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Discussion on the Treatment of Endocranial Complications of Otitic Origin

By E. D. D. DAVIS

A brief review of the treatment of abscess of the temporo-sphenoidal lobe of the brain arising from the ear.—BY reviewing the treatment of a temporo-sphenoidal abscess I hope to demonstrate that this abscess should be approached and drained from the mastoid area. If drainage fails to achieve a satisfactory result in a very few chronic encapsuled abscesses then enucleation may become necessary.

The drawings of three specimens are shown to illustrate the pathology of a brain abscess. The first two are frontal lobe abscesses because they are the best specimens I could find in the London Pathological Museums.

The first drawing (Fig. 1) is a large abscess due to a sharp slate pencil which had penetrated the roof of the anterior cells in the lateral mass of the ethmoid. The point of the pencil projected into the anterior fossa of the skull for 2 cm. The patient was a boy of 17 who was comatose when admitted to hospital and in consequence it was not known when the slate pencil was pushed into the nose. The boy fell on the pavement on the way home from school, he was unconscious for a few minutes and his neurological symptoms commenced from that day five years before he was admitted to hospital comatose ; hence it is probable that the abscess is of about five years' duration. The dura mater and arachnoid in relation to the slate pencil were thickened, opaque and adherent to the brain but there was no subdural or extradural abscess. The abscess cavity measures 4·5 by 5 cm. It is irregular in shape and partly enclosed by a fibrous wall or so-called capsule surrounded by œdematous brain substance. Toward the outer side a recent extension of the pathological process has occurred where a considerable amount of brain substance is seen in various stages of necrosis. In this position a loculus is forming and it is found frequently that a temporo-sphenoidal abscess extends in this way in its deepest aspect towards the descending horn of the lateral ventricle. The formation of a capsule appears to depend on the resistance of the patient and on the virulence of the infecting bacteria. The abscess is more likely to be walled off when the infection is caused by the *Bacillus proteus*, *Bacillus coli* or staphylococci than by the virulent streptococcus or pneumococcus. An

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encapsuled abscess is more common in the frontal lobe than in the temporo-sphenoidal. More frequently a foreign body such as a fragment of bone or a missile lodged in the brain is walled off and may cause an abscess some time later. The pulsation of the brain tends to extrude the encapsuled abscess particularly if the capsule is uncovered by removing a cap of the adjacent brain tissue from its surface. A few but not all chronic abscesses may become encapsuled and it is stated that the capsule may be firm enough to permit enucleation in from eight to twelve weeks. During this period the patient may succumb to meningitis, rupture of the abscess into the ventricle or from œdema and encephalitis, the usual termination of a fatal brain abscess. Moreover multiple abscesses, deep loculi and recent extensions make enucleation impractical and not without risk. The specimen shows a marked enlargement of the left cerebral hemisphere as a result of œdema. The anterior horn of the lateral ventricle is obliterated and the basal nuclei are distorted by compression. This patient died of œdema of the brain and encephalitis with Cheyne-Stokes' breathing. The second specimen, not illustrated, shows multiple abscesses arising from chronic suppuration of the frontal sinus and the damage done by a drainage tube introduced into the brain (see colour plate, *Proc. roy. Soc. Med.*, 1934, xxvii, 1,286). It emphasizes the necessity of placing the drainage tube accurately into the cavity of the abscess and of anchoring the tube by two stitches to the deep fascia reinforced by a firm and suitable dressing. As a house surgeon I assisted my chief, a general surgeon who at one time was assistant to Macewen, at this operation and there is no doubt that the large drainage tube was introduced into the cavity of the abscess and anchored by a stitch. It is presumed that the pulsation of the brain displaced the tube. The pulsation of the frontal lobe is always more marked than that of the temporo-sphenoidal. This patient died of cortical meningitis which was present before the drainage operation was performed.

Fig. 2 indicates the commonest site of a temporo-sphenoidal abscess of otitic origin. It is immediately above and behind the roof of the mastoid antrum with a stalk approaching the surface. It is probable that this abscess would have been missed by an exploratory burr hole or even by an osteoplastic flap which is usually made in the temporal fossa above and in front of the ear.

Fig. 3 illustrates the approach to a temporo-sphenoidal abscess through the mastoid process.

The mastoid operation is done without the mallet by using a skull gouge with the hand and a nibbling forceps. When a diagnosis of an abscess has been made or suspected, the dura is thoroughly exposed by removing the roof of the mastoid antrum and tympanum, then a burr hole is made in the squama of the temporal bone. The bony aperture is enlarged in all directions by nibbling forceps and there is no limitation to the extent of the area of dura exposed. The wound and the area are thoroughly cleansed by ether and a 1 : 1,000 flavine solution. Clean towels, clean gloves and instruments are now used before the dura is opened. The mastoid wound can be cleansed just as efficiently as the skin of the scalp over the temporal area where the neurosurgeon approaches the abscess away from the ear. This point is proved by the fact that the temporo-sphenoidal lobe has been explored on many occasions without any sepsis or harmful results. The exposed dura is isolated

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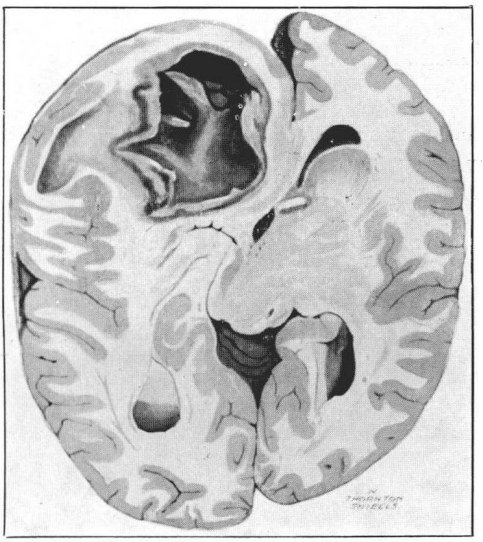


FIG. 1

Large abscess in the left frontal lobe of the brain, arising from a foreign body in the nose (Charing Cross Hospital Museum), *see also* colour plate *Proc. roy. Soc. Med.*, 1934, xxvii, facing p. 1,288.

[*face p.* 130

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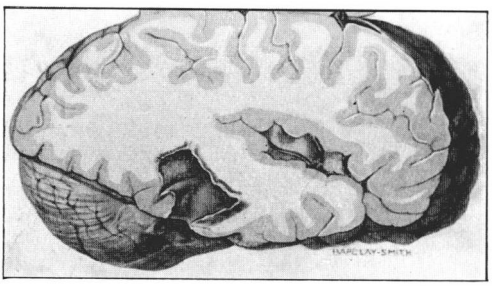


FIG. 2

Brain in section, showing space left by encysted abscess in temporo-sphenoidal lobe. (Copied from William Macewen, *Pyogenic Infective Diseases of the Brain and Spinal Cord*, 1893, p. 94, Glasgow.)

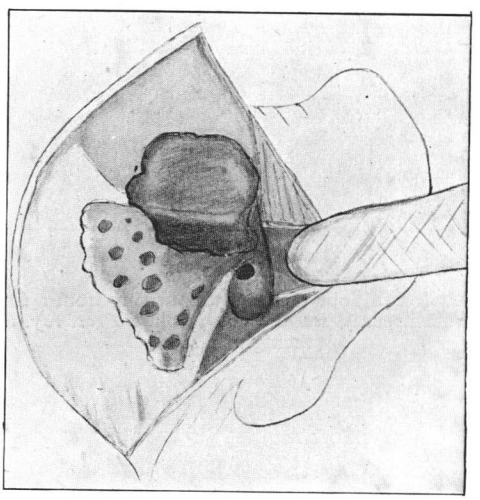


FIG. 3

Second stage of the operation in which the two openings in the skull are converted into one. (After Hunter Tod. Burghard's *System of Operative Surgery*, 1914, p. 309, London.)

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and its blood-vessels compressed by packing sterile gauze between the bone and the underlying dura. It is desirable to follow the track of infection from the mastoid to the brain. Therefore a small crucial incision is made in the dura using a dura mater hook in the area where the dura is thickened, covered by granulation tissue or by an extradural abscess. If the dura is normal in appearance the incision is made in the area covered by the roof of the mastoid antrum. If an abscess is present, in my experience pus escapes with the first thrust of a large needle or sinus forceps. Care should be taken not to lose the abscess and the needle is left in situ until the largest size aural speculum or an encephaloscope is slipped over the needle into the abscess cavity. Pus and debris are removed by gentle suction. If irrigation is employed it must be gentle and Ringer's solution or blood plasma should be used through the speculum. Weed and Wegeforth (1919) of the Rockefeller Institute injected the subarachnoid space of cats with antiseptics and found that chloramine, flavine and mercurochrome caused intense reaction and convulsions. Isotonic saline alone caused toxic and convulsive effects but Ringer's solution is tolerated well. Two small rubber drainage tubes stitched together are inserted into the abscess cavity through the encephaloscope. The tubes must be firmly anchored by stitches and by a reinforced firm dressing of rolls of gauze built up round the tubes to hold them in position. Various types of tubes and methods of drainage have been advocated. Packing the abscess cavity with gauze does more harm than good and should be avoided. Lemaitre's method of introducing the smallest drainage tube followed at frequent intervals by larger and larger tubes is unsatisfactory. If drainage tubes could be avoided results might be better. The dressing is untouched for six days and at this, the first dressing, preferably done in the operating theatre, the drainage tubes are removed and if there is no escape of pus the wound is allowed to close even at the risk of having to open the abscess again.

The advantages of approaching the abscess from the mastoid area as described are: (1) The source of infection can be eliminated. Extradural abscess and lateral sinus thrombosis, when present, are dealt with. Moreover the condition of the dura found at this operation will either confirm or correct the diagnosis. In some cases the important early diagnosis cannot be made without exploration. (2) The track of infection is followed where the spread of infection in the subarachnoid space is prevented by adhesions. Two cases seen by me in which an osteoplastic flap was made in the temporal region died of meningitis. (3) The lowest point of the abscess and where it is nearest to the surface is drained. (4) The area of operation can be sterilized as well as any other part of the skull.

Treatment by aspiration without any form of drainage between the tappings has been condemned by Cairns (1934). The abscess cavity fills remarkably quickly, especially when there is any œdema of the brain. Aspiration of an abscess in other parts of the body is not a satisfactory procedure.

Enucleation of the abscess is possible only when the abscess is completely walled off by fibrous tissue. In a few cases encapsulation takes eight to twelve weeks during which time the catastrophe of meningitis, rupture into the ventricle or œdema of the brain may occur. Loculation and particularly multiple abscesses make enucleation difficult. When the neurosurgeon found

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enucleation difficult and unsafe he has resorted to marsupialization. There is no evidence to show that epilepsy is more common in abscess which has been drained than that subjected to enucleation. If neurological signs and symptoms persist after drainage or if there is a permanent sinus then enucleation is probably desirable.

Marsupialization is done on rare occasions as an expedient when enucleation is unsafe or impracticable. It has always been an unsatisfactory operation for cysts in other parts of the body when they could not be dissected out.

The results of various methods of operation are difficult to express in figures. The large majority of brain abscesses are not seen until the patient is drowsy, comatose or in the terminal stage and are complicated.

The *Proceedings* of the Otological Section records a large number of successful cases treated by drainage only. At one meeting in 1927 18 successful cases were recorded and whenever the subject of brain abscess has been discussed a number of successful cases are shown.

Results of operation.—Macewen, 80 per cent. survived. Neumann, 47 per cent. uncomplicated cases survived; no organisms in C.S.F. Fraser, J. S. (1930), 17; 6 cases survived, 33 per cent. approx.; 6 meningitis before operation. Davis, 28: 4 uncomplicated cases survived. 24 post-mortem complicated cases.—11 meningitis before operation; 7 ruptured into the ventricle; 4 œdema of the brain; 2 multiple abscesses.

Northfield's (1942) analysis of 24 cases of brain abscess: 9 cases drained, 2 survived—cause of death not stated; 15 cases not drained, 10 survived = 8 enucleated, 2 aspirated. The 7 drained cases which were fatal may have had meningitis or some terminal complication before being drained. It is assumed that the 8 successful cases of enucleation were encapsuled and therefore were more favourable than acute cases.

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F. W. WATKYN-THOMAS

Labyrinth and petrous infections.—Although these are not intracranial infections themselves they are so frequently a cause of such infections that it is fitting to consider them here. Under modern surgical conditions neither of these complications is often seen.

For practical purposes the problem of the infected labyrinth is a problem of meningitis. It is remarkable how seldom labyrinthine infections cause cerebellar abscess. This statement is upheld by the figures of Zange and the statistics of the Edinburgh Royal Infirmary. Lateral sinus thrombosis as a result of labyrinth infection occurs as a curiosity. (I have only seen two.)

Extension from the labyrinth may be through the internal auditory meatus (usually), through the bone (sometimes), along or through the aqueduct of the endolymph (rarely), and through the aqueduct of the perilymph (perhaps).

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The treatment of labyrinthine suppuration is entirely conditioned by our view as how best to avoid such intracranial invasion.

If the labyrinth is alive, it is most unlikely that it is infected. If therefore we find a fistula—or, more accurately, an erosion of the labyrinth wall—with an active labyrinth, especially if the so-called fistula sign is present, that labyrinth should be left severely alone. The proper treatment is the adequate mastoid operation and rigid non-intervention as far as the labyrinth is concerned.

If the labyrinth is so very dead that it is a sequestrum, of course it has to come out.

Last, if the labyrinth is dead and the patient already has meningitis the general opinion is that the labyrinth is a probable—or at any rate a possible—focus, and should be drained accordingly. I am not referring here to trans-labyrinthine drainage of the cisterna pontis ; that is quite a different problem.

The principal difference of opinion is what to do when the labyrinth has been killed in the course of a suppuration and the patient has not yet got meningitis. Two conditions are recognized : (1) In the course of a middle-ear suppuration the labyrinth is destroyed by infection, with all the characteristics of a " labyrinth storm ". (2) The patient has a suppurating ear, totally deaf, and dead to caloric tests. Sometimes he can remember severe vertigo, perhaps diagnosed as " gastric influence ", somewhere in the remote past. Sometimes he cannot even remember that. The first condition is " manifest labyrinthitis " ; the second " latent ". In manifest labyrinthitis without meningitis we may safely assume that, as the labyrinth has been destroyed by an aural infection it is itself infected, and so is a danger to the meninges. According to one view, once the labyrinth has been so killed it is a potential danger and should be drained forthwith. Granted that we cannot be certain that it is a suppurative rather than a serous labyrinthitis, it is unusual for a serous labyrinthitis to show complete loss of caloric response and total deafness ; if it should, it is highly unlikely that any useful hearing will survive. The other view is that by early operation we may precipitate the very disaster which we wish to avoid, as well as destroy a labyrinth still capable of recovery. Therefore, do not drain the labyrinth until there is definite evidence of meningeal irritation, e.g. a rise, however slight, in the cerebrospinal fluid cell count.

My personal experience, from 1920 to about 1940, was that of all cases of suppurative labyrinthitis which came into my hands, *before any meningeal signs could be detected*, I lost only three—one, an anæsthetic fatality due to unsuspected myocardial degeneration, one by a spreading necrosis of neighbouring bone (the only case of the kind I have ever seen) and one where I persuaded myself that it would be safer to wait a few days. I was wrong, that patient died of meningitis—I do not say it was cause and effect, I hope it was not, but that was only one in the series. Now, with sulphonamides available, if meningitis should appear there is a good weapon at hand to deal with it, because the bony labyrinth is the only bony cavity in the body where sulphonamides do not act at a great disadvantage. We know that they freely enter the cerebrospinal fluid, and we know that the perilymph is in continuity with that fluid, therefore we can reasonably hope to destroy the organisms in the labyrinth by chemotherapy. At present I feel that the right procedure

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when labyrinthitis appears in the course of a chronic, a recurrent, or a subacute suppuration, *provided always that there are no signs of meningeal irritation as proved by lumbar puncture*, is to load the patient with sulphonamides and, when all is quiet, to do the radical operation and to complete it by opening the labyrinth, just as one opens up any other cell system in an infected area. You may have noticed that I have excluded acute suppuration. I do not believe that the acute fulminant condition in which middle ear, labyrinth and meninges are invaded almost simultaneously is primarily an ear condition at all; I think it is a blood invasion and should be treated as such. Here the aural tract is the victim, not the focus, and opening and draining it will be quite useless. For drainage of the labyrinth I have nearly always done the " double vestibulotomy " which is safe and simple. I have done the Neumann operation, but I do not find it either safe or simple. In the actual technique of the operation I think it is most important to have really sharp chisels and gouges. I have not yet had the good fortune to use an operating microscope but I have found v. Eicken's magnifying loupe useful, but cumbersome. The danger to the facial nerve is—it seems to me—usually at the postero-inferior corner when one is working on the recess of the round window. Injury to the columella should be avoided, as the area is flooded with cerebrospinal fluid. I have seen this happen once or twice, but it has never done the least harm to the patient—all the same it is an accident to be avoided.

In latent labyrinthitis the operative findings dictate the procedure on the labyrinth. If the whole wall is covered by scar tissue, leave it alone; if there is a fistula into the dead labyrinth, enlarge it for drainage; if the labyrinth has sequestrated, take it out.

In the last two years I have not used translabyrinthine drainage for meningitis of labyrinthine origin, because I think that the continual escape of cerebrospinal fluid makes it more difficult to maintain the requisite high concentration of sulphonamide. That does not alter my opinion that, in its day, it was the greatest advance in the surgery of the condition.

By petrous invasion I mean any extension of disease into the body of the petrous; I most emphatically do not limit it to invasion of the apex, which I think is uncommon. About 35 per cent. of adult petrous bones are fully pneumatized, and in most of them some marrow persists. But, whether pneumatized or not there is a hard shell to the bone, with an excellent blood supply, and sequestration or perforation into the cranial cavity is rare. Also, the more complete the pneumatization the more complete the obliteration of the vessels passing from dura to bone and the less the risk of spread by these tracks; on the other hand, in the non-pneumatized bone the marrow is capable of stout resistance. For these reasons a petrous infection can usually be treated with deliberation.

A considerable majority of petrous invasions clear up entirely with an adequate mastoid operation and careful follow-up of any deep cell tracts—especially the group of cells in the petrous angle. I have seldom seen the sublabyrinthine cells. I think the rarity of the condition is a testimony to the care with which we do our acute mastoids. Of course there are cases in which the dangerous cells are so shut off by bone that they are absolutely undetectable at a first operation; these are the cases where petrositis develops

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some days or weeks after a mastoidectomy. In such cases the onset is seldom dramatic, an increase of discharge, a recurrence of pyrexia, sometimes—by no means always—retro-orbital pain, are the usual signs. Odd little disturbances of the labyrinth are not uncommon. Occasionally there is just the suspicion of a facial weakness. I do not think that in these cases operation is indicated immediately; it may not be indicated at all. Radiographs of the petrous pyramids should be taken; the optic discs should be watched most carefully; at the slightest suspicion a lumbar puncture should be done—and on no account whatever should we yield to temptation and mask the signs by sulphonamides. If there is any evidence of meningeal irritation, operation must be done forthwith; otherwise, unless pain and headache increase, or the pyrexia continues I am inclined to wait. The longer one can wait the more likely one is to find a track of carious bone and infected cells to follow. I do not think that a VIth nerve palsy is necessarily a proof of apical invasion, but whenever I have seen this sign in a case of petrositis I have always found other indications such as increasing pain, for operation. As to the method of operation I have always said that we are not engaged on the anatomical exercise of finding the apex, we are doing the surgical routine of hunting the pus, and our object is to protect the meninges. For this reason I have never done the Almour-Kopetsky operation. Although this is a most ingenious route to drain an abscess of the apex, it would not afford adequate drainage for the even more dangerous extradural abscess. The Ramadier operation has the same disadvantage, and as well seems to me unnecessarily dangerous. I have either followed up any available track, or where I could find none, have done the Voss-Ruttin-Eagleton method of unlocking the petrous. I have had no personal experience of approach by the endaural method, nor have I ever succeeded in finding a track through the arch of the superior canal, which Paul Frenckner has described. On three occasions I have seen a forward extension, probably through the zygomatic cells. On a previous occasion I have mentioned invasion of the superior petrosal sinus and spread to the lateral, but I have not seen such a case since these were reported. I have never myself seen an extension into the pharynx, but there certainly are such things. The possibility of approach to the petrous by a nasopharyngeal route has been suggested by Russell Burke, but as far as I know it has only been worked out on the cadaver.

One thing I wish to emphasize most strongly, that in petrositis uncomplicated by meningitis, sulphonamide treatment is not only absolutely useless, it is abominably dangerous. It is useless because the drug cannot reach the organisms, it is dangerous because it masks the symptoms and because it may, by useless over-dosage make it impossible to give the heavy doses which may be needed later if meningitis should develop.

In conclusion, may I express my deep gratitude to my colleagues who have so kindly let me see so many of these cases.

Mr. TERENCE CAWTHORNE: Meningitis was formerly the commonest cause of death in ear, nose and throat diseases. Courville of Los Angeles, in an analysis of 15,000 post-mortems, found 337 cases of septic meningitis, that is to say, meningitis due to organisms other than tubercle bacillus and meningococcus; of these 175 were otogenic. Whilst the mortality rate of meningitis

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has been dramatically reduced since the introduction of the sulphonamides it may still be the commonest cause of death.

The basis of any study of otogenic meningitis should be a consideration of the pathways by which infection may spread from the ear to the meninges, because an appreciation of these methods of spread is the key to the whole situation from the surgical point of view.

It will be convenient to consider the spread of infection from the ear to the meninges under three main headings: (*a*) preformed spaces; (*b*) vascular channels; and (*c*) direct spread through diseased bone.

Preformed spaces.—These can be preformed by Nature, by injury or by operation. There are many natural spaces in the temporal bone through which infection can travel, and the petro-squamosal suture when patent and the various labyrinthine channels deserve particular mention. Injury to the skull may open up a channel between the subarachnoid space and the outside *viâ* the ear through which infection can enter if proper precautions are not taken.

Mastoid cavities, particularly if the dura has been exposed, can sometimes be a way by which infection spreads, particularly if the dura has been roughly handled. I remember a case in which I did an extensive operation of the Neumann type for streptococcal meningitis secondary to ear disease. About a year later this patient developed a severe upper respiratory infection, and within twelve hours she was delirious. The cerebrospinal fluid was found to be teeming with pneumococci and the infection had clearly rushed through the upper respiratory tract to the mastoid cavity with its widely exposed dura. She recovered from this second infection.

A characteristic feature of this spread by preformed spaces is that the meningitis appears within a matter of hours.

Vascular channels.—The second group in which infection may spread from the infected middle-ear cleft to the meninges consists of those in whom the spread is by vascular channels. I do not necessarily mean the lateral sinus or any of the large blood-vessels, but would include the small veins. There are many intimate connexions between the venous drainage of the temporal bone and the meninges, and spread in this way probably accounts for those cases of meningitis which occur within a few days of the original otitis media.

Bone destruction.—We are all familiar with the patient with an otitis media who develops meningitis, either before or after a mastoid operation, perhaps two or three or more weeks after the otitis media. This third group, of course, consists of those to which Mr. Watkyn-Thomas has just referred. I do not think as a rule they occur within less than two weeks from the primary infection.

Then there are the cases of long-standing otitis media which give rise to meningitis. In a way these belong to the group of cases arising from preformed spaces, because the disease may have been slowly eroding the bony boundaries of the middle-ear cleft for years, resulting in an exposed dura and the patient at the mercy of any upper respiratory infection.

Thus meningitis appearing within *hours* of the primary aural infection is likely to have travelled by preformed spaces; within *days* by vascular channels; and within *weeks* or longer by bone destruction.

Nomenclature.—Such tangled verbiage as "sympathetic meningitis",

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" meningismus ", " méningite de voisinage " and " meningeal reaction " are used to indicate what are all part of one disease, namely meningitis.

I think, therefore, that it is better to call all these cases meningitis and then, when the cerebrospinal fluid has been examined, the qualifying adjective can be added—" serous " if there is only an increase in pressure ; " cellular " if there are cells, and if in addition to an increase of cells there are bacteria, it can be called a " bacterial meningitis ", at the same time naming the bacterium.

I have mentioned pressure, cells and bacteria only because these are the three essential features about which information is sought when the cerebrospinal fluid is first examined. At that time or later additional information may be desired such as chlorides, sugar, protein and sulphonamide content.

Certain types of meningitis, especially if they occur following long-standing suppurative otitis media, where the labyrinth has been invaded, show a fairly high proportion of lymphocytes in the fluid, and it may well be that the number of lymphocytes furnishes some idea of how long the infective process has been going on.

Treatment.—Before anything else is done the patient with meningitis requires chemotherapy. But there are certain cases which require surgery in addition, namely, the third group of which I have spoken, in which there is bone destruction. I think that only an otologist can decide which cases require surgery and to overlook such cases is to invite a relapse, possibly fatal. It is not always easy, when a patient has a discharging ear and meningitis, to decide whether or not there should be an operation. An otologist is in the best position to decide this, particularly if he can think in terms of the type of infection and the way it has spread. In fulminating cases the spread to the subarachnoid space is rather like the spread of fog into a house, underneath the door and through any crevice, and to undertake extensive mastoid surgery will not hinder the spread but will jeopardize the patient's chance of survival. The same applies to the cases of vascular spread and I feel that early mastoid surgery should not be embarked upon in any case of meningitis following within a few hours or days of an otitis media.

But in the third type of case, where there is disease of some weeks' standing, mastoid surgery with removal of diseased bone is required, and the only question there is WHEN. These cases are on the whole better left for a day or two—it is difficult to lay down any hard and fast rule. I now think that it is best to start a course of one of the sulphonamides before operating. My experience with sulphadiazine is small, but from what I have read I am led to believe that it is probably the best of the sulphonamides to use. Nor have I any personal experience of penicillin in meningitis. The one drawback to penicillin is that it has to be given intracisternally or intrathecally.

As to the introduction of various sera into the thecal space I have always felt very strongly that the less one introduces into the theca or cisterna the better, for with but few exceptions—and I believe penicillin is one—intrathecal medication does more harm by irritation than good by combating infection.

Mr. F. McGUCKIN : My interest in brain abscess was aroused in dramatic fashion some fifteen years ago, when I watched a lateral ventricle rupture

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through a temporal lobe abscess, the peripheral wall of which had just been incised. This was the first of three such cases published in the *Lancet* in 1936. All three recovered. By 1935, 100 cases of abscess had been summarized, 39 of which were personally surveyed in part or in whole. Though interest remained, enthusiasm was curbed, first by the ups and downs of good and bad run of cases, and secondly by the unfortunate sequelae, fits for example, in some of the recovering temporal cases.

In the series, the causes of death fell roughly into two groups: those from diffuse cerebrospinal infection usually by ventricular erosion, and those resulting from pure pressure exerted finally on the medulla. Correct timing of intervention, if this were possible, was clearly the preventive treatment of the first accident. The pressure factor suggested the cardinal importance not merely of timing but of so limiting intervention as to avoid increase in tension from traumatic œdema.

The study of the morbid anatomy of brain abscess is important. The part played by the Virchow-Robin space in the transmission of temporal abscess is clear enough; though I think not enough attention has been given to the superior petrosal sinus or to thrombosis of veins included in a localized patch of leptomeningitis.

Up to 1935 I had not personally surveyed a single case of cerebellar abscess, alive or dead, in which a labyrinthine transmission was proved to my satisfaction, and in the 100 cases already mentioned there were but 2 out of 44 cerebellar abscesses in which a labyrinthine origin was clearly demonstrated, one *viâ* the posterior semicircular canal and one *viâ* a saccus empyema. In my experience lateral sinus thrombosis has accounted for nearly half the cases, and it was responsible for both my cases of non-suppurative cerebellitis. The mechanism is that of retrograde thrombosis of communicating veins. On the score of the mechanics of internal hydrocephalus, which may be extreme in temporal lesions, tribute must be paid to the brilliant work of Jefferson and Rowbotham on tentorial pressure cone due to herniation of the uncinate process of the temporal lobe. This work, for me, ended ten years of confusion about the matter.

I believe there are three features which suggest that the otologist may justly retain some place in the conduct of this disease. First, the mastoid approach sometimes locates an abscess with very great accuracy and often with much greater accuracy than the neurosurgical approach. Moreover there is a fair chance of crossing an adherent subarachnoid space. Secondly, the otologist may with very little trauma and minimal reactionary œdema, tide over the patient, for subsequent drainage or excision, by the simple process of tapping and re-tapping. Thirdly, in a notable minority of cases the neuroglial agglomeration and fibroblastic proliferation will have produced an encapsulation sufficient for minimal surgery and at the same time insufficient mural rigidity to prevent obliteration of the cavity on emptying. Such cases may recover on simple tapping.

There are two cardinal principles in the conduct of the acute phase of brain abscess: (1) Interference should be properly timed; and (2) it should be as little as may be and as gentle as possible.

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In labyrinthine invasions, I find chronic otitis media and especially cholesteatomatous lesions a frequent cause of erosion *viâ* the lateral and superior canals and less commonly through the promonotory. In many cases I have sucked cholesteatoma from the labyrinth and in not a few found that organ virtually absent, though the facial nerve has usually held its own throughout. I am not convinced that this route, in such chronic and resisted lesions of this kind, accounts for more than a small proportion of leptomeningitic cases falling within my experience and it has been my custom simply to deal with the labyrinth secundum artem during the course of operation. Acute purulent labyrinthitis has been a rare disease among my cases, perhaps because conservatism showed so many invasions, apparently catastrophic, to recover some function quite quickly. Acute invasions, however, are clearly to be feared in relation to meningitis because of the preformed paths but it is pertinent to mention that these paths cut both ways, e.g., the frequency of cochlear destruction in meningococcal invasions. I suggest that it is quite possible for an otogenic meningitis to invade a labyrinth *viâ* the nerve sheaths, and that scrutiny of most careful kind is necessary before accepting a post-mortem labyrinthitis as necessarily indicating the route of invasion.

If all acute ears were X-rayed, petrositis would be established with a frequency quite out of proportion to its real significance. This is but another example of the need to interpret X-ray shadows by the light of clinical experience. This entity can be recognized quite frequently and indeed one's clinical estimate may often be supported by serial X-rays but I do hold that the important partner is the clinical estimate. Potentially dangerous though the condition may be, it is a fact that a relatively small proportion of cases develop real complications and I believe that the mortality attending unrestrained surgery is a greater danger. The majority of cases will react to a careful mastoidectomy, with an odd case here and there calling for a little investigation of the deep track.

Mr. ERIC WATSON-WILLIAMS said that he was amazed to hear of an abscess being tapped three or four times. His own practice had been, when he had found an abscess, to put in the smallest drain he could. He had had a certain number of recoveries by following that method.

He could not see the object, if there was a dead ear, of leaving the suppuration in the labyrinth undrained. The sulphonamides would do a great deal of good, but not too much must be expected of them. One point of importance to the patient was that if the ampulla were carefully curetted he woke up from the anæsthetic completely recovered from the vertigo. He had had one case in the previous week, and what impressed the patient who had had five days of acute labyrinthitis was that he woke up without any sense of giddiness.

In meningitis, again, the sulphonamides did much, but there were cases in which the meningitis recurred. It was very important to drain—he did not mean through the internal auditory meatus, for that was only very rarely indicated—that is to say, to get proper drainage of the infected focus. Before the days of sulphanilamide he had quite an encouraging number of recoveries simply from early operation, with the giving of intravenous silver in large doses.

Mr. J. N. DEACON referred to two cases of acute mastoiditis, each with

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meningitis which had developed in spite of heavy dosage of sulphonamide given prior to admission. In each case organisms were found in the C.S.F. and pus in the mastoid. Penicillin 10,000 units in 1 per cent. glucose was given intrathecally at the time of the mastoid operation and continued daily. The temperature fell to normal in forty-eight hours, in one case after two injections and in the other after three. They had now had six and seven injections respectively, and were doing remarkably well. He was considering now taking them off penicillin.

Mr. DONALD WATSON said that he had started the study of labyrinthitis with J. S. Fraser twenty-four years ago and, with his help, learned a great deal about it. After two years there he went down to Bradford and carried out the teaching which he had learned in Edinburgh, but in the course of four years in Bradford he had three deaths from meningitis following labyrinthitis. On the other hand his colleague had no deaths from meningitis following labyrinthitis. He discovered that the one respect in which his colleague's practice differed from his own was that his colleague did not test any labyrinth. He thereupon followed the same course in those cases where pus was present, and now for eighteen years he had not tested cases with labyrinthine symptoms when infected, and he had had only one death from meningitis which had been preceded by labyrinthitis. That one case was of the fulminating type mentioned by Mr. Watkyn-Thomas in which the mastoid labyrinth and the meninges were all infected, in a child of 6. This had been a negative kind of research but he would make a plea that any case of labyrinthitis should be treated very conservatively and rested, and then, when the symptoms had disappeared, one could use one's surgical judgment to do what was thought to be required.

Major E. P. FOWLER, Jr., U.S.A.M.C., said that in an analysis of anatomical specimens the commonest site of invasion of the meninges was through the wall of the posterior fossa. The invasion was directly through the back of the petrous bone and, most commonly, through a cell which was very close to the anterior way of the internal auditory meatus.

He thought that surgery was necessary with meningitis, though he agreed that it was not important to carry it out as quickly as they used to do. Infection in the temporal bone should be drained. However, if one operated on a mastoid too soon it was likely to spread. On the other hand, the making of a small hole into the mastoid would relieve the pain and give adequate drainage when myringotomy was inadequate. It was a very simple procedure to increase the drainage in this way and not dangerous. With sulphonamide therapy it was sometimes unnecessary to do a complete mastoidectomy at a later date but this was not the rule.

Colonel NORTON CANFIELD, U.S.A.M.C., said that for several years past at his hospital they had worked on their brain abscess cases in conjunction with the neurosurgeons. The location for the drainage had been a source of considerable discussion as to whether it should be through the mastoid or through a posterior incision entirely outside the field of mastoid operation. If the brain abscess were given time enough to become encapsulated the results seemed to be better with the draining of the abscess by a second incision over the abscess site, not through the mastoid wound. He had no figures at the

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present time to bear that out, but it seemed that the handling of the dura and the brain substance itself, with the wide drainage of the abscess which could be done in that manner—and could be done by the otologist if he was able to handle brain tissue—gave better results in these cases.

He wanted to emphasize again the point concerning the testing of the labyrinth by stimulation in the presence of infection. He had not carried out that procedure because of the possibility of increasing the intratemporal bone pressure. He wondered whether Mr. Watkyn-Thomas had found cases in which it had been followed by a severe reaction.

Another way of cold caloric stimulation of the labyrinth in the presence of pus was to use air and it would then be possible to decrease the temperature without the disadvantage of introducing cold water.

Mr. Cawthorne's presentation had opened up a good many points which might be discussed. He was of opinion that the fulminating cases which occurred in certain patients belonged to a group in which the middle ear and temporal bones constituted only a portion of the entire picture, and it was not necessarily the fact that the meningitis made its appearance in the meninges by means of or through the ear. To give such cases a sulphonamide drug was one of the greatest protective measures at their command.

Mr. W. Stirk Adams described a case under the care of his colleague Dr. J. M. Smellie, in the General Hospital, Birmingham, some years ago, which supported the suggestion, raised during the discussion, that caloric tests carried out in the presence of labyrinthitis prejudiced the recovery of the patient.

The patient was a young man in the early twenties who was admitted to hospital for investigation of a left facial paralysis. His history was that six years previously a paresis of the left side of his face appeared after taking part in a cycle track race. The paresis disappeared within twenty-four hours, but recurred after every race, at first disappearing rapidly, but in the course of time taking longer to recover. He gave up racing for this reason some eighteen months before admission, but his facial paralysis did not recover and persisted unchanged until his admission for investigation.

General and neurological examination was negative apart from the paralysis of his left facial muscles, and deafness in his left ear. In this he was only able to hear a whisper at 1 in. distance. His left drumhead was intact, retracted, and almost immobile. Caloric tests were carried out to ascertain whether his labyrinthine function was affected. The evidence of the labyrinth tests and the presence of residual hearing proved a live labyrinth.

Within twenty-four hours of this caloric test the patient developed headache, a positive Kernig and neck rigidity: lumbar puncture obtained a C.S.F. containing several thousand cells per c.mm. Though the condition appeared to be of great gravity he made a rapid and uninterrupted spontaneous recovery, and within a few days the storm was past.

Shortly afterwards Mr. Stirk Adams explored his left mastoid which had been shown to be sclerotic by X-ray. The bone was eburnated and the lining membrane in the mastoid antrum and air cells was thickened. No exudate was present, and there was no cholesteatoma. Bone was removed to allow examination of the middle and posterior fossa and dura. No extradural abscess was present. He made an uninterrupted recovery from the operation.

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The speaker said it was clear that the meningeal reaction (meningitis aseptique) was produced by the use of the caloric tests in an ear which was the site of a chronic low-grade infection with an intact drumhead.

The PRESIDENT remarked on the diminishing number of complications such as brain abscess, meningitis or labyrinthitis. Where they saw ten cases in the old days they did not see more than one to-day. It was therefore more and more important that they should have clear-cut views as to how these cases should be treated when they did appear.

Concerning brain abscess his own viewpoint was rather altered from the time when, in consultation with a neurosurgeon, he was asked to say whether a brain abscess in the temporo-sphenoidal lobe was of aural origin. He gave the opinion that it was and intimated that an operation on the ear was desirable. That, however, was not first proceeded with, for the neurosurgeon decided to open the abscess through an osteoplastic flap in the temporal region. Later on he was asked to carry out the radical mastoid operation, with the result that the patient was still alive and perfectly cured.

That episode rather made him wonder whether they should always consult a neurosurgeon in these cases or not. He came to the conclusion that it was not always necessary, especially in those cases where the diseased track leading down to a rather superficial abscess was found in the first instance, and at the mastoid operation. It was his practice to drain brain abscesses with rubber tubes ; he had never resorted to aspiration. His cases had been few, but he had achieved successful results by putting in a tube and treating it carefully so as to disturb the surrounding parts as little as possible. On the other hand in cases where one was not quite certain of the track a neurosurgeon should be consulted.

Mr. E. D. D. DAVIS said, in reply to Mr. Watson-Williams, that the results obtained by the otologist were very much better than by the neurosurgeon. The otologist should be a general surgeon with a sound knowledge of neurosurgery and otology.

Neumann attributed his good results—47 per cent.—to the fact that he made an early diagnosis before the presence of naming aphasia. The acute or subacute abscess might be fatal before the abscess was encapsuled. He supported Major Fowler when he said that the large majority of cases of otitic meningitis arose in the posterior fossa of the skull.

Mr. F. W. WATKYN-THOMAS said that he was glad to have so much support for his view about petrositis. He was convinced that the great majority of cases would subside with adequate mastoid operation. It was exceptional to have to carry out an actual operation on the petrous bone. He, too, inclined to the " heresy " that cholesteatoma was not itself infective, but by the time the otologist saw the cholesteatoma it probably was infected. Facial weakness after labyrinthectomy was not common. If a patient had a facial palsy *before* the labyrinth was operated on it complicated the condition considerably. Such palsies were not always recoverable, but from the results of ordinary labyrinth drainage (he was not referring to translabyrinthine drainage through the meatus) he could never remember getting a permanent facial palsy. He had once or twice found a transient weakness.

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Major Fowler's suggestion of making a little hole in the mastoid to give relief of pain in fulminating cases was very intriguing. He had been tempted to do it but up to now he had never summoned up the courage.

An interesting point had been raised by Mr. Donald Watson, Colonel Canfield and Mr. Stirk Adams about the possibility of flaring up trouble by a cold caloric test. He supposed it would be possible that just that amount of irritation, the setting up, so to speak, of a wave, however trivial, in the endolymph, might cause enough labyrinthine disturbance to trickle a dose of poison through one of the preformed tracks.

Mr. TERENCE CAWTHORNE did not think it was necessary to do the cold caloric test on the affected side though he did not share other speakers' fears of the consequences. As Hallpike, Fitzgerald and himself had shown, it was possible to do the hot and cold caloric on the sound side and to obtain, together with other indications, the required results.

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