on the opposite side. The lateral and the posterior half of the longitudinal sinus contained old organised clot.

In the third a child, two years old, caught cold, became ill, emaciated, and was drowsy, finally dying two months later, without otorrhoea. A thin veneer of old clot was found lining the wall of the longitudinal and the lateral sinus on one side, where the tympanic cavity was filled with pus.

As with cerebral abscess we found that the otorrhoea had always lasted for months or years, the same is usually true for thrombosis. In only five had the otorrhoea lasted less than seven weeks. The shortest duration was three weeks, which occurred three times. The duration of life after the first symptoms of thrombosis was six to eight weeks in six cases, three to six weeks in three, less than three weeks in ten. The most acute cases lasted a week and a week and a half respectively. The most constant symptom was pyrexia (16), of which only one report fails to make mention. Headache (11), and rigors (10) were also very frequent. (Only sixteen of the cases have full clinical reports.)

In no other complication are erratic pyrexia and rigors so constantly present, and it will always be justifiable to assume that they probably indicate thrombosis in any patient in whom freely opening the deeper mastoid cells and draining the ear has not been followed by their subsidence. In such a case prompt action is necessary, remembering that in half of the cases death occurs within three weeks from the onset of the symptoms. Earache (9), as distinct from headache, is more common than with meningitis or abscess. The other symptoms which were noted were vomiting (7), coma (5), listlessness, giddiness, delirium, and convulsions, each twice; diarrhoea, emaciation, acute nephritis, and paraplegia, each once.

The patient often passes into a typhoid state, and may waste considerably; the tongue may be dry and the pulse rapid, the symptoms being essentially those of septicaemia. Locally subcutaneous oedema over the mastoid, and in some mastoid tenderness, were noticed, but these were only occasionally present. Some patients complained that their pain was at the back of the head, ran down the neck, and was made much worse by movement, so that the neck was kept stiff and the head as still as possible. When the clotting extended into the internal jugular vein it was frequently associated with a local fulness, a diffused inflammatory exudation, or an abscess along its course.

Only once were the optic discs stated to be normal. In six instances there was optic neuritis. In two the thrombosis was of two weeks', and in two of three weeks' date. In these there was no other complication, and the neuritis was secondary to the condition of the vein and the petrous bone. In the two remaining cases the thrombosis was of old date, and was, at the time of observation, associated with suppuration in the brain and cerebellum respectively. Neuritis is not common with abscess, having occurred in only two other instances, and it is probable that in all the six above mentioned the neuritis was due to the thrombosis.

We may conclude that, on the whole, optic neuritis is more suggestive of sinus thrombosis than of the other lesions. As a further example we may give this case of Mr. Lane's, which recovered:

Thrombosis of the Lateral Sinus; Optic Neuritis; Removal of the Clot; Recovery.—G. S., aged 10, under Mr. Lane and Dr. Perry, in August, 1888. He had had otorrhoea for years. Admitted with earache, tenderness, and oedema; temperature 102.2°. A week later inattentive, rigor, optic neuritis on the right side. The mastoid was explored without result. The rigors and pyrexia con-

tinued. Two days later, the lateral sinus was explored, a mass of foul clot was removed, and the jugular vein was tied. The boy made an uninterrupted recovery, and is now well.

As in three-quarters of the cases death ensues from pulmonary pyæmia, and in more than a third there are no other lesions, even at the time of death, it is clear that if thrombosis ensue in a case of otitis media, more especially if the discharge be foul, the only treatment that is of any value must be directed to the prevention of the pyæmia. When such a danger has to be combated we must be willing to run great risks in order to save some of the patients.

The internal jugular vein should be ligatured in the neck, the lateral sinus should be opened, and, if the clot be very foul and septic, if it may be scraped out, rendered aseptic as soon as possible, and, if be thought desirable, the sinus may be irrigated. It may, in some instances, be desirable to ligature the jugular vein low down in the neck, and to seal the wound; then, higher up, the vein may be again ligatured and divided, and the upper end brought out, so that if septic material passes down it may escape externally. This line of treatment, which may seem too heroic, has been recommended by some surgeons, and some such can alone avert the pulmonary infection which carries off these patients.

This same treatment deals with the dura mater over the posterior surface of the petrous bone, which also, if neglected, is a source of danger to nearly half its victims. If the lateral sinus, after it has been punctured, whether purposely or accidentally, be found to be healthy, thrombosis need not necessarily ensue. I have seen this happen three times, and no evil results followed, the patients dying from other causes.

The deductions to be drawn from the cases of lateral sinus thrombosis are that: 1. The disease more often spreads from the posterior wall of the middle ear than from the mastoid cells. 2. The otorrhoea is generally of some standing, but not always. 3. The onset is sudden, the chief symptoms being pyrexia, rigors, pain in the occipital region and in the neck, associated with a septicæmic condition. 4. Well-marked optic neuritis may be present. 5. The appearance of acute local pulmonary mischief, or of distant suppuration, is almost conclusive of thrombosis. 6. The average duration is about three weeks, and death is generally from pulmonary pyæmia.

EXTRACTS FROM

THE MILROY LECTURES ON THE ETIOLOGY AND PREVENTION OF PHTHISIS.

Delivered before the Royal College of Physicians of London.

By ARTHUR RANSOME, M.A., M.D., F.R.S.,

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LECTURE IV.—THE PREVENTION OF PHTHISIS.

Preventable Causes: a. Inherited; b. Acquired; c. Sources of Infection: d. Favouring Conditions.—Deterrent Measures against Marriage.—Treatment of Children of Phthisical Parents: Hygienic, Educational, Medicinal.—Choice of Work.—Ventilation and Warming of Schools.—Selection of Subjects of Acquired Predisposition from Chest Diseases.—Prevention of Dusts from Work.—Change of Climate.—Legislative Enactments respecting Tubercular Cattle, Flesh Meat, and Milk.—Combination of Sanitary Authorities.—Measures respecting Buildings; Compulsory Clauses.—Measures against Atmospheric Impurities.—Healthy Homes Societies.—Treatment of Excreta, Sputum.—Disinfectants.—Combustion.—Measures after Death.—Notification of Phthisis.—Isolation or Supervision.—Special Hospitals.—Principles of Treatment of Incipient Phthisis.

We are now in a position to consider what is possible to be done, and what should be attempted, to prevent the disease from attacking both human beings and animals associated in some relation or another with human beings.