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OTITIC MENINGITIS

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WHEN Charles West left St. Bartholomew's twenty years ago, he suggested to some of us that the two outstanding problems of otology awaiting solution were the mechanism of the auditory focus, and the treatment of otitic meningitis.

Auditory focus, I fear, is still a vague blur somewhere just over the edge of the otological subconscious, but in the treatment of meningitis a great—a really great—advance has been made. How great that advance has been we may not fully realize. Statistics are convincing, but still more convincing is the simple fact that twenty years ago the most retiring surgeon felt it his duty to mention the survival of a single patient in whom otitic meningitis had progressed to the extent of organisms in the cerebrospinal fluid. Some six hundred years ago a public-spirited philosopher, one Ockham, forged a weapon which should be in the hand of every doctor. He called it his razor. It was a formula, a simple formula, and it ran thus: "Entities must not be multiplied."

So I shall say that by meningitis, I mean the reaction of the pi-arachnoid system to an advancing infection from the middle ear and its adnexa. "Meningism", "serous", "bacterial" or "suppurative" meningitis, "encysted" meningitis—are all stages of a single process as modified by virulence or resistance.

Meningitis is not particularly dangerous because organisms are present in the cerebrospinal fluid: it is the other way

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round ; the organisms are present in the fluid because the meningitis is particularly dangerous ; because the resistance has been overcome, and the infection has reached its most dangerous stage. For this reason I avoid the statistical sub-classifications. They only make a difficult problem more difficult.

If we accept, as we are bound to accept, this view of meningitis, it is clear that the principles of treatment are the principles applicable to every advancing suppuration ; elimination of the focus, drainage, and the appropriate counter-medication, serum treatment or chemotherapy as the case may be. And here may I remind you of the fact, so obvious that in the past it has often been ignored that the patient who dies of meningitis is killed by septicæmia, not by increased intracranial pressure. The principles are simple enough—they usually are ; how exactly to apply them has been the difficulty —it usually is !

A short time ago a colleague of ours, realizing like Mr. Michael Finsbury that " there's nothing like a little judicious levity " in a delightful essay, advised us to study the great detectives of fiction. Such a study should make us sympathize deeply with their unhappy biographers, who were always being exhorted to apply " my methods " or to exercise their little grey cells without any hint as to how they were to set about it. We have been too often in their position—especially in the treatment of meningitis.

In one striking instance the application of the principles of elimination and drainage was rewarded by brilliant success. Invasion of the pi-arachnoid space by suppuration extending through the labyrinth was dreaded as the most formidable attack of all. The infection of the cisterna pontis by way of the internal auditory meatus resulted in a spread of infection so swift and so massive that it was almost invariably fatal. The fear of it was one of the greatest hindrances to any surgical procedure on the labyrinth.

Nevertheless, the condition was most suitable for resolute surgery. The infecting focus, the labyrinth, could be thoroughly and easily eliminated ; adequate drainage at the site of infection was provided by a ready-made non-collapsible drainage tube, the internal auditory meatus. In 1908 West and Sydney Scott did the first translabyrinthine drainage and saved the patient ; the surgical principles were vindicated.

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Unfortunately the method is rigorously limited ; it is invaluable for labyrinthine infections, but only for labyrinthine infections, and with the advance of mastoid surgery such infections have become steadily less frequent.

Our difficulties in applying the principles have been anatomical and physiological, especially the irreproachable neutrality of the choroid plexus, which obstinately refused admission to all therapeutic remedies offered to it.

Between the anatomical and physiological problems we can draw no rigid boundary, for structure is only the machinery of function. Still, for convenience we may say that the major difficulties of elimination of the focus are anatomical : the major difficulties of meningeal drainage, physiological.

Elimination of the Focus

Apart from extirpation of an infected labyrinth, drainage of a leaking brain abscess, or clearance of an infected venous thrombus, the orthodox elimination of focus was a mastoid operation, done as thoroughly as possible, with exposure of the dura of the middle and posterior fossae.

The brain abscess that leaks is usually one that has already doomed the patient ; an infected sinus thrombus is usually detected before meningitis starts, and is operated on for its own sake.

As Eagleton, Kopetzky, and Marx have shown, in the majority of cases the track of infection is through the petrous bone, and reaches the perivascular sheath by a retrograde venous thrombosis, and it is to Eagleton that we owe the demonstration of the method of attack. Extradural, intradural (venous sinuses) and pial veins all communicate. These last, the veins of the cortex and cerebral substance, are endothelial tubes, with no adventitia except the arachnoid mesh, save for a short collar of fibrous tissue where they enter the sinuses. At what Eagleton calls " the points of venous vulnerability " they are in close contact with bone which may become infected. If once organisms enter these endothelial tubes they can readily pass through the walls and into the arachnoid mesh, into the perivascular space, that double cuff of pi-arachnoid which surrounds and accompanies the vessels and is the channel of the cerebrospinal fluid. The most dangerous of these " points of vulnerability " are (*a*) a small venous plexus on the upper surface of the petrous in the angle

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between the superior and lateral canals. (Here we should remember that Frenckner has described a series of cases in which he followed a line of infected cells through the arch of the superior canal up to the apex ; also, that all of us have at some time or another, found considerable difficulty in elevating the dura from the bone in this region.) (*b*) The vein of the subarcuate fossa. (*c*) The petrosal vein in the internal auditory meatus. (*d*) An inconstant plexus perforating the dura and entering the cellular bone near the apex (Pietrantoni), and (*e*) at the point of communication of the cortical veins and the sinus, especially around the upper edge of the petrous.

To these " vulnerable points " we must add another. Steurer states that in the infant there is direct vascular communication between the dura and the submucosa of the middle ear. Normally these vessels undergo involution during the process of pneumatization and are reduced to strands of connective tissue, but if pneumatization is arrested they persist. According to Witmaack there is one at the tegmen and another below the angle of the petrous pyramid near the dura of the sinus. This makes a diagonal link from the antrum up to the sinus angle. It may be from 2-7 mm. long and as thick as a " knitting needle ". It is most usually found when the mastoid is dense and the cells are few.

I have encountered this vessel in a well-pneumatized mastoid, and had some difficulty with it.

This leads us to a brief consideration of the petrous itself.

The petrous is laid down in cartilage, which in embryonic life is largely replaced by red marrow. When pneumatization advances the red marrow is converted to yellow, but faced by infection it can revert again to red. Mass figures show complete pneumatization in 35 per cent. of adult petrous bones ; marrow is present in more than 90 per cent. It can be stated, generally, that where pneumatization is complete, the freedom of communication between blood-vessels of the dura and bone is largely abolished, so that danger of meningitis is diminished, although infection can travel more easily through the bone. When pneumatization does not occur the dural communications are more persistent, but the red marrow (which, as has been said, reforms in the presence of infection, even after it has advanced to the yellow stage) is capable of an admirable resistance. In neither case is necrosis and sequestration of the petrous surface common ; in pneumatized bone there is

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excellent blood supply by vessels of the subendosteal layers ; in the non-pneumatized bone by vascular loops in the free spaces of the marrow. These facts account for the frequency with which petrous infections clear up with the simplest adequate drainage, and the comparative rarity of meningitis as a complication of aural suppuration. For we must remember that it is, fortunately, not a common complication.

Eagleton, in 1929, advocated an operation for " unlocking " the petrous. Almour and Kopetzsky devised an operation for draining the petrous apex. Ramadier and Frenckner suggested other ones. These attacks on the apex are more adapted for the prevention of meningitis than for its cure, but they have all added to our knowledge of the surgical possibilities of the petrous bone, and have helped greatly to simplify the first of our tasks—the elimination of the focus.

In the establishment of drainage we failed for a long time because of our faulty tactics. We sought an anatomical approach, and ignored the physiological aspect. We tried to drain accumulations, rather than to re-establish the normal circulation of the cerebrospinal fluid.

In one instance, as I have said, we had a brilliant success ; that was in translabyrinthine drainage for meningitis of labyrinthine origin. But here many factors were in our favour. The very fury of the meningeal invasion drove us to early operation ; to operate before the fluid was heavy with protein. Looking back on our results, we may ask whether the real reason for our success was not the perfect elimination of focus provided by the operation ; not only elimination of focus in the bone, but also prompt removal of a massive dose of highly infected fluid in the cisterna pontis. We must remember that the arachnoid mesh in the internal meatus is extraordinarily fine ; so fine, that some localization of infection is likely to happen here : Politzer describes a " chronic abscess " of the internal meatus. Another valuable aid is the free flow of fluid into the labyrinth, which must help to wash out any organisms lurking in the bony recesses. It was often noticed that when there was no free rush of fluid on breaking through into the internal meatus, the operation usually failed.

George Jenkins worked out a method of intrathecal lavage which, in his hands, sometimes gave excellent results. Unfortunately, the difficulties of an elaborate technique prevented its general adoption.

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Some patients survived after mastoidectomy and continuous lumbar drainage.

Drainage of the cisterna magna was often tried. As far as I know, no patient survived this.

Incidentally, it is not conclusive proof that because a patient survives an operation, that operation has necessarily saved him.

Incisions of the membranes and the insertion of drainage tubes into the lateral cistern were useless.

In the belief that the great difficulty of drainage was due to loculation by adhesions, some surgeons tried to break them down by intrathecal injections of air and of acetylene. A few good results were claimed (as I have suggested, the power of survival of some patients is remarkable), but after a short time nothing more was heard of the method; which speaks for itself.

Our tactics were all wrong. Dixon and Halliburton had shown us that the cerebrospinal fluid is formed by the choroid plexus. Weed, and his co-workers, proved that it is absorbed into the blood-stream by the arachnoid villi, and if the arachnoid villi are blocked absorption is prevented. We know, too, that the fluid not only passes from the ventricles into the main pi-arachnoid spaces, it also percolates the whole brain substance by way of the perivascular sheaths.

As meningitis is an inflammatory reaction, the cerebrospinal fluid is quickly loaded with cells and coagulable protein, which block the exits of the arachnoid villi, and also obstruct the perivascular sheaths of the deep vessels. The physiologists know the answer. Massive intravenous injections of hypotonic saline increase the flow of cerebrospinal fluid. Kubie applied this clinically and showed that it was possible to wash out the meninges from within, removing the surplus by repeated lumbar taps. This method has the further advantage that it dilutes the protein-laden fluid; that the pi-arachnoid space is kept open by the free flow, and the risk of loculation is diminished. Further work along these lines should solve the difficulty of drainage.

Counter-medication

Here the difficulty was not of our making. The choroid plexus put up a barrier which was, for a long time, insuperable. The only chemical antiseptic which it would sanction was

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hexamine, and that was quite useless. Hexamine only acts as an antiseptic when it breaks down, and it does not break down in the cerebrospinal fluid.

Sera and antitoxins were rejected unhesitatingly by the plexus. Intrathecal injection of antitoxin succeeded often in tetanus and meningococcal meningitis, but the same method failed pretty consistently in suppurative meningitis.

Intrathecal or intracarotid injections of substances such as mercurochrome and bile-salts may possibly have destroyed the organisms; unfortunately they usually also destroyed the patient.

A more promising line of attack was again suggested by the work of the physiologists. As hypotonic saline increases the flow, so hypertonic saline intravenously, or even massive depletion by magnesium sulphate enemas, will draw off cerebrospinal fluid (probably to the extent of reversing the current in the perivascular spaces) and shrink the brain. As we know, the Monro-Kellie doctrine lays down that the contents of the cranial box must always remain the same in bulk, so the loss of cerebrospinal fluid must be compensated by an increased blood volume. Accordingly attempts were made by salt-depletion and intravenous transfusion of immune blood, to overwhelm the resistance of the choroid in a " thirsty " brain. This work was still in progress when the discovery of the sulphonamides gave us a powerful antiseptic which could traverse the barriers, and so solved the outstanding difficulty of chemotherapy.

The Management of a Case of Otitic Meningitis

The fact that a patient has otitis and meningitis is not proof that the meningitis is due to the otitis.

There is one group of cases, usually in children, when meningeal symptoms and acute otitis media are practically simultaneous. These fulminant cases are not, in my opinion, cases of otitic meningitis. They are blood-borne invasions which strike both the meningeal spaces and the middle ear. It is probable that the organisms to a large extent reach the meninges by way of the middle-ear vessels, through the preformed paths which exist in the petrous temporal, but the meningitis is not strictly speaking, otogenous or otitic.

If we accept this view in such cases, we will refrain from surgery in the early stage, because there is no focus in the

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temporal bone to eliminate and pin our hopes on chemotherapy. If the drum bulges, we shall incise it, and if, later on, we find a definite mastoid suppuration, we shall regard it as a fixation abscess and drain it as such. But the rules of treatment laid down for otitic meningitis do not apply, because these cases are not due to middle-ear infection.

Again, it is not impossible for a patient with an otitis media to have, independently or otherwise, meningococcal or tuberculous meningitis. The cerebrospinal fluid will give us the evidence.

Further, a patient with otitis media may have, often does have, accessory sinus suppuration, and there is always the possibility that his meningitis may be due to his sinus suppuration, not to his ear.

The diagnosis of established meningitis is generally easy. The signs are those of acute and increasing intracranial pressure with severe toxæmia.

Usually the onset is sudden, after a day or two of vague and ominous discomfort. It is our ambition to detect the condition at its earliest, and not to await the classical syndrome —which, as is often the case with classical syndromes, is the prodrome of approaching death.

Headache and pyrexia are the first and the most constant warnings. We can make a clinical rule that no patient with an uncomplicated mastoid suppuration has any right to have a headache. He may have pain in the ear or radiating from it, but pain not so localized should always raise the suspicion of intracranial irritation. Pyrexia is nearly always an early sign. Slowing of the pulse is uncommon. Some stiffness of the neck is usual and early, but it is not always found even in advanced cases. Probably the severity varies with the extent of invasion of the posterior fossa.

Some congestion of the discs is common and early. My personal experience is that it is present in a considerable majority of cases. Vertigo and nystagmus are not unusual, even in cases where there is no labyrinth lesion. I have never been able to fit them into any set pattern to my own satisfaction, or to correlate them definitely with other findings, but Eagleton, whose opinion no one can afford to neglect, definitely states that:

(1) Absence of response to the cold caloric test of the opposite ear in the upright position with positive response in the horizontal position, is produced by increased pressure.

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(2) Absence of response to the cold caloric test with good hearing is due to exudate in the pi-arachnoid prolongation around the VIIIth nerve.

(3) Irregular nystagmus with good hearing indicates posterior fossa effusion.

(4) Irregular attacks of vertical nystagmus indicate effusion around the bulbar region.

Focal signs, when present, are usually caused by a loculated collection. These collections enlarge fairly rapidly, and as they enlarge they leak, for the barrier adhesions are weak and imperfect. The commonest site for them is said to be either above the tegmen, or in the posterior fossa, internal to the bend of the lateral sinus. Above the tegmen they may cause quadrantic hemianopsia by pressure on the optic radiation. I have seen such a collection in the region of Broca's convolution give an excellent imitation, from the neurological aspect, of an increasing middle meningeal hæmorrhage.

The determining fact in our diagnosis is the result of the lumbar puncture, and lumbar puncture should be done in every case where there is the least suspicion of meningeal invasion.

It should be done carefully, for there can be no doubt that sudden death has followed excessive withdrawal of fluid, although we do not really know how, exactly, death in these cases was caused. Also, it is probable, that excessive withdrawal of the fluid, if bacteriæmia is present, can actually induce a meningeal invasion. The slow withdrawal of 2-3 c.c. can do no harm, and that amount is quite enough for the pathologist.

We want information, above all, about the cell content; then the bacteriology, if any. The albumin and sugar content are more interesting than important. The estimation of chlorides is, I think, more valuable for prognosis than for diagnosis. The lower the chloride content, the worse are the patient's chances. We want to catch and treat the condition before the chlorides fall.

In special cases diagnosis is made difficult by causal or concomitant conditions.

(1) Septic thrombophlebitis of the lateral sinus seldom causes meningitis unless it is left untreated for far longer than is usual now. Even in such untreated cases, cerebellar abscess is the more usual sequel. But sinus thrombosis and meningitis do sometimes occur together, and at quite an early stage.

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I have seen three such cases in the last two years. In all of them the focus of infection was in the petrous. In two of them the thrombus was at the junction of the superior petrosal and lateral sinuses. Probably in all three cases thrombosis and meningitis were both due to the petrous infection, not to each other. Cases have been reported in which sinus thrombosis accompanied suppurative labyrinthitis (I have seen two such cases), and in such cases there is a great risk that the sinus condition may be overshadowed by the more urgent and dramatic signs of meningeal invasion.

It is a possibility which we must always remember, for however efficiently we deal with the petrous or the labyrinth, however vigorously we administer sulphonamides, all will be in vain if we overlook a spreading infection in the lateral sinus.

(2) Brain abscess: It is reasonable to assume that a brain abscess starts like any other abscess, with exudation of fluid and infiltration of leucocytes. It starts, in fact, as an encephalitis and it must be accompanied by meningeal reaction. Borries has shown that a clearing fluid and increasing symptoms are diagnostic of brain abscess. If, at the first suspicion of encephalitis we attacked the focus and treated the condition as we should treat a meningitis, we would stand a good chance of arresting the further development of an abscess.

The leaking brain abscess is quite different. It is uncommon, but it happens. Dan McKenzie and Broughton Barnes described cases in which recurrent attacks of meningitis were caused by cerebellar abscesses. A cerebral abscess which leaks into the ventricle is nearly always fatal, but Jenkins saved one by intrathecal lavage. In these cases, the abscess usually develops silently, and is only found when the onset of meningitis demands operation.

(3) Labyrinthitis and meningitis: If a patient with labyrinthine suppuration, total loss of hearing on the affected side and nystagmus of destruction, gets pyrexia and a headache, he has got meningitis. The patient with latent labyrinthitis (so-called) is more difficult. In plain English "latent labyrinthitis" means that the patient has had an attack of suppurative labyrinthitis, which has been diagnosed as a "bilious attack" or "gastric influenza", and has recovered with a dead (and infected) labyrinth. If months, or years, later the organisms invade the meninges by the internal

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auditory meatus, there will be no warning labyrinth storm. The first signs will be the signs of meningitis.

In this way, too, a patient who has suffered labyrinth destruction from a fractured base may get a meningitis without warning from an acute otitis media and invasion through the unclosed fracture line.

In every case of meningitis we should investigate the labyrinth by the caloric test and hearing tests (making sure of these tests that the opposite ear is efficiently masked by a noise box, or other method).

(4) Petrositis: The difficulty here is that we may pay too much attention to the localized Vth nerve pain around and behind the eye, and too little attention to the fact that the patient is getting more generalized headache. Pyrexia and extension of headache are our warning signs. When we find them we should certainly do a lumbar puncture and probably we should operate.

Treatment

Our diagnosis is made, and as far as possible that diagnosis has included the source of infection.

It is frequently said that "sulphonamides have revolutionized the treatment of otitic meningitis". This is an unhappy example of that chronic looseness of statement for which our profession is so justifiably notorious. Sulphonamides have done nothing of the kind. They have enormously improved our results, but the principles of treatment are the same. What has happened is that we have another, and most valuable, weapon to our hand.

The sulphonamides are most efficient in the tissue fluids and the blood; they are at a disadvantage in bony spaces, and they are almost powerless against a collection of pus. Our first duty is still to eliminate the focus. As Jansen said many years ago: "The first sign of meningitis is the last call to operate."

In meningitis of labyrinth origin I feel that the right course is still to open and drain the labyrinth, and to complete the operation by breaking through into the internal auditory meatus. With further experience of chemotherapy we may be able to modify this. We know that sulphonamides freely enter the cerebrospinal fluid, and that there is free communication between the cerebrospinal fluid and the perilymph. In fact, the labyrinth is a bony space in which, against the

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usual rule, sulphonamides should be able to act well. For this reason, we may hope that in the future many cases of labyrinthitis will be cured by chemotherapy, and that the need for opening the labyrinth will be exceptional. But when labyrinthitis has advanced to meningeal invasion, I do not think we can take any risks until we know far more. There is a definite focus and that focus must be eliminated. The final step—opening the meatus—is simple and rapid, and we should take it. The stakes are too high for us to omit any precaution.

The actual method which I have usually followed is that of West and Scott—opening the vestibule and the external canal, and then entering the internal meatus through the fundus of the vestibule. If this is done with a small, very sharp, gouge, held firmly without tilting, and if we are careful to avoid any pressure against the upper border of the oval window, it is usually possible to avoid the facial nerve. A few strands of silkworm gut can be put into the meatus for drainage, but if the flow of cerebrospinal fluid is encouraged by intravenous hypotonic saline, I do not think it is necessary.

In the majority of cases the only evident focus is the infected middle ear and mastoid. Here our right course is to explore the petrous by a route which will give us a good exposure of the lateral sinus and the dura of the posterior and middle fossae. The methods of drainage suggested by Almour and Kopetzky, Frenckner and Ramadier, apply to collections localized in the petrous, and are unsuitable for the extending infection. If we have any clinical evidence as to the state of the petrous, so much the better. If an expert radiologist is available his help may be most valuable, but once meningitis has appeared we are not justified in delaying operation to get a skiagram.

Unless the labyrinth is involved, a radical mastoid is not usually needed. I start with a wide epitympano-mastoid operation and free removal of the outer attic wall, and clear the cells as far as possible before exposing the dura. The lateral sinus is exposed up to and beyond the entrance of the superior petrosal, then the tegmen is removed and the squamous temporal freely resected. Next the bone of Trautman's triangle and the posterior margin of the petrous are attacked. My greatest difficulty is always in stripping the dura over the superior canal. I have found the curved-on-flat end of a Hill's septal elevator the most comfortable weapon. Another

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spot which is sometimes difficult is the outer extremity of the petrous angle. A suboccipital puncture sometimes helps to make the brain more manageable. I have never done a ventricle puncture as a help to the operation, but I see no reason why one should not do so in a suitable case. There can be no risk of infecting the brain tissue thereby, as the brain is already percolated by the infected cerebrospinal fluid.

The dura is stripped up to the apex and posteriorly to the lip of the internal meatus. It can then be thoroughly examined —the dura of the cerebellum on the posterior petrous aspect should be inspected too. Unless there is an encysted collection of fluid or evidence of an abscess, the dura should not be incised. It is quite useless to do it for drainage, as the brain sits down firmly in the hole and blocks it. The upper and posterior surfaces of the petrous are carefully examined, and any softened or inflamed bone is cut away. Any abscess in the bone can be opened with a curved curette. If pus is found a rubber dam drain should be put in and the wound kept widely open with rubber packing.

During the operation pieces of bone should be put into broth for the pathologist. As far as possible these pieces should be blood free, because blood will hinder the growth of pneumococci. A swab from the nasopharynx will often reveal the causal organism.

Also, during the operation, an assistant should give an intravenous injection of half-strength normal saline, about 500 c.c. for an adult ; a lumbar or suboccipital tap should be done and as much cerebrospinal fluid as possible drawn off.

Chemotherapy should be started as soon as the vomiting from the anæsthetic has passed. If the organism is known the appropriate sulphonamide should be given. If it is not known, pending the pathologist's report, it is probably well to give sulphapyridine, which has wider activities than sulphanilamide. It should, of course, always be given for pneumococcal infections, for streptococcal infections sulphanilamide seems better.

To get the best results from sulphonamide treatment we should have the help of the physician and the pathologist. We must know what the organism is ; what is the concentration of the drug in the blood serum and in the cerebrospinal fluid ; and we must watch the blood-count and the urine. Too often, as far as chemotherapy goes, we fight with one hand tied behind

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our backs because, before he comes to operation, the patient has endured a long, futile and dangerous medication with too small doses, perhaps of the wrong drug, spread over too long a period. His symptoms have been masked, his white cells already lowered, and he has already had so much of the drug that we cannot risk the heavy loading dose necessary for saturation.

After-treatment consists of full sulphonamide dosage, lumbar puncture as long as headache persists, and hypotonic saline intravenously until the cerebrospinal fluid has cleared.

Complications in our field are unusual, but there is one, and a new one, which occasionally occurs. That is, recurrence of meningitis after the patient is apparently cured. I have seen one case ; in this the attack was more spinal then cerebral, and was probably due to the rupture of an encysted collection in the spinal theca. The patient recovered on sulphapyridine. The origin lesion was a pneumococcal petrous, plus superior petrosal sinus invasion.

To sum up : The cardinal principles of the treatment of otitic meningitis are surgical intervention ; the establishment of drainage and the application of chemotherapy. By the combination of these three, our results have been enormously improved, and we can hope for still greater improvement.

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