SOCIETIES' PROCEEDINGS

ROYAL SOCIETY OF MEDICINE—SECTION OF OTOLOGY

December 7th, 1934

President—E. A. PETERS, F.R.C.S.

Discussion on Meningitis of Otitic Origin

MR. T. B. LAYTON: I propose to describe otitic meningitis in three stages; but shall first make some preliminary remarks upon: (*a*) The limitation of my subject. (*b*) Lumbar puncture. (*c*) Treatment that is common to all stages.

(*a*) Clinically, cases of otitic meningitis fall into two groups: (1) Those which arise from the newly-infected mastoid, and (2) those which arise in the presence of old disease.

I propose to refer only to the former, and to exclude extension of inflammation to the brain.

(*b*) I divide lumbar puncture into: (1) Diagnostic; (2) prognostic; (3) therapeutic.

(1) By *diagnostic* lumbar puncture I mean one which is performed to distinguish an otitic from other forms of meningitis. By it we gain help to determine the presence of tuberculous or cerebrospinal meningitis in a patient suffering from an otitis media when the case does not present the usual symptomatology.

(2) By *prognostic* lumbar puncture I mean one which is performed to tell the stage to which the disease has progressed. By it we gain help to determine whether the routine treatment common to all cases is sufficient, or whether this need be supplemented by some other therapy.

(3) *Therapeutic* lumbar puncture may be classified thus: (i) Drainage of the theca, which may be intermittent or continuous. (ii) Insertion into the theca of drugs and sera.

(*c*) The essential treatment in every case of otitic meningitis is to remove the factory of organisms in the mastoid or other part of the temporal bone which is in contact with the dura mater. If this be done no case is hopeless. If it be not accomplished the chance of recovery is negligible, even in the earliest case.

https://doi.org/10.1017/S0022215100041529 Published online by Cambridge University Press

The treatment that the medical officers of the fever hospitals of London have helped me to work out, and that is now the routine at those hospitals which I attend, is as follows:—

The mastoid is opened, and if a track of disease can be recognized leading down to the dura mater, it is followed, and the bone in contact with this is removed widely around the area of contact. If no such track can be recognized, the bone is removed widely from both middle and posterior fossae. Steps are then taken to fix the wound widely open. To do this the margin of the pinna should be sewn to the skin of the temporal region. It cannot too strongly be emphasized that the ordinary mastoid wound when left, even if packed with gauze, remains a closed one, with no more than 15° between the two walls. By sewing the pinna forward the angle approaches 180°.

The cavity is then hosed with saline, using anything up to a couple of gallons. The container is held high, and the nozzle at some distance from the wound, so that a good stream plays upon it to remove every scrap of bone, of pus, of granulations or of blood-clot. It is then lightly packed.

In the after-treatment I lay most stress upon large quantities of fluid. The patient must be propped on pillows, with the head sideways, so that no movement of the neck is needed when he drinks or takes food. The fluid must be kept ready by the bedside, so that anyone coming to see him can put the feeding-cup to the lips and coax him to take an ounce or two. In this way 300 oz. (2½ gallons) have been taken by an adult. For a child of six the minimum is 120 oz. (¾ gallon). The amount should be lessened as soon as the patient has turned the corner.

Secondly I give solid food at once. Thin bread and butter can be given in less than twelve hours from the operation, and other things added during the day. The benefit is seen on the second or third day, for it is always an effort for a patient to start taking solid food, and he may thus be taking a quantity of nourishment at a time when otherwise he would be trying to start. The early administration of solid food is also a valuable guide to the progress of the patient; the refusal to take it on the second or third day after having done so on the first may be the earliest sign of there being something needing further operative treatment or some accessory therapy.

For two, three, or even four days after the operation, the wound is dressed and the hosing is repeated twice or thrice daily, according to the condition of the patient and the progress of the disease. Absolute rest is essential at all other times. No visitors should be allowed, and it is well if the room can be darkened. By the end of the fourth day, and probably of the third, the patient is well on the way to recovery, or is going downhill and the case is hopeless.

With these preliminary remarks I pass on to the description of otitic meningitis in three stages.

(*A*) *The early stage.*—The symptoms of otitic meningitis are bilateral and proceed from above downwards. The first is a rigidity

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of the neck. The first stage then is that at which this alone is present. The symptom is absolutely characteristic and should be elicited thus :

> Take the pillows away so that the patient lies flat on the back ; put the two hands as you face him behind his head so that the thumb and three fingers of each hand are behind the occiput while the little fingers lie against the muscles of the neck. Then get the patient to leave the movements of the head in your hands, and bend the head towards the chest. If he can put the chin freely down on to the sternum he has not got meningitis. If, however, he cannot do this he almost certainly has, and the suspicion is confirmed by two other points. First, the character of the pull of the muscles. It is the sensation one gets when pulling longitudinally on a wet piece of string. Second, the feel to the little fingers of the complexus muscles. These stand out rigidly under the trapezius and cannot be pushed in by the finger.

As soon as this symptom is present the treatment outlined above should be instituted. Recovery ought to ensue. Every hour, however, that is lost endangers the patient's life. Lumbar puncture should not be done. It results in delay. It hurts and distresses the patient unnecessarily. It tells you nothing that you do not know already. The fluid will be under some increase of pressure, it will be unaltered in naked-eye appearance, the chemical and cytological examination will not show sufficient change to tell more than you know from the neck rigidity and the report on it will arrive too late to be of use. Lastly, by disturbing the intrathecal circulation, it may do harm.

There are those who say that this stage is not one of meningitis. I do not mind what you call it so long as you get busy. But do not call it *meningism*. This word by origin, by tradition, and by its very sound, is associated with inactivity. I find by experience that practice is usually based upon an association of ideas and in this you need a term that infers immediate and vigorous business. If you refuse to call this stage meningitis, call it *meningeal irritation*, and as you roll it off your tongue it will spur you to activity and will help you to sweep away all those suggestions for delay that come from parents, doctors, nurses, and from yourself, urging that the operation should be done to-morrow instead of to-day.

(*B*) *The second stage.*—Where rigidity of the neck alone is present the great majority of patients should recover.

The second stage is that in which Kernig's sign is present in addition to rigidity of the neck.

The state in which Kernig's sign is not present, but you cannot be sure of its absence, forms the boundary zone between the first and second stages of otitic meningitis.

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The similar boundary zone between the second and third stages is that state in which the abdominal reflexes are lost with or without the presence of headache.

Headache is due to distension of the theca with fluid. I have not found it an early symptom and would class it in the late secondary stage.

Neck rigidity and Kernig's sign are the only symptoms of otitic meningitis of clinical importance. To save the patient's life you must act before anything else develops.

In this second stage a prognostic lumbar puncture should be done either when the patient is under the anæsthetic before the operation or on the next day at the time of the first dressing. Which occasion is chosen will depend upon the time at which the operation is done and the accessibility of the laboratory, for the report upon the fluid is vitiated unless the examination be made within an hour or two.

If the fluid in the manometer is clear and the pressure not high nothing more need be done. If clear, but under an increase of pressure, sufficient should be removed to reduce it to normal; but if cloudy more should be drawn off, the exact amount depending upon whether the patient is under an anæsthetic or not.

If the fluid is clear the treatment should be that described for the first stage, but if it be opalescent this should be supplemented by lumbar puncture twice or thrice daily for several days. This should be performed sufficiently often and freely to relieve headaches and rigidity of the back.

(*C*) *The late stage.*—In this the patient lies on his side with the knees bent up, the back extended and the head thrown back. A cry escapes him from time to time owing to the severity of the headache. The lips are parted, the mouth is dry, the tongue is heavily furred and the temperature steadily mounting; but it is characteristic of the condition that the symptoms are exactly bilateral.

A prognostic lumbar puncture must be done. In the examination of the fluid I lay most stress upon two observations, the number of cells as indicated by the degree of opacity, and the presence of organisms.

With regard to the latter, a clear distinction must be made between those cases in which a hæmolytic streptococcus can be grown *in vitro* and those in which an organism, probably the hæmolytic streptococcus, can be observed by direct observation in a smear. The former is of pathological interest only, it is of no clinical value. The fight is over before the report arrives.

Intermittent thecal drainage is, I believe, still our stand-by for the milder cases of this group, where the symptoms are not as yet very severe, where the fluid is not opalescent, and where no organisms can be detected under direct observation.

The insertion of any antiseptic drug is, I believe, valueless.

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When organisms are seen by direct observation in a smear I used to think the case absolutely hopeless. Is this so? or will a case occasionally recover? It is the possibility of this occasional recovery which must stimulate us to ponder on what we should do.

The treatment common to all cases is not enough. Whether the addition of intermittent thecal drainage will occasionally save a case I do not know. It is not to be relied on. What more must we consider? Every British otologist of our generation owes a debt of gratitude to Mr. G. J. Jenkins for his perseverance and devotion to duty in dealing with otitic meningitis, and yet I believe his work has lain chiefly with clinical types different from those I have been dealing with to-day. I do not think his operation of translabyrinthine drainage is applicable to these cases except where the access of the disease to the meninges is across the labyrinth.

It seems that the time is ripe for two other lines of treatment to be attempted. The one is to take a leaf from the book of the physicians and to add to intermittent drainage of the theca the replacement by a serum either of a specific or of a non-specific nature, and the other is to replace intermittent by continuous thecal drainage.

It is along one of these lines that each of us must work in the future, but I urge that the first thing to do is clearly to define the type of otitic meningitis in each case that is recorded.

Mr. NORMAN JORY: The cerebrospinal fluid is of great interest to otologists in the various intracranial complications of aural disease.

Normal cerebrospinal fluid.—Before proceeding to consider the fluid in pathological conditions, it would be well briefly to enumerate the more important characteristics of the normal.

Pressure: The normal pressure, with the patient horizontal and relaxed, is between 100 and 200 mm. water. Greenfield advises that the pressure should always be taken before any fluid is withdrawn by puncture, and that it is unwise to continue withdrawal after the pressure has fallen to two-thirds of the initial value.

Massermann (1934) finds that after the removal of from 10 to 20 c.cm. the fluid re-forms at the rate of about 0·3 c.cm. per minute, while in pathological conditions Levinson (1929) found as much as 1 litre produced in twenty-four hours.

The normal fluid contains all the inorganic ions present in blood-plasma, as well as readily diffusible organic substances such as glucose, urea, uric acid, lactic acid, amino-acids and creatinine. Chlorides and magnesium are in much greater amount than in plasma; calcium, potassium, inorganic phosphorus, creatinine, amino-acids, uric acid and sugar in much less.

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Cytology : The fluid arising within the ventricles from the choroid plexuses is devoid of cells, but as it passes through the ventricles, the iter of Sylvius, and down the spinal canal it acquires from one to five cells per c.mm. from the surface of brain and meninges with which it has been in contact.

The mechanism of production of the fluid is still not known with certainty.

Fremont-Smith (1933) has considered the arguments for and against the theories of secretion and dialysis, and comes to the conclusion that while, on the whole, the dialysis theory is the more probable, yet it cannot be assumed to be the whole truth in the present state of our knowledge.

The fluid in pathological conditions.—Under pathological conditions the characteristics of the cerebrospinal fluid are altered, and these alterations may be helpful in diagnosis and prognosis.

Pressure : Pressures above 200 mm. are definitely pathological. Any factor tending to raise the intracranial venous pressure will raise the capillary pressure in the choroid plexus, and result in increased fluid formation. The same mechanism will delay absorption. A practical application of this principle is the Queckenstedt-Tobey-Ayer test for occlusion of a lateral sinus.

In meningitis several factors operate to raise the tension, mainly cerebral vasodilatation, and obstruction to absorption due to exudate blocking the circulation of the fluid, and blocking the Pacchionian bodies.

Cytology : An increase in cell-count up to 6 or 10 per c.mm. is suspicious, and over 10 definitely pathological. The cells found are mainly derived from the blood-stream—polymorphonuclears, lymphocytes, and mononuclears. The total count usually ranges between 5 and 500 in cases of so-called aseptic meningitis, and between 1,000 and 10,000 in cases of acute septic meningitis.

It should be remembered, as Greenfield points out, that a leucocytosis in the cerebrospinal fluid may be found with a degenerating glioma of the brain, very similar to that associated with cerebral abscess. As a general rule, however, a high polymorphonuclear count indicates a bacterial infection of the meninges or central nervous system.

Chemistry : (1) *Protein*, mainly albumin, shows varying increase. A total of over 45 mg. per cent. is considered abnormal, and it may rise to 1,000 mg. per cent. ; but its amount appears to give no guide to the prognosis of an individual case.

(2) *Chlorides :* The normal content, expressed as NaCl, varies from 710 to 745 mg. per cent., average about 725, whereas normal plasma chloride varies from 570 to 620 mg. per cent., average 595. Mestrezat (1912) was one of the first to note that the chloride concentration of the cerebrospinal fluid was higher than that of serum,

https://doi.org/10.1017/S0022215100041529 Published online by Cambridge University Press

and he further reported its decrease in meningitis, especially in the tuberculous variety. Greenfield, studying cases of meningitis following head wounds, found that in seven fatal cases the chlorides fell to under 680 mg. before death, while in one case in which the patient recovered, although pus and Gram-negative diplococci were persent in the cerebrospinal fluid, the chlorides remained at the normal level. He supposed, with Mestrezat, that the fall in cerebrospinal fluid chlorides was due to increased permeability of the meningeal blood-vessels. Linder and Carmichael have shown, however, that there was a coincident fall in the plasma chlorides.

Four cases of meningitis were investigated at St. Bartholomew's Hospital, two tuberculous, one chronic meningococcal and one streptococcal.

Taking the usually accepted ratio $\frac{\text{Cl plasma}}{\text{Cl C.S.F.}}$ as 0·895

In the tuberculous cases ratio was 0·86 and 0·90
In the chronic meningococcal cases ratio was 0·87
In the streptococcal cases ratio was .. 0·90

Greenfield and Carmichael point out that after exposure of the dura mater by operation, especially in the posterior fossa, the chloride content of the cerebrospinal fluid varies enormously for two or three weeks. This would explain apparent anomalies in the rule laid down by Greenfield that a low chloride content presupposed a generalized meningitis, and so carried with it the gravest prognosis.

(3) *Glucose:* The normal relationship of 45-65 per cent. of blood-sugar remains fairly constant in disease, except in meningitis. Here the reduction is due to glycolytic ferments liberated by dead cells or bacteria, and is progressive after the fluid has been withdrawn from the body. In aseptic purulent fluids the amount of glucose varies within wide limits, depending apparently on the number of polymorphs which have been killed. It is evident that the glucose content *per se* is not so important as the chlorides as a diagnostic and prognostic aid. All we can say is that in generalized septic meningitis glucose is reduced or absent, and that in aseptic meningeal reactions to intracranial septic foci it is usually normal, unless large numbers of dead polymorphs are present, when it is reduced in varying amounts.

(4) *Magnesium* has been extensively studied by McCance and Watchorn (1932) in a series of cases of meningitis, tuberculous and pyogenic, and they have their results summarized thus: In chronic meningitis, tuberculous or pneumococcal, subnormal amounts of magnesium have been found in the cerebrospinal fluid. A fall has not, however, been observed in the acute cases. The fall has been found roughly to run parallel with the well-known fall in chlorides. In one chronic case in which the patient recovered the cerebrospinal fluid magnesium returned to normal.

https://doi.org/10.1017/S0022215100041529 Published online by Cambridge University Press

Other constituents of the cerebrospinal fluid have been the subject of extensive study, but are of little practical interest to otologists.

Bacteriology.—The organisms most commonly isolated from otogenous cases of meningitis are streptococci, pneumococci, staphylococci, and the influenza bacillus.

Naturally the growth of organisms from the cerebrospinal fluid implies the gravest prognosis, though the converse does not always hold, as it is a common experience to meet cases with sterile fluid which prove fatal, and showing at autopsy purulent meningitis.

I have been able, by the courtesy of Mr. Sydney Scott, to investigate thirty-nine cases of acute septic leptomeningitis occurring in the Aural Department of St. Bartholomew's Hospital during the past ten years. Thirty-six cases proved fatal; in three recovery took place. Of the latter, two had streptococci in the cerebrospinal fluid, the other presented clinical, but no bacteriological, proof of meningitis. In fairness it should be realized that many of these patients when first seen were moribund with the disease; in some the condition was so desperate that any operation was frankly hopeless, in others an operation was performed in the hope that it might effect a miraculous recovery, such as is recorded from time to time in the literature. In the 36 fatal cases the sexes were equally represented. The great majority were in children; 18 cases being 10 years of age or under, 5 cases 20 years or under, 4 cases 30 years or under, no cases 40 years or under, 2 cases 50 or under, 4 cases 60 or under, 2 cases 70 or under, 1 case under 80. In nine of these cases (quarter of the total), the otitis media was chronic, and as might be expected these comprised the older patients, average age 38, youngest 4, oldest 74. Four cases were subacute, youngest 5, oldest 64, average 30. In the remaining 23 cases the otitis media was acute. The mastoid was the seat of acute or subacute inflammation in 29 cases, including one case with cholesteatoma formation; and of chronic inflammation in 3 cases, including one of cholesteatoma, and one on which a radical mastoid operation had previously been performed.

In the remaining four cases the mastoid condition was not ascertained either *ante* or *post mortem*.

Micro-organisms were recovered from the cerebrospinal fluid by lumbar puncture in 21 cases. They comprised streptococci in 11 cases, of which 5 were hæmolytic; pneumococci in 9 cases, including 1 associated with *B. Pfeiffer*, one type III and one thought to be type III; and hæmolytic *Staphylococcus aureus* in one case. In two cases no examination of the cerebrospinal fluid was made, but in one of these the diagnosis was confirmed at autopsy. In the remaining thirteen cases the cerebrospinal fluid from lumbar puncture proved sterile, and no organisms were seen in stained films. In

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eight of these cases an autopsy was secured, and in each confirmed the diagnosis made during life.

Treatment.—The appropriate mastoid operation was performed in 31 cases, Schwartze's operation in 26, radical mastoid in 4, and Küster's operation in 1.

Lumbar puncture was done in all cases, and repeated, where possible, once or twice daily—in one case on seventeen occasions.

One pneumococcal case and two streptococcal cases were treated with the appropriate antiserum, given intrathecally by the lumbar route. One other streptococcal case was treated by combined cistern and lumbar puncture, spinal wash-out and introduction of scarlet-fever antitoxin. In six cases thrombosis of the lateral sinus was present, and in one of these cases there was in addition cavernous sinus and, in another, multiple sinus thrombosis. In all these cases the lateral sinus was obliterated, clot evacuated, and the internal jugular vein ligatured. In one further case thrombosis occurred, and was treated as above, ten days after the primary operation. One case presented signs of acute labyrinthitis, and a superior vestibulotomy was performed.

Translabyrinthine drainage was resorted to in two cases only. In three cases an abscess of the temporosphenoidal lobe was drained, in one case the cerebellum, and in another the temporosphenoidal lobe, was explored, with negative result. In two cases nasal sinusitis was treated, in one the antrum, and in the other the sphenoid being infected. In seventeen cases an autopsy was secured, and in each case confirmed the diagnosis of meningitis. In four of these the internal ear was found to be infected; in one an unsuspected temporosphenoidal lobe abscess was present; in two there was osteomyelitis of the temporal bone, involving the petrous part in one; in four pus was present in one or more intracranial venous sinuses, and in three there was involvement of the sphenoidal and ethmoidal air cells.

Cerebrospinal fluid findings.—The highest cell-counts recorded were 26,400 leucocytes in a pneumococcal case, and 18,800 in a hæmolytic streptococcal case. Chlorides, where estimated, were always below 700 mg. per 100 c.cm., the lowest being 570 mg. per cent. the day before death in a streptococcal case. Quantitative sugar estimations were not made, but in all cases in which qualitative tests were performed shortly before death, sugar was greatly diminished or absent.

Quantitative protein estimations were made in most cases, but, as noted by Greenfield, gave no reliable index to prognosis.

One of the three patients who recovered was shown by Mr. Sydney Scott. This was that of a boy suffering from cerebellar abscess, in whose cerebrospinal fluid streptococci were found on more than one occasion. The report on his cerebrospinal fluid was:

https://doi.org/10.1017/S0022215100041529 Published online by Cambridge University Press

Cells, 484 polymorphonuclears per c.mm.; protein, 20 mg. per cent.; chlorides, 560 mg. per cent.; cultures, streptococci. The other two cases I will describe in greater detail.

I.—A boy, aged 7, admitted to hospital on account of vomiting and pyrexia.

Previous history.—Three weeks before admission he had sore throat, followed by earache and headache, with some vomiting.

Five days before admission he attended hospital and was kept in under observation for three days. At that time the left tympanic membrane was red, not bulging, there was some tenderness over the left mastoid process and in the left frontal region, with puffiness of that region and of the left upper eyelid. No pus was obtained by myringotomy, and skiagrams showed undeveloped frontal and clear ethmoidal sinuses, and ? opaque left mastoid.

On re-admission he was not in pain, and had no otorrhœa, but there was tenderness over the left mastoid. All reflexes were normal and the œdema previously present had disappeared.

Three days after admission the temperature rose suddenly to 103° F. Pulse 146. Schwartze's operation was performed by Mr. T. H. Just, and disclosed a moderately acellular mastoid, with thickened mucosa and sticky secretion in the antrum and cells.

The day following this operation headache and vomiting persisted, and slight head retraction was noted. 5 c.cm. cerebrospinal fluid were withdrawn, at pressure of 90 mm. water. Report: Cells 800 per c.mm., 66% polymorphs; protein 30 mg. %; sugar normal; chlorides 670 mg. %; cultures: two colonies staphylococci; probably contamination.

Second day after operation: Vomiting persisted. 30 c.cm. cerebrospinal fluid withdrawn, 10 c.cm. in morning, 20 c.cm. in afternoon. Report: Cells, very large numbers of polymorphs; protein 160 mg. %; sugar absent; chlorides 680 mg. %. Cultures: pure growth of hæmolytic streptococci. Lumbar puncture was performed twice daily for two days, and once daily for a further eight days, averaging 37.5 c.cm. per day. Further cultures were sterile.

On the twentieth day after operation he had a convulsion, with conjugate deviation of the eyes, and slow rotation of the whole body. He vomited, and temperature and pulse rose moderately. Cerebellum was explored: but no abscess found; then temporosphenoidal lobe with same result.

Four days later vomiting continued, so 70 c.cm. cerebrospinal fluid were withdrawn. Report: Cells 78 per c.mm., 62% lymphocytes; protein 60 mg. %; sugar normal; chlorides 720 mg. %; cultures sterile.

After this the boy gradually made an uninterrupted recovery.

II.—A girl, aged 7½ years. On the sixth day following Schwartze's operation, her temperature rose suddenly. Vomiting and head retraction were present, and Kernig's sign was positive.

https://doi.org/10.1017/S0022215100041529 Published online by Cambridge University Press

Lumbar puncture the following day disclosed turbid fluid under pressure of 250 mm., containing 2,000 cells per c.mm., mainly polymorphs, but with no organisms demonstrable by film or culture.

Treatment was confined to repeated lumbar puncture, and the patient left hospital on the fifty-seventh day after operation. About one year later she came under observation on account of epileptiform attacks, which have since been pronounced functional.

Cases of aseptic meningitis, sometimes known as serous or sympathetic meningitis, have not been included in these statistics. The cerebrospinal fluid in such cases is under increased tension and may be clear or turbid, with increase of protein. Cells are increased from 5 to 500, and may be mainly polymorphonuclears. But the fluid is sterile, the sugar is normal—unless very large numbers of cells are present—and chlorides are normal, or only slightly reduced.

Carmichael examined the cerebrospinal fluid in several cases of lateral sinus thrombosis, one of extradural abscess, and several of cerebral abscess, and found in all cases the above characteristics of an aseptic meningeal reaction.

I wish to express my thanks to Mr. Sydney Scott for permission to use the records of cases under his care at St. Bartholomew's Hospital.

Dr. C. P. SYMONDS: It has been borne in upon me as I have listened to Mr. Layton and Mr. Jory, that meningitis is not an easy condition to define. Mr. Layton has laid special emphasis upon neck-stiffness as the earliest evidence of meningitis. We should admit the value of this sign when present, but I would submit that its presence depends upon involvement of that part of the meninges which covers the dorsal aspect of the medulla. I have seen two patients with tuberculous meningitis die without any cervical rigidity. In one of these an autopsy was obtained and showed the exudate confined to the cisterna pontis, the cisterna magna having escaped. Again, in cases of meningitis arising from a source of infection in the middle or anterior fossa, one may have symptoms such as headache and vomiting with a turbid spinal fluid before neck-stiffness appears. The absence of this sign, therefore, cannot exclude the diagnosis of meningitis. There are cases in which examination of the spinal fluid provides the first evidence.

The distinction between septic and aseptic meningitis based upon examination of the spinal fluid is, I think, of doubtful value, and may be dangerous. A clear fluid, with moderate increase of cells and protein, may be found when there is a septic focus present from which bacterial toxins only are leaking into the subarachnoid space, or if organisms are gaining entry, they are rapidly destroyed. On the other hand, a similar fluid may contain living bacteria which can be grown on culture. If the distinction between aseptic and septic

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meningitis is that between an organism-free fluid and an organism-containing fluid, then we should remember that the latter represents an early stage of the former, and that the difference in time may be a matter of a few hours. As Mr. Layton has pointed out, there is not time in these cases to wait for the results of culture.

Mr. Layton has given us a very clear account of the uncomplicated meningitis developing from acute otitis media. This, however, is a type of case which seldom comes within the view of the neurologist except when the middle-ear disease has been missed by the general practitioner. I propose, therefore, in my remarks, to cover a wider field, which includes meningitis arising from chronic as well as from acute middle-ear infection.

I take it there are three main pathways or modes of progression of infection from middle ear to meninges :

(1) Directly along thrombosed and infected vessels in the bone which ultimately communicate with those in the subarachnoid space.

(2) More or less directly through the labyrinth, whose fluid communicates with the cerebrospinal fluid.

(3) Step by step through bone, dura, and arachnoid with a reactive process on the part of the newly infected tissue at each step.

In the first two instances the invasion of the subarachnoid space is sudden and may be extremely rapid. When the infection invades *viâ* the labyrinth there will be appropriate premonitory symptoms. If the spread is by way of infected vessels there may be none. The symptoms and signs of a diffuse meningitis, as Mr. Layton has described them, may simply be added rapidly to those of otitis media. I suppose, from what Mr. Layton has told us, that this is a common mode of invasion in acute otitis media, and that this is why he has observed neck-stiffness to be the first evidence of meningitis in this group.

As I have said, my experience of these cases is small, but from the study of diffuse meningitis of other origin I should have expected that symptoms such as headache, vomiting, and restlessness might have found a place among the earliest warning signs. When the process of invasion has taken the form of an osteomyelitis with progressive spread through the meninges, I believe that such warning signs are the rule rather than the exception, and would lay especial emphasis on the importance of headache and vomiting, especially if these symptoms are associated with a rise of temperature. It is in such cases, I think, that lumbar puncture may be of value in converting a suspicion into the certainty of early meningitis before neck-stiffness has appeared, providing an indication for exploration and clearance of the infected cavity.

It may be argued that such symptoms as I have described not infrequently occur in the course of convalescence from otitis media, or after a mastoid operation, without the development of frank

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meningitis. This I expect is true, but in many cases which I have been called to see at a later stage, with extradural or cerebral abscess, I have found an episode of this kind in the earlier history which has suggested that it was at this point of time that the infection first reached the meninges. My suggestion is that the aural surgeon should have the cerebrospinal fluid examined more often in such instances. I do not believe that it is a dangerous procedure.

There are two possible objections to lumbar puncture in such cases. One is that if there be a septicæmia, lumbar puncture may determine the passage of organisms from the blood-stream into the meninges. This fear is based upon experimental and clinical observations published by Wegeforth[1] and others, but considerable doubt has since been cast upon their validity (*vide* Kolmer).[2] The other objection is that the withdrawal of spinal fluid may lead to the rupture of adhesions and so to the diffusion of a meningitis which has been safely localized. This risk is, I believe, a small one. I have seen one case of extradural abscess in which the sequence of events justified the suspicion that it had occurred.

My desiderata would be that the patient should be within easy reach of the aural surgeon before the lumbar puncture is performed; that not more than 2 c.cm. of fluid be removed; that a fine needle should be used, allowing a slow removal, and that the report should be available within six hours. Cell-count and protein estimation are all that are necessary, and it should not be difficult to get this done. I should like to emphasize the need for a differential count of the cells. The normal cells of the spinal fluid are small lymphocytes. In one case under my care the cell-count was reported by Dr. Greenfield as 3·5 per c.mm., but he added that among the eighteen cells counted there were three large mononuclears and one polymorphonuclear. As events showed, this represented the reaction to an extradural abscess with early meningitis.

I am going to join issue with Mr. Layton when he lays stress upon the importance of neck-stiffness and Kernig's sign in prognosis. These signs indicate the extent of the meningitis rather than its severity. It is not the irritation of the meninges that kills the patient, but the action of the organisms or rather their toxins, upon the vital centres. Therefore it is the toxicity of the organism that matters, on the one hand, and the patient's resistance on the other.

The points that are helpful in prognosis are the presence and nature of organisms in the cerebrospinal fluid, and the patient's general condition. A brisk reaction of the meninges, as evidenced by a turbid fluid, is not necessarily of bad prognostic import. I have no doubt that we could all provide, from individual experience, examples of this generalization. One in particular comes to my mind—a case seen seven years ago:

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A boy, aged 12, developed an acute left otitis with discharge on *January 18th*. On *January 28th* he complained of pain in the back of the neck and his temperature rose to 101°. Subsequently his temperature remained high. There was vomiting, drowsiness, and irritability, and on *February 4th* he complained of numbness in the left side of his face. On *February 5th* I found him delirious with high fever, well-marked neck stiffness and Kernig's sign and absent abdominal reflexes. The left corneal reflex was absent and there appeared to be blunting to pin-prick over the left side of his face. There was no swelling of the mastoid or any tenderness except at its tip.

Lumbar puncture gave a turbid fluid under great pressure. I advised that an aural surgeon be called in, but the parents were unwilling to have an operation and the doctor took a favourable view of the case. This was justified by his report written on *February 12th*: "I am writing this in the bedroom of the boy you saw with me last Saturday. He has completely recovered; all cerebral and spinal symptoms have disappeared, headache, stiffness and photophobia. Temperature has been normal for five days with the exception of two rises to 99°."

I have only to add that this boy remained well; I saw him eight months later on account of a stammer; his ear-drum was healed.

Such cases as this support the opinion of Neumann,[3] who says:

"In analysing the cases (of otitic meningitis) which end in recovery, one might be tempted to believe that it is a matter for the most part of cases of benign meningitis whose recovery would appear to depend rather upon the nature of the affection than the method of treatment."

I am, of course, not suggesting for a moment that Mr. Layton's argument in favour of a complete removal of the factory of organisms is open to criticism. I have quoted my case, together with Neumann's dictum, as evidence only of the fact that prognosis cannot be judged entirely in terms of neck-stiffness, Kernig's sign, or turbidity of fluid. Given an organism of low virulence and a patient with good resistance, the battle may be won.

As to the possibility of recovery in those cases in which there are organisms in the spinal fluid: In the series reported by Neumann in the paper from which I have quoted, of 59 cases with otitic meningitis 22 recovered, and among these 22 there were 6 in which organisms were present. In four they were found in smear and culture, and in the other two in smear only. In three instances the organism is described as a streptococcus, in two as a diplococcus, and in the other as *Streptococcus mucosus*.

Such a report is most encouraging to one like myself who had thought that recovery when organisms could be demonstrated in the fluid was quite exceptional.

It is for such cases that I should be inclined to reserve the plan of frequent drainage of the cerebrospinal fluid. Continuous drainage

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has been practised and advocated both in this country by Hampson and by Kubie in America, combined with forced fluids by the mouth or hypotonic intravenous saline. In principle the plan appears to be sound. I should, however, be somewhat afraid of secondary infection through the track of the needle, and should imagine that the procedure might impose a considerable strain upon the patient's resistance.

As to the value of intrathecal serum in these cases, I am in doubt. It has one grave disadvantage in that the serum itself causes irritation and congestion of the meninges, with exudation of plasma. The subarachnoid spaces may thus become so full of gelatinous fluid that drainage becomes difficult or impossible.

I should prefer, therefore, to have these patients with severe meningitis treated on the lines which Mr. Layton has advocated—immediate surgical drainage of the infected focus, adequate nursing with forced fluids, and drainage by lumbar puncture twice in the twenty-four hours.

REFERENCES

[1] WEGEFORTH and LATHAM, *Am. Journ. Med. Soc.*, 1919, clviii., 183.
[2] KOLMER, *Association for Research in Nervous and Mental Disease*, 1931, xii., 500.
[3] NEUMANN, *Rev. de Laryng.*, 1934, lv., 1.

Mr. E. WATSON-WILLIAMS said that he was not able to deal with the subject quite on all fours with Mr. Layton's opening, as that was confined to meningitis following primary acute otitis; whereas in his (the speaker's) experience, meningitis was far more frequently a complication of chronic or recurrent otitis. This might explain the differences between the clinical pictures seen by Mr. Layton and himself. In the first place, he had not recognized the "early" stage of meningitis, in which there was neck rigidity without other signs. Neck rigidity was not pathognomonic of meningitis; one saw it in hæmorrhage, in brain abscess, and sometimes even in uncomplicated otitis media where there was no other evidence of meningitis. Also, one sometimes saw cases in which meningitis was manifest, but there was no demonstrable neck rigidity—cases of supratentorial meningitis. Three years ago he had published a series of twelve consecutive cases of septic meningitis observed during six years,* to which he could now add a further seven cases. Severe headache, fever, and usually vomiting, marked the onset of meningitis; in four cases there was certainly no rigidity of the neck when this diagnosis was made (and in three of these the patient died). On the other hand, definite head retraction and a positive Kernig's

* "Septic meningitis," *Practitioner*, 1931, ii., 659.

https://doi.org/10.1017/S0022215100041529 Published online by Cambridge University Press

sign might be seen within a few hours of the onset; out of seven cases in which these signs were present, only one patient had died; these were mostly early cases. Similarly, drowsiness might be an early sign, and was not correlated directly with a bad prognosis.

It was very important to emphasize, as Mr. Layton had done, that when one was confronted with a case of septic meningitis, one should not wait for the results of a pathological report, or animal inoculation, but should have the theatre made ready at once: meningitis was an urgent indication for operation, and the time to operate was as soon as ever it could be arranged. This was, in fact, one of the few surgical emergencies in which every hour wasted before the operation diminished the patient's chances of recovery.

He did not believe that there was any objection to diagnostic lumbar puncture, and always had it performed; very valuable information might thus be gained, particularly in the early stages.

Naturally, if *living* organisms were found in the cerebrospinal fluid the outlook was worse: but even patients, comatose, incontinent, and having purulent cerebrospinal fluid containing living cocci, might recover, and the only factor which the surgeon could influence was the interval between diagnosis and operation. Of the nineteen cases to which he had referred: in six cases operation was performed within three days of the onset; all the patients recovered, although clinically three were cases of severe septic meningitis. Of the thirteen not operated on until the third day or later, in three the patients recovered and in ten the patients died, including four whose clinical signs were relatively mild. Could one better illustrate the supreme importance of prompt operation?

What further measures should one adopt besides such operation on the ear as local features indicated? He had never had the courage—or, perhaps, the energy necessary to assemble the elaborate equipment—to carry out the treatment by transthecal drainage with irrigation of the arachnoid with iodoformized serum or other fluids; and he did not believe that any good was done by such measures or by the injection of disinfectants into the theca. Indeed, he doubted whether intrathecal injection of anti-sera was of much benefit; he certainly would not use mercurochrome thus. He had concluded that the best way to employ drugs was by intravenous injection, and a severe case of meningitis should be treated by giving 40 c.cm. of colloidal silver intravenously, just before lumbar puncture. Therapeutic lumbar puncture was carried out daily (he had not attempted more frequent puncture), and enough fluid was drawn off to reduce pressure to normal. In two cases of the most severe and desperate type, following labyrinthitis, he had carried out drainage through the internal meatus (one patient survived). If a patient did not recover when treated thus, he doubted whether any more drastic surgical measures would succeed. The keynote of surgical

https://doi.org/10.1017/S0022215100041529 Published online by Cambridge University Press

treatment of septic meningitis was early operation on the ear and repeated lumbar puncture.

REFERENCE

" Operative Treatment of Meningitis," *Bristol Medico-chir. Journ.*, 1926, xliii., 160.

The PRESIDENT said that there were two groups of cases with which this discussion was concerned. In the first there was what might be called infection owing to approximation, i.e. a streptococcal infection passing through all protecting tissues and infecting the meninges. The second group included chronic infection by the more generally recognized paths of which Dr. Symonds had spoken. In these matters *post mortem* results had been very indefinite, and it was necessary to rely on the findings in cases of cerebrospinal fever. In 1906 Georki published a series of cases, in which he showed that in eleven cases infection took place along the auditory nerve, in one case *viâ* the saccus endolymphaticus, and in three cases through the aqueduct of the cochlea.

He agreed with Dr. Symonds that the condition of effusion which some called meningismus was present in a greater number of cases than was generally recognized. He had been told that it took forty-eight hours for cells to come from the meninges into the lumbar region. Some years ago he had seen several cases of cerebrospinal fever, in which the onset of the illness was very acute, and although during the first twenty-four hours the fluid was under pressure, infected cells were not recognized until forty-eight hours afterwards. Did Dr. Symonds consider that increased pressure was a very important sign, or did he favour the practice of not waiting for the appearance of cells? He would also like to know whether Mr. Layton had used serum intravenously in his early cases of meningitis.

Mr. SYDNEY SCOTT: As regards Mr. Layton's reluctance to perform lumbar puncture for diagnostic purposes, I am glad to hear that he is less opposed to this invaluable method of diagnosis than I understood him to be.

With reference to neck rigidity: Resistance on the part of the post-cervical muscles permitting the head to be fixed forwards is a most valuable sign, often the first to raise suspicion of lepto-meningitis. It is very rarely absent, though only a few days ago I saw with Mr. Just a patient with proved lepto-meningitis (*Streptococcus hæmolyticus*) who lay in bed with his head on three pillows and flexed forwards.

I agree with Mr. Layton as to the necessity of free drainage of the primary focus, but I have never seen recovery from elaborate methods devised to irrigate the cerebrospinal system, though we

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have had recoveries (outside Mr. Jory's series) from repeated lumbar puncture.

As regards the operation of translabyrinthine drainage: I have always held this to have a scope restricted to cases of lepto-meningitis following proved labyrinthitis in the earliest stage. I cordially agree with Dr. Symonds' observations that after drainage of the primary infection recovery depends on the virulence of the organisms versus the antibodies produced by the patient.

I think that fatal termination can be ascribed to three processes: (1) increased intradural pressure; (2) accompanying encephalitis; (3) septicæmia. Repeated lumbar puncture aims at defeating the first condition, the others appear to be beyond the scope of surgery unaided by what may be called general medical treatment.

Mr. Jory has asked me to give some details of one of the cases of recovery in his series.

The patient, a boy aged 6, was shown at the meeting of the Section held in March, 1934, when other cases of brain abscess were also shown.* He was not shown as an example of lepto-meningitis though he had this complication too. The evidence of lepto-meningitis was the presence of streptococci in the lumbar cerebro-spinal fluid withdrawn many times during a period of over three weeks, namely: *July 1st, 14th, 25th*, 1927. On each of these dates the pathologist reported the presence of cocci in the cerebrospinal fluid. Fortunately the boy is perfectly well and quite free from after-symptoms, in spite of the double complication—cerebellar abscess with meningitis.

The statistics quoted are not comparable with some international statistics showing a recovery of something like 60 per cent., perhaps because at St. Bartholomew's Hospital we only record as cases of lepto-meningitis those in which the infection is proved pathologically as well as clinically, not clinically alone.

Mr. HOLT DIGGLE said that it was interesting to note how the pendulum had swung in the matter of treatment of meningitis. Eight years ago, under the stimulation of the work of Eagleton and of Jenkins, several major operations were performed for this very severe complication—e.g. translabyrinthine drainage, thecal drainage with lavage, and even more extensive operations in the way of approach to the foramen magnum. At the meeting at which Mr. Jenkins had discussed the subject, he (the speaker) had still felt that repeated lumbar puncture would be the method of choice, having had two or three recent successes from it.

On the question of therapeutic lumbar puncture, he was to some extent in agreement with Mr. Layton. The otologist's chief function was to cure, not to provide statistics. If a patient had a headache,

* *Proceedings*, 1934, xxvii., 1674 (Sect. Otol., 143).

https://doi.org/10.1017/S0022215100041529 Published online by Cambridge University Press

perhaps with a little vomiting, but with no other clinical evidence of meningitis, one should approach the primary focus in the mastoid and eliminate the sepsis. But why disturb the lumbar theca by reducing the pressure, if there was the slightest chance of thereby spreading the infection? If after forty-eight hours the headache was still present, a lumbar puncture could be done, which would make it possible to decide whether meningitis was present, and whether repeated lumbar puncture should be carried out.

He was interested to hear Mr. Watson-Williams speak of intravenous injection of colloidal-silver salts combined with repeated lumbar punctures. He believed this was a method, adopted by those treating neurosyphilis, in which intravenous injections of arsenical compounds were given, followed by lumbar puncture. He had no knowledge as to results of this treatment, but felt that there was a field for research in order to ascertain whether there was any type of antiseptic which could be passed into the cerebrospinal fluid from the venous circulation. The choroid plexus was, of course, very selective in this matter, and possibly few drugs of any value could thus be diffused into the cerebrospinal circulation. In the few cases which he had had, he had relied on the intravenous injection of antiserum, combined with repeated lumbar punctures, in the belief that some of the antiserum would pass over.

Sir JAMES DUNDAS-GRANT said that he was interested in a paper by Dr. Maloens of Brussels,* who showed the effects of the intravenous injection of an antiseptic drug preceded by lumbar puncture, whereas drugs injected into the theca found their way quickly to the kidneys.

Mr. C. P. WILSON said he wished to ask Mr. Layton a question, in view of what he said about tying the auricle forwards in order to ensure free drainage of the infected area. To what extent was he prepared to go to ensure free drainage in a case in which meningeal infection was diagnosed? Even in a Stage I case did he, in all those cases, carry out a complete mastoid operation? If not, why not? Did Mr. Layton, as a routine, expose the dura mater of both fossae? If so, how much bone did he remove in order to ensure that exposure? If he did not proceed to that extent, he (the speaker) would like to know why.

Mr. LAYTON (in reply) said that the request made to him was that he would open a discussion on otitic meningitis, and for that reason he had not included notice of the various cases brought to hospital in which the meningitis had arisen from other parts of the body. As to nasal infections, he was in agreement with Dr. Symonds but he did not think that remarks applicable to the nose could be applied to the ear, or *vice versa*.

* "Sur la chimiothérapie postopératoire des méningites purulentes généralisées septique d'origine otitique." *Brux. Med.*, 1924, **5** (i.), p. 81.

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There were twenty different subjects which he had excluded in order that he could concentrate on a clear-cut description of one type of case. One of these was the differential diagnosis of otitic meningitis from meningitis of other kinds occurring in people who had otitis media. There he was in agreement with other speakers as to the value of lumbar puncture. He had not discussed diagnostic lumbar puncture to-day, because in a typical case of otitic meningitis arising from the previously uninfected ear it was not necessary. Though Mr. Sydney Scott appeared to differ from him, both opened the mastoid, and that was the point upon which he (the speaker) insisted. He was not dead set against lumbar puncture, he was against house surgeons and others jabbing needles into the backs of little children without due thought as to what they were going to gain thereby. He maintained that the surgeon did not make the patient well. All he did was to put the patient in such a condition that recovery resulted from the unremitting care of the doctor and the nurse, perhaps particularly of the latter. He again emphasized the importance of getting rid of the factory from which organisms were getting in from outside the dura mater to the meninges. He liked Dr. Symond's description of the three kinds of path by which the micro-organisms entered. With regard to the channel from the vessels into the bone, he doubted whether it often occurred without preliminary thrombosis of the lateral sinus. These he would call *the three lesions of access*. In the acute cases the same kind of thing occurred, but these were not complicated by chronic progressive changes, such as fibrosis, and sclerosis of bone.

As to the question of virulence, it was true that, in the end, the virulence of the organism present was the factor which determined whether or not the patient would get well, but in the case which Dr. Symonds had described there occurred that concatenation of unassessable factors called " luck ", and the patient had recovered.

With regard to neck-stiffness, he laid more stress on that symptom than most of the speakers appeared to do—probably because he did not feel sure that all observed it when it was present. It was not the same thing as head retraction. Neck-stiffness could be recognized long before head retraction occurred. It was true that in nasal meningitis one did not encounter it. The two were not comparable symptomatically. He had never seen a case of otitic meningitis in which neck-stiffness was absent. If no neck-stiffness was present in a case of meningitis he would like to know the result of the lumbar puncture. He did not mind the term " meningeal irritation ". He agreed that a case, here and there, would get well if left alone. One of his own first cases was that of a small child, who was brought into Guy's with a request from the physician that he (the speaker) would perform the mastoid operation, as there was neck-rigidity. He (Mr. Layton) had said that it would be well to

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do a paracentesis; that was done and the patient got well. He would not follow that course to-day, however, because most of the cases passed from neck-stiffness to Kernig's sign, and then showed the " symptoms and signs of meningitis " as described in the books. If those signs were waited for, the patient would die in the meantime. If a chance for recovery was to be given, operation should be performed when there was neck-stiffness.

With regard to the warning signs given by Dr. Symonds, it was necessary to distinguish between the symptomatology of the lesion of access and the symptomatology of the meningitis. If there was an inflammation of the bone spreading throughout the petrous bone, Gradenigo's syndrome might be present with paralysis of the eye. The latter was not a sign of meningitis but if such a patient had meningitis the paralysis of the eye was a sign of the lesion of access which preceded it. If one met with a patient who had labyrinthine symptoms, these latter were not symptoms of meningitis, but of the lesion of access which preceded the meningitis. He thought that was the meaning of the vomiting in the early stage of acute cases. He looked very gravely on vomiting in all children suffering from otitis media. What first made him do so was a series of cases of which Dr. Symonds had taken charge, cases which he (the speaker) had had at fever hospitals and in which late neurological symptoms or complications had developed. In each of those cases there was a note of the child having vomited. It was said that any child was likely to vomit when it was ill. It might do so as a result of acidosis. In a child who had no acidosis, vomiting must be regarded seriously if there was a running ear. It pointed to the need for considering the necessity of exploring the mastoid.

Dr. SYMONDS (in reply) said that meningococcal cases were difficult ones to argue upon, because the first stage of meningococcal meningitis might be entirely septicæmic, so that there would be a normal fluid at that stage, which might be under some increased pressure owing to cerebral congestion. He would be disinclined to accept a pressure-reading as evidence of meningitis unless there was some abnormality in the cells of the fluid.

CASES

Operative Isolation of the Petrous Bone.—E. A. PETERS, F.R.C.S. (President).

Otitic Suppuration. Meningitis. Operation: Isolation of Petrous Bone. Pressure on VIIth and VIIIth Nerves. Recovery of VIIIth Nerve and part of VIIth Nerve.

Leonard B., aged 38.

Previous history.—Three years' history of purulent discharge from the right ear. No pain. Deafness. For one month, attacks

https://doi.org/10.1017/S0022215100041529 Published online by Cambridge University Press

of giddiness when standing up or lying down ; throbbing in right temporo-sphenoidal region. Occasional vomiting. Bowels opened with medicine.

Condition on admission (30.11.32).—Right ear ; granulations, touched with silver nitrate (solid).

Hearing : Right $\frac{28}{30}$ c mast. $\frac{30}{30}$. Left.
Nil. c value of middle ear $\frac{15}{60}$.

Pupils equal ; react to light. Horizontal nystagmus on looking to left. Right knee reflex + + plantar gives flexor response. Abdominal reflexes present. Blood-pressure 155/90. Wassermann reaction negative.

8.12.32 : Better. Aural polypi again cauterized.

16.12.32 : Temperature 100·4° F. Pulse 84. Respiration 20. Has severe pain in head. Lumbar puncture : Cerebrospinal fluid under pressure ; cells 103 per c.mm.

19.1.33 : Lumbar puncture : 1,632 cells per c.mm. ; large and small lymphocytes, some polymorphonuclears. No organism on culture.

20.1.33 : Lumbar puncture : Cerebrospinal fluid under pressure ; *total cells, 1,272 per c.mm.*

Diagnosis : Meningitis by labyrinthine infection.

23.1.33 : *Operation : Isolation of the petrous bone.*—A radical mastoidectomy exhibited some osteitis but no obvious fistula in either the foramen ovale or external canal. The tegmen tympani was removed and the middle fossa exposed ; later a packing of b.i.p. ribbon gauze was inserted between the dura and the bone.

The lateral sinus was exposed down to the bulb, and the bone cleared away anteriorly till the dense bone of the canal system was reached. Then, in the area between the inferior and superior petrosal sinus, the dura mater was separated from the bone with a special sharp spoon. The point of the spoon was directed to the internal auditory meatus as located by a view of the outer wall of the internal ear.

The facial nerve became paralysed, as shown by loss of tone of the eyelid and it was thought that the VIIIth nerve had gone as well. B.i.p. packing was introduced into this triangular extradural space. The middle-ear pus gave growths of coliform bacilli and streptococci.

25.1.33 : Cerebrospinal fluid under pressure. Cell count 817 per c.mm.

10.2.33 : All the packing removed. Cerebrospinal fluid 44 cells per c.mm.

Patient appeared to be stone deaf with TF c applied to right ear. The hearing has gradually improved in the right ear and at the present time though the c value of the middle ear is nil, bone conduction is equal on both sides. The facial nerve has partly recovered.

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There is some giddiness but I have not carried out any labyrinthine tests.

In this operation on a case in which meningeal infection *viâ* the labyrinth appeared certain, an attempt was made to pack off the infecting petrous bone, divide the VIIth and VIIIth nerves, separate the saccus endolymphaticus and possibly deal with the aqueduct of the cochlea and petrosal veins by pressure.

Though at the operation the VIIth and VIIIth nerves appeared to be divided, recovery has taken place.

The sheath of the nerve was opened as indicated by a rush of cerebrospinal fluid but complete division could not have been effected; the temporary paresis must have been due to pressure by the plug.

Acute Otitis Media—Acute Mastoiditis—Schwartze's Operation—Onset of Meningitis during Convalescence—Pneumococci from Cerebrospinal Fluid—Recovery.

—F. C. W. Capps, F.R.C.S.

J.M. was admitted to hospital on *March 17th*, 1930, suffering from acute mastoiditis (right). A Schwartze operation was performed.

18.3.30: Patient developed pneumonia at left base. He progressed satisfactorily until 3.4.30 when he developed pneumococcal meningitis. Sudden onset with neck rigidity while up on couch for the first time. He was treated with immediate and repeated lumbar punctures and replacement of the cerebrospinal fluid by Type 1 pneumococcal antiserum. This was done on speculation as there had been no time to group the pneumococcus.

1.5.30: He was stated to be fit for discharge, but was detained, as the ward was closed on account of diphtheria.

12.5.30: He was sent to the Fever Hospital with Klebs-Löffler bacilli present in his throat. Uneventful recovery.

Mr. Capps (in reply to a query) said that in the first three days lumbar puncture was done twice a day, and afterwards it was carried out once a day for the next week.

Bezold's Mastoiditis: Cholesteatoma with Intact Tympanic Membrane.—N. A. Jory, F.R.C.S.

Patient, a man, aged 24, was admitted to hospital on account of a Bezold's abscess.

History.—Swelling and pain in right side of neck for three days. Discomfort and deafness in right ear for four days. Past history of scarlet fever and transient otorrhœa as a child.

On admission.—Right tympanic membrane red, malleus appeared to be fixed, with some swelling of the membrane anteriorly and posteriorly. No perforation; no discharge.

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Operation.—A large cholesteatoma was found occupying the mastoid process; this had eroded the posterior wall, exposing the lateral sinus for ¾ in., and had perforated the tip of the mastoid, leading to a Bezold's abscess containing about ℥j pus. The mastoid antrum and aditus were free from cholesteatoma, and the mucosa was only moderately inflamed. No myringotomy performed.

Hearing returned three days after operation, and the tympanic membrane appeared to be normal.

? Acoustic Tumour, ? Ménière's Syndrome: Case for Diagnosis.—MAURICE SORSBY, M.D., F.R.C.S.

Sidney J., aged 54, complains of giddiness and vomiting.

Previous history.—Two years ago he fell off a bus, hitting his head; he was unconscious for a few minutes, but felt quite well afterwards.

Eighteen months ago an "acute peptic ulcer" developed; on several occasions the patient vomited large quantities of blood; he had melæna and felt faint. Five months ago he became suddenly deaf in the left ear. No obvious cause was found. Four months ago he suddenly became giddy and several times vomited the entire contents of the stomach. The attack lasted two hours and he felt quite well afterwards. He states that he tended to fall to the left side. Three months ago there was an identical attack of sudden onset, and he felt well afterwards.

Since then he has had frequent attacks. Eustachian catheterization during the last three weeks has greatly reduced the severity of these attacks. Lately he has been having dull headaches, and a general heaviness of the head. The sense of taste has become poor.

Condition on examination.—Left facial paralysis; practically total loss of sense of taste for sweet, salt, and bitter; taste for sour is fairly good. Left ear: There is a considerable loss of air conduction, and bone conduction is shortened. Rinne negative and Weber referred to the left side. There is loss of low tones. To the caloric test the affected ear is slightly more sensitive than the healthy ear. Right ear: Air conduction normal; bone conduction shortened. Rinne positive.

There is no other evidence of a neurological lesion.

DISCUSSION.

MR. SYDNEY SCOTT said he had not seen a case of auditory-nerve tumour in which the caloric reaction was present.

MR. CAPPS asked whether the Wassermann test had been made in this case.

MR. SORSBY (in reply) said that the Wassermann reaction was negative and the caloric test elicited a normal response.

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Royal Society of Medicine

Acute Mastoiditis : Meningitis.—T. H. JUST, F.R.C.S.

The patient, a boy, aged 7, was admitted to hospital on account of vomiting.

History.—Three weeks previously had sore throat, followed by earache, left side, and frontal headache. Five days previously was seen to have red—not bulging—drum. Drum incised, but no pus obtained.

On admission.—Tender mastoid. No otorrhœa. No abnormal neurological signs.

Three days later.—Mastoid operation performed.

Day after operation.—Signs of meningitis.

Second day after.—Streptococci grown from lumbar puncture fluid.

Repeated lumbar puncture. Recovery.

Temporo-sphenoidal Abscess. Drainage. Recovery.—MAURICE SORSBY, M.D., F.R.C.S.

Rose S., aged 44, admitted to St. Andrew's Hospital, *April 4th*, 1934, complaining of otorrhœa, giddiness, and vomiting.

History.—The left ear had been suppurating for fifteen years ; a sense of fullness had been present for the last fourteen days. Vomiting and giddiness had set in three days previously.

Condition on examination.—Profuse, pulsating fœtid discharge from the left ear. Total destruction of the drum, associated with severe deafness ; left facial paralysis. Lateral nystagmus to the right. Past-pointing with the right finger.

First operation.—Radical mastoid ; a large cholesteatomatous mass removed. The external semicircular canal was found to be eroded. The dura mater and lateral sinus were exposed and found to be normal. Lumbar puncture : Cerebrospinal fluid was clear and not under pressure.

Pathologist's report (Miss K. M. Collis) :—Cells, 7 per c.mm. (lymphocytes) ; protein, 0·025 per cent. ; globulin, no excess. No organisms seen in direct film. Culture sterile.

Post-operative history.—After initial improvement, during which the vomiting and giddiness practically disappeared, the patient became worse on *April 27th* (eighteen days after the mastoid operation). Severe headache developed on the affected side ; the temperature rose to 101·6°, the pulse to 110, and drowsiness, rapidly passing into unconsciousness, set in.

Condition on examination.—Dilatation of homolateral pupil ; homonymous hemianopia ; past-pointing ; bilateral papillœdema ; optic aphasia ; nystagmus to the right ; left abdominal reflex absent ; left knee-jerk accentuated ; plantar reflexes flexor.

Second operation : Temporo-sphenoidal lobe explorations.—The original operation wound was reopened : the dura was found

https://doi.org/10.1017/S0022215100041529 Published online by Cambridge University Press

bulging. On exploration with a needle, pus was found at a depth of three-quarters of an inch. (Culture : *Staphylococcus albus*, pneumococci, and *B. proteus*.) The dura was incised, and the abscess was drained and a drainage-tube inserted. Lumbar puncture : fluid clear ; under pressure.

Post-operative history.—Consciousness soon returned, and the patient was able to recognize people and objects ; the papillœdema slowly subsided. On *May 4th*, however (eight days after the second operation), the drainage from the abscess practically ceased and the patient again became drowsy. She could be aroused with difficulty and took no food. The temperature remained normal, but the pulse was slow, varying between 60-70. Lumbar puncture : Cerebrospinal fluid slightly yellow ; a little blood present ; protein, 0·08 per cent. ; globulin present in excess ; chlorides, 770 mg. per 100 c.mm. Cells, 70 (practically all lymphocytes) and 150 red blood-cells per c.mm. No tubercle bacilli found ; culture sterile.

The drainage-tube in the abscess cavity was manipulated, and a great flood of pus was obtained. The drowsiness soon disappeared and the patient made an uneventful recovery.

Streptococcal Meningitis secondary to Acute Suppurative Otitis Media : Simple Mastoidectomy. Recovery.
—E. WATSON-WILLIAMS, M.C., F.R.C.S.Ed.

Eva B., aged 49, complained on *July 2nd*, 1933, of pain in and behind the right ear, and in the throat, during three days, after a bad cold ; had never had any previous ear disease. The day before admission she was giddy, walking towards the right, and seeing " things go round clockwise ". On the day of admission she was very giddy, and vomited several times. The ear had begun to discharge thin bloody serum, and was painless, but she had a terrible neuralgic headache (subject to this) across the front of the head. Quite sensible, but drowsy, and preferred to lie on the right side ; slight right facial weakness ; well-marked rigidity of neck, no ataxia, no past-pointing ; plantar reflexes normal, right knee-jerk normal, left absent, pupils normal. Tuning-fork not heard at all at meatus ; on mastoid heard less well than normally. Weber referred to left. Rather slow, coarse mixed nystagmus to left. Temperature 102° F. ; pulse 100. Caloric test, water at 107° F. in right ear ; after 2′ 40″ horizontal nystagmus to right on looking to right, but still some mixed nystagmus to left on looking to left. Lumbar puncture ; pressure much raised, fluid turbid. Report : " Purulent fluid containing pus cells and streptococci." Later, streptococci cultured. Immediate operation, right simple (Schwartze) mastoidectomy. The bone was of very cellular type, dripped blood ; cells full of old blood ; at tip some contained a little thin sero-pus ; antrum empty. Lateral sinus, dura apparently normal. (Streptococci grew from

https://doi.org/10.1017/S0022215100041529 Published online by Cambridge University Press

pus.) " Bipp " and loose dressing ; 30 c.cm. antiscarlatinal serum intramuscularly.

July 3rd: General condition unchanged ; very thirsty ; nystagmus, facial paresis less ; temperature 100° F. Lumbar puncture, after intravenous injection of 40 c.cm. collosol argentum ; magnesium sulphate per rectum. *July 4th*: Same treatment. *July 5th*: Enormously better, temperature 99·2° F. No headache, little neck rigidity. *July 6th*: Treatment as on *July 3rd*. Convalescence uneventful.

September, 1934 : Healed ; some deafness and humming tinnitus on right side.

November, 1934 : No change ; still has severe headaches at times as before the attack : recent exploration of nasal sinuses negative.

ABSTRACTS

EAR

The caloric reaction in Morbus Ménièri. A. THORNVAL, Copenhagen. (*Acta Oto-Laryngologica*, xx., 3-4.)

The caloric tests in cases of *Morbus Ménièri* were carried out using water at 7° C. above or below rectal temperature, and with patients lying down in the prone or supine position. The irrigation apparatus held a thermometer which had been built into it so that the temperature of the water could be accurately observed as it flowed into the ear under examination.

In these investigations an increased caloric reaction was never observed in the affected ear, but a difference in the reaction between the diseased and healthy sides was found in a number of the cases. This difference might not have been observed except under the particular conditions described in these experiments.

The writer considers that the question might be asked how, on the basis of these observations, may we explain the violent paroxysmal attacks of patients with *Morbus Ménièri*. He gives the following reply to this question—" Ménière attacks are brought on by a sudden, often almost explosively developing hyperfunction of the vestibular system on one side. For analogy, consider a periodic neuralgia in a nerve such as the trigeminal ; the slight decrease in function, shown in certain cases, may correspond to a mild anæsthesia in the peripheral area supplied by the nerve."

H. V. FORSTER.

https://doi.org/10.1017/S0022215100041529 Published online by Cambridge University Press