SUPPURATIVE LABYRINTHITIS: A
CRITICAL REVIEW OF ITS DIAGNOSIS
AND TREATMENT.*

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ALTHOUGH the subject of orientation and equilibration, and the intimate connection of the vestibular apparatus with this function, have occupied the attention of otologists for more than a century; and although much of the most important experimental work connected with it was done thirty or forty years ago; the surgery of the internal ear lesions resulting from inflammatory invasion has been developed well within the past two decades. I remember in the course of a summer in Berlin, about fifteen years ago, seeing the operation for suppurative labyrinthitis performed by Dr. Jansen on two chronic cases and one acute case. At that time the test for loss of hearing was very inadequate, and no one had suggested separate examination of the vestibular apparatuses. I recall that the operations impressed me with the importance of more careful anatomical study of the petrous bone with reference to operative work, and that the technic of the operation interested me much more seriously than the diagnosis or the indications for operation.

It was not until Bárány, about ten years ago, presented the noise apparatus, which enables one quickly and positively to demonstrate complete loss of hearing; and the caloric reaction, which enables one separately to test the presence or absence of the static function in each ear, that surgery of the labyrinth was placed upon its present sound footing. Compared with all other means for determining the necessity of surgical interference in the internal ear, these two tests stand out in paramount importance. Indeed, in my own opinion, they are the only necessary functional tests. While the other functional tests, like whirling, and the galvanic current, are interesting and useful in clarifying our minds regarding the static function, and therefore valuable in demonstrating certain reactions, in the study of the subject, when we get down to an actual clinical case on which the question of surgical interference is to be settled, they are, to my mind, useless in comparison with the ones first mentioned. This is due to the fact that the *whirling test* can never cause a reaction in one vestibular apparatus at a time, provided that both are functioning; and despite the fact that an imbalance of the two apparatuses may be demonstrated, by the after-nystagmus resulting on turning, first in one direction and then in the other, it is well known that compensation takes place, sometimes in a longer, sometimes in a shorter, period after complete destruction of one side, and therefore a negative result will never be convincing. I

believe it is absolutely useless in acute cases when a spontaneous nystagmus is present; and that it may be so in the chronic cases, owing to partial or complete compensation by the sound side. It seems useless in the acute cases because, with an imbalance sufficient to have caused the spontaneous nystagmus, the added confusion of the whirling brings out only phenomena which it is almost impossible to observe accurately. From another point of view, even if the information gained should be accurate, it seems hardly wise to jeopardize the chances of recovery of so ill a patient by subjecting him to this additional insult when a much more positive test (the caloric) may be used with comparatively little discomfort. The whirling chair, then, in my opinion, should be relegated to the armamentarium of those who are teaching or studying the phenomena of static equilibrium, and should be discarded as a positive diagnostic factor in labyrinthine surgery. No one would think of not verifying its positive indications by the more accurate caloric test, and no one should accept its negative indications as being of any particular value.

The *galvanic reaction*, while it enables one to test each labyrinth separately, is confusing from the fact that it may excite reactions by stimulating the vestibular nerve-trunk, even when the end organ is not functioning. It is, therefore, in my opinion, useless except for the teacher or student, and is of no positive aid in determining the necessity for surgical interference, as compared with the caloric reaction.

These two tests stand in relation to the surgical diagnosis of labyrinthine involvement in about the same importance that transillumination occupies in the diagnosis of accessory sinusitis. Transillumination may be of positive value or it may not; in these days of radiography, no one would think of accepting its most positive indication without the confirmation of a radiogram.

The "*fistula test*" may be positive in the absence of a positive caloric reaction, or it may be negative in the presence of a positive caloric reaction. This, of course, will depend on whether there is a fistula present or not, and whether there is a vestibular function present, or not; but one will seldom find the caloric test negative, although it may be delayed or weak; the fistula test is positive, while the fistula test, even in the presence of a fistula, has no diagnostic value unless it is positive.

There is another important factor in connection with this test—and since I am on the subject I may as well finish it now, although it may be somewhat out of place. The fistula symptom is present only in cases in which a suppurative process has extended to the membranous labyrinth, either by erosion of bone, by a necrotic process, or by extension through a dehiscence in the bony capsule. We seek for it in cases in which the symptoms point to a lesion which has

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not invaded the endolymph. This is in a type of cases (the so-called "circumscribed") which often suddenly become active, acute, and develop into the most serious complete endolabyrinthine involvement. When we find a fistula during the course of an operation, every surgical instinct urges us not to probe or in any way exert the slightest pressure or traumatism on the fistula, and we so instruct our students. (This applies to cases where a functioning labyrinth has been demonstrated). Under these circumstances, who can say that the fistula test, once applied, may not be the cause of converting a perilabyrinthitis into an "acute diffuse suppurative one?" The pneumatic pressure necessary to bring about the reaction cannot be calculated to fit all cases, and certainly the fistula test, applied as we usually see it, is quite as dangerous to the welfare of the patient as the probing or curetting during an operation,—which we all abjure with such holy horror.

If the caloric test is positive, then, or can be demonstrated in even its weakest degree, one can well afford, in the interest of the patient, to wait and discover by inspection, at the time of a radical operation, whether or not a fistula is present. If the caloric test is negative,—hearing also being entirely ablated,—there may be a slight justification in applying a fistula test to determine whether or not there is any remaining vestibular reaction. With any hearing present, however, there is sufficient contraindication to operative interference on the labyrinth to make the fistula test unnecessary, since at the time of the radical operation this can be determined by actual inspection. Here again, then, a test which is interesting and often very illuminating can only be practiced with certainly always a slight, and in many cases a grave, danger to the patient. If this test is used at all, it should be undertaken with great care, using the slightest pressure at first, and only increasing it, on negative results, up to a point which the investigator thinks (it must be guessed at) would not be sufficient to break through the possibly weakened membranous labyrinth wall. Having once demonstrated the presence of a fistula by this method, I consider it very bad practice to go on demonstrating it repeatedly, as we often see done for the illumination of one surgeon after another, in both hospital and private practice. The life of the patient with such a lesion is so seriously menaced, by possible extension into fatal areas by the experiment, that one should—if he attempts it at all—record the result once for all time.

This leads me into another digression. Much of the experimentation or demonstration of various interesting phenomena connected with an inflammatory involvement of the labyrinth might, in the interest of the patient, be well taken for granted. For example: as I have just said, if one will make use of a dangerous fistula test, he might at least be considerate enough,

if positive, not to repeat it. If a patient suffering with an acute labyrinthitis has manifested symptoms which show that both the hearing and static sense are ablated, one may as well take it for granted that if made to stand he will fall toward the side of the lesion; that if whirled, he will probably show certain well-known reactions; that if moved about, he will exhibit nausea and vomiting. Now, in most of the cases reported we see the results of these various tests recorded. We all know that these phenomena would have occurred, and the man who has demonstrated them again has in many instances done so to the detriment of his patient; for we know that perfect quiet is very essential to the prevention of extension from an endolabyrinthine lesion to the meninges, and may be so for the prevention of an extension from a peri- or para-labyrinthitis into the endolymph. To say in one breath that a patient should avoid undue excitement and any action which might jar the delicate barriers which nature is attempting to erect to prevent extension to the meninges; and in the next breath to take such a patient out of bed to demonstrate that he will fall in a certain direction on attempting to stand or walk, or that he will show certain reactions to the insult of whirling him in a revolving chair; or even to stirring him about in bed, or having him sit up in order to demonstrate a difference in the direction of nystagmic movements; or to provoke nausea and vomiting; is, to my mind, an exhibition of bad judgment, or bad practice in the management of the case.

All these questions of whether the patient will fall in a given direction or react to certain experiments, have been settled; we know they will do it. If that is unknown ground to anyone, he should either accept it as a fact established by others, or learn it for himself on chronic cases, operated cases, and animals, rather than by demonstrating it by experiments on his acute case in which the demonstration may be the last straw which "breaks his camel's back."

This leads me to another somewhat critical position regarding the clinical diagnosis of labyrinthitis. We are deeply indebted to the Vienna school for their painstaking efforts to record the last detail of their cases, for their enthusiastic rivalry to present some new phase of an intricate subject, or some added factor in the differential diagnosis of the various types of labyrinthine inflammation. Their efforts to subdivide their cases and reduce each one to its lowest terms have been due in part, I think, to a desire to present a fixed or standardized formula, with definite symptoms for each, and a definite treatment for each. This might at first thought seem to simplify the matter for students; but I believe that instead of accomplishing this object they have unnecessarily complicated the question, and have led those who approached their first cases of labyrinthitis into a

feeling of bewilderment lest on the one hand, they should misinterpret the clinical symptoms and be led into performing an unwarranted operation, or on the other hand be influenced into too long deferring a necessary one. The cases I am speaking of are all those which have resulted from a suppuration in the middle ear.

The question which confronts one in such a case is: Is this acute or chronic? We know that no acute case of labyrinthitis can be present without manifest symptoms. We must remember that symptoms of an acute labyrinthitis may be present in either an acute or a chronic suppurative otitis. The first evidences of an acute inflammatory involvement of the labyrinth are: impairment or loss of hearing, and tinnitus aurium from involvement of the cochlear apparatus; and loss of equilibrium, vertigo, nausea and vomiting, and nystagmus resulting from loss or impairment of the vestibular apparatus. Either one or both of these impairments or loss of function may be present in any acute case. If either function is present in any degree, it is safe to say that an acute suppurative endolabyrinthitis, at least of an operative character, is not present at that moment. If both functions are ablated, the case may or may not be one of acute suppurative endolabyrinthitis. If the loss of function has been very rapid (within a few hours) following a virulent acute suppurative otitis, or following the fistula test, or any operative interference in a case in which there has been a perilabyrinthine inflammation, the chances are greatly in favor of the lesion being an acute suppurative endolabyrinthitis. If such is the case, there will be no return of function, even though the case recovers without operation. On the other hand, if the loss of function came on slowly, with irritative symptoms showing first for a number of hours, or days, or weeks, there may be an eventual complete loss of both functions, followed by final recovery or partial recovery of one or both, provided the labyrinth is not operated.

It can be readily seen from these incontrovertible facts, that in any case of acute labyrinthitis we are brought face to face with the question of whether or not we shall operate upon a case which is in imminent danger of an intracranial involvement. The clinical symptoms from which we are able to elucidate the question may be identical, and if all cases were operated upon to relieve such symptoms there would undoubtedly be some which had they been let alone would have recovered with considerable function; on the other hand, some might be caught at the proper moment and drained with sufficient skill to prevent an intracranial involvement; on still another hand, there would undoubtedly be some cases in which the operative interference itself would precipitate the very cataclysm which the operator was endeavoring to avert. By this, I mean that the excavation of bone necessary to accomplish

the drainage—removed in the most ideal method imaginable—cannot help but be a menace to the delicate fabric which nature in every such case is attempting to erect between the meninges and the infected area. The thought of this great danger of traumatic production of the very mischief one is trying to avert, has been growing upon me for years, and although I have carefully avoided employing mallet and chisel,—the use of which I consider extremely bad technique on account of the violent concussion,—I still feel that in these acute cases even the most careful excavation by the use of rongeurs, curettes, and drills will, in a slight degree, present the same possible danger.

Under these circumstances, I am strongly of the opinion that any acute labyrinthitis showing no symptoms outside of the labyrinth stands a better chance of recovery unoperated until the acute symptoms have subsided. This may mean—depending upon the condition of the mastoid—a few days or a few months, or for all time. During this stage of acute symptoms, the endeavor should be to secure complete rest for the patient. If there were a question of whether I would operate on such a patient, or not operate, and in the meantime subject him to the insults to which he is usually subjected,—I should say by all means operate. I mean by this, that to take such a patient and stand him up, or ask him to endeavor to walk, to note which way he falls; or to have him sit up in bed and put him through hot and cold water irrigations; or to put him in a whirling chair and revolve him several times in each direction; or in any way to disturb him so as to provoke the violent vomiting which such patients are likely to have at that time, is quite as likely to add the traumatic shock which might break down the barrier to the meninges as a carefully wrought operation, but without presenting him at the same time with the advantages which the drainage afforded by the operation would provide.

Now, what symptoms are necessary to hold us in this waiting position?

Just two. First, loss of hearing. How is this demonstrated? With a suitable noise apparatus shutting out the sound side, the patient is unable to hear shouted words or a Galton whistle.

Second, he has a nystagmus of the vestibular type,—the slow movement of which is toward the diseased ear. (I shall speak more at length on this symptom a few moments later.) It does not matter whether the nystagmus is horizontal or rotatory in character,—you simply wish to know if a nystagmus characterized by a slow movement in one direction is followed by a rapid movement in the other direction. If the slow movement is toward the diseased side (it is always sure to be, if the hearing is ablated), it is the result of a great impairment or a complete loss of function on the diseased side. If

once or twice in your lifetime you see the slow movement away from the diseased side, with quick recovery toward it, you may be certain that the labyrinth on the affected side is functioning; that the nystagmus is the result of stimulation; and that the case has no question of an immediate operation in sight, because there can be no endo-labyrinthitis present. In such a case, there would almost certainly be some hearing present.

We all know that the acute symptoms in such cases rapidly subside. The function—hearing, static sense—may partly recover, entirely recover, or be completely lost. This applies to one or both senses. In the meantime, the condition which will be left in the labyrinth, depends upon what sort of resolution is taking place. Of course, the cases which recover function have not had a suppurative invasion of the endolymph. It is even possible that some of the cases which recover with loss of function may not have had a suppurative endo-labyrinthitis. This is drawing pretty fine, however, as all of them have had an inflammatory invasion of the labyrinthine walls sufficient to impair or completely destroy the function of the delicate end organs in the cochlear and vestibular apparatus. The distinction only indicates how imminent meningeal involvement had been. There is no way of knowing which is present at the time. The case will either have recovered with or without function, or will have rapidly passed into what, for all practical purposes, may be considered a chronic labyrinthitis.

What symptoms during this anxious period of waiting should lead one to operate?

First: a temperature of over 100°, accompanied by headache, photophobia, exaggerated reflexes, a positive Kernig, might be an indication that a beginning meningeal irritation was taking place, and no one should hesitate for a moment to operate, if a lumbar puncture verified the suspicion,—not only draining the labyrinth spaces but also uncovering and incising the dura as nearly as possible at the external auditory meatus, and possibly over the temporo-sphenoidal lobe as well. This operation would be done not alone for the labyrinthitis, but also for a beginning meningitis which had not been averted by zealous care and watchfulness.

Another situation might be present which would present some of these symptoms and which might justify an operation in the midst of an acute labyrinthitis, but which might not demand an invasion of the labyrinth. This situation is more likely to arise in an acute labyrinthitis coming rapidly on top of an acute otitis before or without any clinical evidence of involvement of the mastoid. Such a case might be running a high temperature owing to an acute follicular tonsillitis or an acute nasopharyngitis, or from an acute suppuration of

one or several nasal accessory sinuses,—the direct infection of the ear having occurred in the midst of their acute symptoms. Headache might be present from the high temperature; there might be some question of stiffness of the neck from glandular infection; there might be a questionable or very positive evidence of an acute mastoiditis; or a sigmoid sinusitis. In such a case, I believe one is warranted in operating the mastoid, with as little concussion as possible, in the presence of an acute labyrinthitis,—being guided at the time of operation as to whether or not the labyrinth should be drained, by a spinal puncture and examination of the fluid for symptoms of meningitis. This can be done, where laboratory facilities are easily available, without any delay, the report being returned to the operator long before the mastoid excavation has been completed.

In the absence of evidence in the cerebro-spinal fluid, of a beginning meningitis, I should, even under these circumstances, advise leaving the labyrinth alone except in rare instances. I am quite aware that this practice is considered too hazardous by many, but my own experience and that of others with whom I have seen cases, has been fortunate enough to warrant that conclusion, for the present at least.

Reverting for a moment to the subject of spontaneous nystagmus (one of the manifest symptoms of acute labyrinthitis) you will notice that I spoke always of the slow movement of the nystagmus first, and always of the slow movement as the one of diagnostic importance. You are aware that all nystagmus of a vestibular origin is characterized by a slow movement in one direction followed by a quick recovery movement in the opposite direction. You are also aware that the slow movement is the only one for which the vestibular sense is responsible. It is a very good thing also to keep constantly in mind that the slow vestibular movement is always away from the side which is exerting the most powerful influence. The vestibular impulses which keep us constantly informed of our position in space and influence our vision in fixing on moving objects, or on stationary objects whilst we are moving, might—for purposes of demonstration,—be compared to a billiard ball held in a stationary position by three rubber fingers on either side by an equal pressure from three different angles. You can well imagine that the least let-up in tension on any of the fingers would mean that the stronger pressure from its opposing finger would move the ball toward the weaker side, and exactly in the plane represented by a line drawn through the weakened finger and its protagonist. Now a labyrinthine stimulation might increase the impulses on that side to make a stronger "push" than that of the opposite normal side; the eyes would then rotate slowly toward the normal side, making a rapid recovery movement toward the stimulated side. This may

happen in the very earliest stages of labyrinthine involvement.

In an involvement of the labyrinth which in the least impairs or destroys the function of the vestibular apparatus, the impulses from that side are weakened or ablated entirely. Going back to our billiard ball the normal impulses from the sound side will push it toward the weakened one. The vestibular movement of the eyes (the slow movement of any vestibular nystagmus) then, is invariably away from the side sending out stronger impulses, so that it follows that a slow movement away from a diseased ear must mean increased function of an over-stimulated vestibular apparatus.

On the other hand, a slow movement of the eyes (of vestibular origin) toward a diseased ear, cannot be anything but the result of a weakened or absent function on that side. The normal impulse of the sound ear has produced this imbalance.* Suppose that we imagine another influence, quite outside of the little rubber fingers, which intermittently, violently, and rapidly jerks the ball back to its original position by a much more powerful impulse than they exert. This would typify a central influence on the eye, acting from a centre quite separate from the vestibular apparatus which in vestibular imbalance is entirely designed as a compensatory act. Why, under such circumstances, have we fallen into the habit of naming the nystagmus from this quick compensatory movement, instead of from the slow movement absolutely of vestibular origin? Even if it does not upset our train of thought in observing any case with labyrinthine symptoms, it fails to do so only because we transpose our terms. We cannot think of it properly without transposition; therefore why should we not at least cease designating nystagmus by the direction of the quick recovery movement?

We might say, in describing symptoms, that a nystagmus of vestibular character is present. This would mean that there is a slow and a quick movement. If we wished to be logical, we might say "the slow movement (the vestibular one) is away from or toward the diseased ear,"—as the case might be. Without transposition of terms the beginner—and the experienced diagnostician also, for that matter—might then have a definite notion of what was happening in the vestibular apparatus.

We have thus far said nothing about the diagnosis or treatment of a chronic case. All chronic cases are characterized by the fact that there are no gross manifest symptoms. We discover indications of a chronic labyrinthitis in the course of a functional examination of a case

of suppurative otitis which has formerly passed through an acute labyrinthitis, with destruction of both hearing and static sense. The clinical history of a previous train of symptoms similar to those just recorded in the acute cases, might lead us to expect to find such a lesion. The demonstration of the presence of a dead labyrinth is perfectly simple. The functional test of hearing will show its complete ablation. The caloric test will show complete loss of function on the diseased side. You will remember that for practical purposes I included as chronic cases all acute cases which had ceased to exhibit spontaneous evidence of labyrinthine involvement. There is no need whatever of hesitating to employ for purposes of experimentation any of the tests which were so severely criticised in the investigation of acute cases. The rotation test may demonstrate a very marked imbalance in the vestibular apparatuses,—the after nystagmus resulting from rotation in one direction being much more marked than that in the other. If this is the case, standing with the eyes closed; walking forward or backward with the eyes closed; walking forward and suddenly looking in either direction; attempting to do things in the dark which formerly could be easily done; standing on one leg; walking or standing on an inclined plane,—will all, in various degrees, show an unstable equilibrium, amounting in some cases to complete loss. If the patient is whirled, and his head placed in one position, he will fall in a certain direction; and if whirled and the head placed in another position, he will fall in another direction. The extent of his response to these experiments will depend upon the rapidity with which he has compensated for the loss of function of one of his vestibular apparatuses.

In a general way, the length of time following the complete destruction, will have much to do with his responses to these experiments. Nevertheless, just as one person may learn to walk a narrow plank or do some unusual athletic feat very quickly, while another may take a long time to do it or never be able to do it at all, so we may measure the variation in compensation for loss of one static apparatus, that we may find in any such case. It follows, then, that after all, no matter how many experiments we may try from an interest in the phenomena—all of which we may predict if we know there is a dead labyrinth present—we are led always to the confirmation of any evidence we may derive from them by the result of the caloric reaction. If there is any remaining function in either the cochlear or the vestibular apparatus, an operation for draining the labyrinth is unjustifiable.

For practical purposes, therefore, if we have neither the time, nor the apparatus, nor the inclination, it is not necessary to do more than make the functional tests of hearing and the caloric test for vestibular reaction, in order to

NOTE: I wish to make it quite plain that I am speaking of vestibular lesions only. It is well known that a cerebellar lesion would induce a nystagmus in just the opposite direction. Destruction of a labyrinth would produce a nystagmus the slow movement of which would be toward the destroyed side. Subsequent involvement of the cerebellum on that side would reverse the nystagmus. There are many differentiating phenomena brought out by pointing tests, direction of falling, etc., a discussion of which is quite beyond the limits of this paper.

determine—so far as functional tests go—whether or not a chronic labyrinthitis should be subjected to operation. To my mind, a fistula test in these cases is fraught with quite as much danger as in the acute cases, because one never knows how large the fistula may be nor how insecure the barrier which nature has erected between the meninges and the suppurating focus.

It has been stated by a man of wide experience, and reiterated by many who have followed him, that complete compensation for loss of function on one side, as evidenced by the whirling test, is a contraindication to invasion of the labyrinth at the time of a radical operation, on the ground that in such cases the labyrinth has been healed by the deposition of bony or fibrous tissue in place of the former purulent material. This seems an irrational conclusion, inasmuch as I have operated on several cases in which the whirling test showed almost perfect compensation, yet which at operation revealed necrosis of the labyrinthine wall and purulent foci in both the cochlea and vestibule.

The fiat has gone forth from another man of large experience, and has been widely accepted, that no radical operation should be performed on a chronic suppurating ear, in which a dead labyrinth has been demonstrated by functional tests, without opening the labyrinth at the same time. This also I think is an unwarranted conclusion, inasmuch as a radical operation on such a case of very long standing seems justifiable without invading the labyrinth, unless a very definite lead into the labyrinth is revealed on careful inspection. This is more than ably supported by the conclusion just mentioned by the other man of wide experience.

From my own experience, I would say that no radical operation should be done on an ear showing a dead labyrinth without the intention of entering the labyrinth if careful inspection showed any lead in that direction.

The reason why this fiat went forth was, I believe, because so many such cases operated, without drainage of the labyrinth at the same operation, resulted in the conversion of a latent focus in the labyrinth into an acute meningitis. The cause for such a catastrophe could be attributed only to a traumatic rupture of barriers which had been walling off the purulent focus from the meninges. This only emphasizes the position of those who insist that operation in such cases should be done with extreme caution for the avoidance of concussion or rough manipulation of any area which might possibly be leading into the labyrinth, unless such manipulation was to be immediately followed by free opening into both vestibule and cochlea.

Those who will operate with such care that they feel certain they have not broken down barriers which have held for months or years,

may—if they find no lead into the labyrinth—safely omit drainage of the labyrinth, with the expectation that the majority of such cases will recover without it. Occasionally a case may require a later labyrinthine exenteration, owing to the fact that latent foci of infection were present, which did not show sufficiently gross evidence on inspection to lead the operator to enter it at the first operation. It is my conviction that this practice would avoid the necessity of the more extensive operation in many instances, and would not subject the patient to added danger in those cases that may subsequently require the second operation.

A word as to the kind of operation advisable in these different cases.

Inasmuch as no acute case is operated unless early symptoms of meningeal involvement are present, it follows that any operation is inadequate which falls short of draining the dura at the same time that the labyrinth is opened. Such cases should have the vestibule opened both in front and behind the facial nerve, the cochlea uncapped, and the scala and modiolus entirely removed so that the meningeal fluid washes freely through. The plate from the lateral sinus to the petrous pyramid should be entirely removed, a slit made in the dura as near the internal auditory meatus as possible, and drainage of the meninges at that point facilitated by a ribbon of rubber tissue inserted through the slit. A similar drainage of the temporo-sphenoidal region should be made if there is evidence of rapidly extending meningitis.

Any chronic case exhibiting evidence of meningeal involvement should have a similar operation. The chronic case, however, which exhibits only a suppurating labyrinth, should be opened in front and behind into the vestibule, the cochlea uncapped with extreme care to avoid breaking the modiolus down to a point which will open a communication with the meninges. If the granulating, suppurating cavities are opened and washed out, they will heal rapidly. It is quite inadvisable, in my opinion, to curette granulations from these cavities, for fear of opening up an avenue of infection either through the aqueductus cochleae or the aqueductus vestibuli. It is advisable to avoid removal of these protecting granulations, just as we now avoid curetting the granulations in epidural or perisinuous abscesses. Free exposure, washing, and adequate drainage, will be more successful. The removal of the inner plate, from the sinus to the petrous pyramid, in these cases without any meningeal symptoms, prolongs the operation and increases the danger, without in any way aiding the recovery of the lesion.

I have said nothing definite about the classification of inflammatory conditions of the labyrinth, except to hint at the idea that it had been made too complicated. You will note that I have mentioned only acute cases in which the endo-lymph was invaded, and those which gave

acute symptoms from the peri-labyrinthine inflammation. I have no idea of insisting that the classification into (1) circumscribed labyrinthitis, (2) diffuse serous secondary labyrinthitis, (3) diffuse purulent manifest labyrinthitis, and (4) diffuse purulent latent labyrinthitis, should not be adopted if you wish to do it; but I do insist, since they all arise from a suppurative otitis, that you will be unable to differentiate between the diffuse secondary and the diffuse purulent manifest labyrinthitis in the majority of cases, except by time and the outcome regarding function; and that, having made this distinction, even on that evidence, you may often still be wrong.

Dr. John B. Rae read a very excellent paper on "The Diagnosis of Inflammatory Diseases of the Labyrinth," before the Medical Society of the State of New York at Saratoga Springs, May 16, 1916, in which he simplified the classification into: (1) acute diffuse labyrinthitis; (2) chronic diffuse labyrinthitis; (3) para-labyrinthitis,—(a) with fistula; (b) without demonstrable fistula. To my mind, this covers the ground quite sufficiently, and includes all the cases under the more confusing classification first mentioned. If it were to be put to a vote, I might offer the amendment that the conditions be called: (1) acute endo-labyrinthitis; (2) chronic endo-labyrinthitis; (3) para-labyrinthitis,—(a) with fistula; and (b) without demonstrable fistula,—but I am not at all sure that the amendment might very justly be voted down in favor of the original proposition.

PARAVERTEBRAL ANESTHESIA.

By FRANK C. W. KONRAD, M.D., BOSTON.

THE application of conduction anesthesia to blocking of the spinal nerves at their points of exit through the intervertebral foramina was first used by Sellheim in 1905, and termed by him "paravertebral anesthesia." The more or less imperfect results obtained by him were most likely due to the high degree of toxicity of the drugs, especially of the cocaine, which were used, and which made it difficult or impossible to distribute over a sufficiently large area a sufficient quantity of the drug, and still keep within the bounds of a safe total dosage. Also, he attempted to make the injections as near as possible to the intervertebral foramina, and therefore it is conceivable that some of the drug entered the peridural space by diffusion, or even the dural space by direct puncture of the needle, thus increasing the toxic effect of the drug. Later Braun blocked the sacral nerves along their course in the hollow of the sacrum, and called this procedure "parasacral anesthesia." Numerous other investigators have applied the

conduction anesthesia more or less extensively, but nowhere has it found such general application as at the University Frauenklinik of Freiburg, Germany, of which Geh. Prof. Dr. Kroenig is director.

My experience with paravertebral anesthesia covers the work done at the Freiburg clinic from June 1, 1914, till Oct. 1, 1915, during which time I was assistant there. I have since then received a report from Dr. Siegel, first assistant at the clinic, covering the work to December, 1915, and including 770 cases.

I am indebted to Dr. Kroenig for the kind permission to use his paper,¹ and to Dr. Siegel for his latest report.² I shall use the term "paravertebral anesthesia" as it is used in the Freiburg clinic, to include "parasacral anesthesia." As the technic of this anesthesia is fully described in Dr. Kroenig's paper, I shall not again review it here, but wish to add a few notes on cases that I have done since my return from Germany, including some of the later observations from the Freiburg clinic as well.

Through the kindness of Dr. N. R. Mason, first assistant visiting surgeon to the Boston City Hospital on the gynecological staff, I was given opportunity to make my first public demonstration of paravertebral anesthesia in this country.

A multiparous woman 37 years of age, had come to the hospital for treatment of extensive lacerations of the perineum and prolapse of the uterus, but, owing to a daily elevation of temperature from 99 to 101°, operation was postponed. There was an obvious tubercular process involving both lungs, and, as this did not readily improve under treatment in the wards, she was about to be discharged as inoperable on account of it. At this time the advisability of operating under paravertebral anesthesia was considered and finally decided upon.

An extensive perineal repair, followed by a ventral suspension of the uterus and appendectomy were done. Only at one point during the course of the operation did the patient evidence any sign of pain, and that was at the time of packing back the intestines with a Weston strip. At this point the patient was given a whiff of ether, but after operation remembered neither the pain nor the ether. The rest of the operation was completed without further mishap. There were no apparent after-effects of the anesthesia. The patient was allowed to drink water on the operating table during the course of the operation, and immediately afterward. During the afternoon she was given chicken broth, and in the evening a light supper, going onto a light house diet on the day after operation. On the afternoon of the day after operation the patient's temperature rose to 101.4°, but after that fell gradually to 98.2° on the fifth day after operation, and did not again rise above 98.6°. She was discharged, relieved,