

The Journal of Laryngology and Otology

(Founded in 1887 by MORELL MACKENZIE and NORRIS WOLFENDEN)

December 1938

A CONTRIBUTION TO THE STUDY OF MIDDLE-EAR SUPPURATION WITH SPECIAL REFERENCE TO THE PATHOGENY AND TREATMENT OF CHOLESTEATOMA

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(Continued from page 709)

THE HISTOLOGICAL EVIDENCE FOR THE EPIDERMIC NATURE OF CHOLESTEATOMA

It has always been assumed that, under the microscope, the outer layers of a cholesteatoma were demonstrably epidermic in nature. I do not think that this is correct. McKenzie's drawings are most illuminating on this point. He shows part of a cerebrospinal epidermoid in which perfect prickle cell formation is visible. Below it he shows the cholesteatoma tissue which he claims is identical. On examination of its multicellular layer, we can see no sign whatsoever of prickle cells. It is a multi-cellular tissue, such as might easily arise from chronic irritation of a pavement epithelium. Furthermore, no other authority, to my knowledge, has ever claimed to have seen anything more convincing than this. Not even in Wittmaack's album do we see any such evidence.

Speaking entirely as a clinician, it has always seemed to me extraordinary that no one has ever demonstrated true skin in the *healed* lining of a cholesteatoma cavity. In the inflamed lining we can always find a multi-cellular desquamating layer,

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and yet as soon as we cure the inflammation this lining loses all resemblance to epidermis. True prickle cells (so far as I know) have never been demonstrated, and certainly to the naked eye this healed membrane looks exactly like the epithelial lining of the normal promontory.

In addition, it is usual to refer to the "typical keratinized layers". Admittedly, these are regularly found in cholesteatoma, but are they proof of an epidermic origin? Certainly not! They are merely proof of a desquamating multi-cellular tissue. We have only to remember that such a tissue with its desquamating surface layers is quite commonly found in bronchial carcinoma (and even bronchiectasis) to realize that there is nothing in the keratogenic power of cholesteatoma to prove an origin from epidermis.

The contribution of Jefferson and Smalley (admittedly of the first rank, clinically) is marred by this same unfortunate acceptance of statements which (to say the least) have never been proved. They, as usual, find that all their troubles would be solved if only they could accept the metaplasia theory—but they cannot. Let us see why not. First, because "a differentiation of mastoid mucosal cells towards an epithelial type does not occur". Now if by "epithelial type" they mean pavement epithelium, then the answer is that such differentiation apparently occurs in every human being, in the normal process of development. Perhaps they mean by "epithelial type" a multi-cellular desquamating epithelium. The statement would then be that "a differentiation of mastoid mucosal cells towards a multi-cellular desquamating epithelium does not occur".

The answer to that is—that no evidence has ever been brought to support such a statement.

Such a statement begs the whole question.

Furthermore, evidence is by no means lacking that such a metaplasia from ciliated columnar to stratified squamous epithelium can occur.

(1) Lyman Richards,²⁸ in describing a case of acute laryngo-tracheo-bronchitis which was fatal in three days, shows a section of a bronchus with the following description :—

"The bronchial lumen contains large masses of hyalinized desquamated tissue infiltrated with products of inflammation. Focal areas of necrosis appear in the epithelial surface although

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portions are still intact. At C is seen the reaction already taken place of a metaplasia to a more stratified and squamous type of epithelium." Again,

(2) G. E. Hodge,²⁹ in a paper on bronchiectasis, writes, "The reaction of the mucous membrane of the respiratory tract to infection is the same whether situated in the upper or lower portions, and in long continued infections a metaplasia of the epithelium from a ciliated columnar to a stratified squamous takes place."

Let us, however, turn for enlightenment to Nager²⁶ himself, who is quoted as the source of "the best pathological work". He writes as follows:—

"For the better understanding of the Bezold-Habermann theory it should be pointed out that the mucous membrane of the tympanum, especially in its higher parts, consists of a flat and frail epithelium, which was originally ciliated, as is usually seen in the neighbourhood of the Eustachian orifice. Under the influence of inflammation, however, this low or flat epithelium betrays its character as mucous membrane, because it becomes cylindrical and so reminds us of its origin. Owing to necrosis of mucous membrane, ulcerations arise in the tympanum.

"The formation of epithelium cannot proceed from the cylindrical layer, because, as is well known, this kind of epithelium does not possess any regenerating power. The squamous epithelium of the meatus, with its strong growing tendency, will push its way over the bared margo into the middle ear, far more easily.

"We find similar conditions in the nose after long suppurations, where the mucous membrane becomes overgrown and is replaced by the epidermis.

"In its pathogenic aspect the cholesteatoma in cases of isolated perforation of the membrane Shrapnelli assumes a place of its own, because the middle-ear suppurations never perforate at this spot and cannot be held responsible for the origin of cholesteatoma."

Within the last ten years we have seen a mass of evidence gradually accumulating which is directly opposed to Nager's conceptions. The regenerating power of ciliated epithelium has actually been shown by numerous authors to be enormous.²⁷ Admittedly most of their work has been done in reference to the relining of the maxillary antrum, but similar evidence

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is also available in reference to the intestinal mucosa. Presumably the tympanic mucosa is entirely comparable in its properties—certainly no one has ever produced any evidence to the contrary.

The occurrence of “similar conditions in the nose” needs no discussion. The exceptional rarity of cholesteatoma in the nose makes any such comparison absolutely invalid. Lastly, “that middle-ear suppurations never perforate the membrane Shrapnelli” is certainly untrue. I can readily confirm, from my own experience, Otto Mayer’s description of an acute perforation in this region. Indeed, it constitutes the definite clinical entity which I have described here, namely, “acute perforating epitympanitis”.

There is no need to pursue this matter farther. Presumably we have not reached certainty. I shall be content if I have aroused some doubt in the minds of clinicians.

PROPOSED NEW CLASSIFICATION FOR INFLAMMATIONS OF THE MIDDLE-EAR CLEFT

A review of these arguments leads inevitably to the conclusion that we are in urgent need of a standard terminology to deal with the various lesions herein discussed.

The word cholesteatoma should indeed be abandoned. Admittedly, the cholesteatoma “perle” is a striking pathological entity, nevertheless it is one which can be closely simulated by quite different conditions, such as the cerebrospinal epidermoid. Certainly the presence of cholesterol crystals cannot in any way be considered as specific. These crystals are found in all kinds of chronic accumulations. Lastly, as I have shown, the otologist is constantly dealing with middle-ear conditions of a quite diverse appearance but of identical origin. Probably no two otologists would agree as to which of these could be allowed, and which denied, the title of “cholesteatoma”.

We classify the infections of ciliated epithelium under the general heading “mesotympanic”. In the case of the pavement epithelium I have made the further subdivision into epitympanic (attic), retrotympanic (mastoid) and pretympanic (petrous). Any such classification has the usual disadvantage that there is often a continuous gradation between different groups. Thus, it is impossible to draw a sharp dividing line

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between acute suppurating and acute catarrhal otitis media. Nevertheless, each of these is a well-defined clinical entity.

I have attempted to stress the essential difference between the two epithelia by the use of different adjectives. Thus the ciliated epithelium reaction is either catarrhal or suppurative. The comparable reactions of the pavement epithelium are either the desquamative (which I have called enclosed to stress the outstanding feature of the primary cholesteatoma) or perforating as seen typically when an acute polyp perforates Shrapnell's membrane in perforating epitympanitis.*

Lastly, for clinical completeness, I have included *gross suppurations* in the epi-, retro- and pretympanum. Strictly speaking, these are not reactions of the pavement epithelium, but of the underlying bone. Acute and chronic suppurations of the bone of the mastoid, or petrous processes, are far too important to be excluded. Nevertheless, it must be repeated that suppuration is not a characteristic reaction of pavement epithelium as I see it.

Taking the groups in detail we may first note that reactions in the sub-groups of epi-, retro- and pretympanitis are practically identical. In each case a chronic enclosed infection will yield the condition, so well known as the primary cholesteatoma (*wahre cholesteatomtumor*).

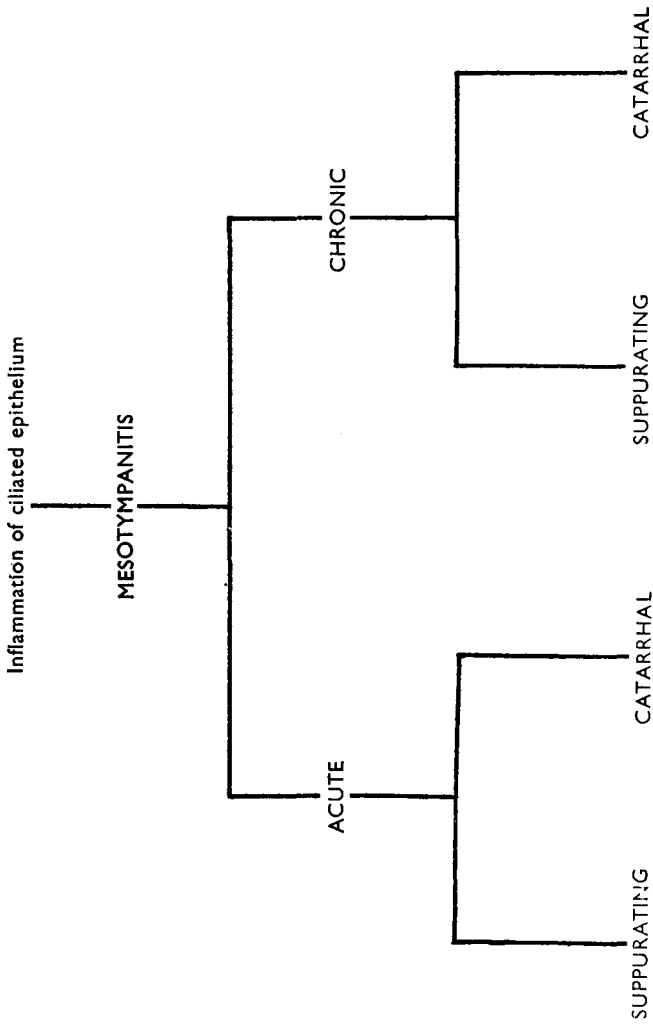
When the condition perforates we get the standard type of secondary cholesteatoma.

The acute conditions are also closely allied. The acute suppuration of mastoid or petrous needs no further description. In the case of the epitympanum, however, I should like to draw attention to "acute perforating epitympanitis" in which there is comparatively little purulent exudate, but Shrapnell's membrane is perforated by the acute epitympanic polyp. The acute catarrhal (or rather enclosed) inflammations of mastoid and petrous are unlikely to be encountered clinically, although it is obvious that they must occur, and that they are the starting point for the primary cholesteatoma. The accumulation of a few squames or even polyps in the mastoid or petrous

* It might perhaps be more accurate pathologically to label this type as "plastic" to stress the presence of the polyp or granulation tissue. The word perforating, however, *does* convey the outstanding feature of this condition, namely, that the drum is eroded, thus leading to all the associated complications of secondary infection.

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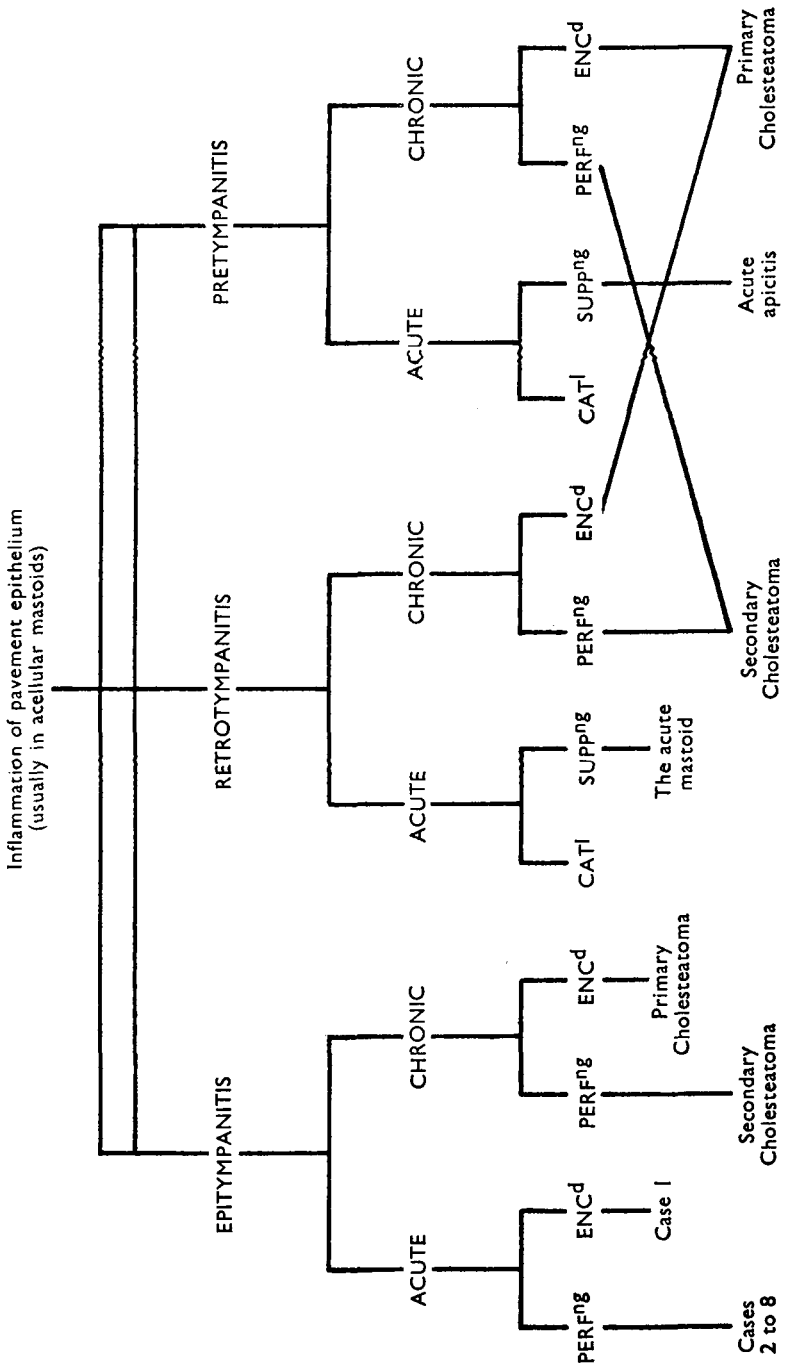


FIG. 10.

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cells is not likely to produce any immediate dramatic symptoms. In the epitympanum, on the contrary, a quite trivial accumulation can be a very serious matter. The incudo malleolar articulation becomes embedded in a glutinous mass and the hearing becomes markedly, if not permanently, impaired.

Case I is typical, and I have observed a similar one recently. In addition, two patients who were treated for acute perforating epitympanitis now have dry ears, but are obviously not normal. Shrapnell's membrane is congested rather than retracted, there is still impaired hearing and troublesome tinnitus. I am convinced that this condition provides the explanation for those by no means rare patients in whom a comparatively mild otitis is followed by persistent and progressive deafness. May we not, indeed, suspect that a similar pathogeny will explain some, at least, of those inexorable types of progressive middle-ear deafnesses in which no history of any acute otitis is obtainable?

The mesotympanic inflammations need no elaboration. The four groups are all clinical entities, which will be recognized immediately on substituting "otitis media" for mesotympanitis. For instance, acute catarrhal mesotympanitis is the same as acute catarrhal otitis media, and so on.

DIFFERENCES BETWEEN THE AUTHOR'S THEORY AND THE METAPLASIA THEORY

The exponents of the metaplasia theory have given different versions of it from time to time, some accepting the classic epidermoid theory for primary cholesteatoma, others not. In the main, however, they are at one with the writer in deriving the secondary cholesteatoma from tissues normally present in the attic, antrum and mastoid. Despite this, even whilst propounding the metaplasia theory they have apparently adhered to the view that the cholesteatoma suppuration was in some peculiar way different from other suppurations in the *same situation*. In other words, they accepted Groups 2 and 3 as different entities.

We can, therefore, summarize our differences as follows:—

- (1) We deny the extraneous or fortuitous origin for

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primary cholesteatoma as for secondary cholesteatoma. (See p. 747.)

(2) We affirm the identity of *all* posterior-superior perforations. All the protean manifestations of this condition outlined under Groups 1 to 4 (not to mention others such as enclosed epitympanitis) can be explained by stressing one or other of the characteristics of the pavement epithelium.

(3) We deny, in fact, that metaplasia takes place. As pointed out on p. 694, the conception that cholesteatoma is produced from metaplastic ciliated mucous membrane is quite unnecessary, and leads to fresh difficulties. On the other hand, there is ample evidence to show that by no means all mastoids are equipped with epithelium capable of producing typical epitympanitis. It practically never occurs in well pneumatized bones, although these are much commoner than other types.

We agree, therefore, with Wittmaack that in earliest infancy or even *in utero*, some reaction must have occurred, which not only interfered with normal pneumatization but also modified the cellular lining so as to give it a predisposition to epitympanitis in later life.

KÖRNER'S EIGHT CRITERIA

(1) We are dealing with true cholesteatoma when, together with a dermoid tumour, no perforation or scar is found in the drum.

(2) We are dealing with true cholesteatoma, as in a case of Schwartze's, when the tumour has produced, through destruction of the labyrinth, a nerve deafness without preceding otitis and suppuration for many years before it declares itself by opening externally.

(3) We are dealing with true cholesteatoma when there is a perforation in the drum whose condition makes epithelial immigration impossible.

(4) True cholesteatoma is present when the delicate enclosing membranes of the tumour itself are completely intact in the middle-ear cleft.

(5) We are dealing with true cholesteatoma when the tumour is in a position where, so far as our present knowledge goes, epidermis never invades.

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(6) We are dealing with true cholesteatoma where suppuration has been present for a short time, and the tumour is so big that it cannot possibly have developed in that short time. This applies especially to cases in children.

(7) According to Kummel a true cholesteatoma is present when we find on opening a mastoid that the whole process is full of a dry pearly tumour, which can be easily removed from the bony wall, leaving the latter apparently polished with only an occasional indentation here and there.

(8) We speak of true cholesteatoma when the tumour is covered or intermingled with gold or silvery shining cholesterol crystals, and when the latter are present to a marked degree in the pus.

Let us now analyse these criteria in the light of the preceding arguments.

No. 8 can obviously be rejected at once as being without any significance at all. With regard to the first seven criteria, it should now be quite clear that these are merely the characteristics of chronic enclosed epitympanitis (or retrotympanitis, or pretympanitis, as the case may be). An exacerbation which finally results in perforation of the drum with external discharge makes this a chronic perforating epitympanitis, as visualized in criteria Nos. 2, 3 and 6. We are able to recognize from these criteria that such a chronic perforating epitympanitis did, in fact, originate as a chronic enclosed epitympanitis, and not as an acute perforating epitympanitis.

Let us compare three hypothetical patients each complaining of a scanty malodorous discharge of eight weeks' duration, onset insidious, slight deafness, and so on. Case A—under conservative treatment, heals. Case B—resists treatment and finally a mastoid exploration reveals that the antrum is partly filled with granulation tissue and a little whitish lamellated detritus. Case C—also resists treatment, and operation reveals a large cavity occupying much of the mastoid, and occupied by a cholesteatoma *perle*.

Standard theories find little or no relation between these three conditions. C being labelled a congenital tumour which has fortuitously eroded through the tympanic membrane. B is a cholesteatoma, either a "genuine cholesteatoma" which has become infected, or a secondary cholesteatoma produced by epithelium growing into the attic *viâ* an inflammatory

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perforation. A is a form of chronic otitis media. The writer's theory, on the other hand, unites these three apparently diverse entities as follows.

A and B are essentially identical, being both "acute (or subacute) perforating epitympanitis". Case C arose also as an epitympanitis, but in this case the reaction was much more insidious, having been present as a chronic enclosed epitympanitis for many years. During those years the "perle" was formed by the continued desquamation of the inflamed lining. All three cases are inflammatory reactions of the epitympanum.

The important point, therefore, is that Körner's criteria can in no way be regarded as proof of a congenital origin; they merely indicate that *some pathological process* has been present for some time before the discharge declared itself externally. That process, however, was not a congenital tumour formation, but simply a chronic enclosed epitympanitis.

DOES THE MIDDLE EAR NORMALLY CONTAIN ECTODERM?

The embryological statements made by Teed are of considerable importance in this problem, although we need not necessarily agree with his deductions. It is to be hoped that other workers will make similar investigations, and supply us with the necessary confirmation. Briefly, he draws attention to the epibranchial placode—an ectodermal thickening occurring at the junction of the pharyngeal pouch with the branchial cleft. These placodes occur in pairs, and are closely associated with the ganglia, in particular the facial and glossopharyngeal. In fish they produce the spiracular organ, and in birds the paratympanic organ. In mammals, this epiblastic structure is vestigial, nevertheless it is said to be represented by an epiblastic thickening in the dorsal pole of the tympanum. Teed thus claims that epiblastic elements are normally present in the hypoblastic middle-ear cleft. That under certain (unknown) conditions they can take on anomalously the function of ordinary epidermis, and so produce the true cholesteatoma. He also quotes Prěcechtěl as having found "ectodermal splinters in various other parts of the middle-ear cleft".

It will be seen at once that this evidence, if confirmed, would apparently bolster up McKenzie's theory.

It is also able to supply "anlagen" for primary tumours

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wherever we want them. It is a minor difference that McKenzie regarded the mere presence of the cells as anomalous, whereas Teed claims they are constantly present, but have to be stirred into activity by some unknown stimulus.

At this point, it will be apparent that Teed's statements can equally be regarded as supporting the metaplasia theory as well. On p. 694 I have already declared that the pathological evidence suggests that there are two quite different types of tissue in the middle ear, and I there suggested a mesoblastic origin for the posterior-superior type. In point of fact, there is no objection to an epiblastic origin, so long as we remember that these tissues are the normal lining. It does not matter where the *lining comes from so long as we realize that cholesteatoma manifestations are the standard reactions of the normal lining and not freak appearances due to occasional abnormalities.*

It is further to be noted that Teed also apparently accepts the immigration theory for secondary cholesteatoma. At any rate, he distinguishes emphatically the "true primary or congenital cholesteatoma" from the "secondary type" produced as a result of chronic infection.

This, I need hardly say, is diametrically opposed to the present thesis and must be rejected. Incidentally, Teed gives no hint as to what he thinks is the exciting factor which *occasionally* stimulates these normally placed ectodermal cells to take on an anomalous activity and thereby to produce a "true cholesteatoma".

THE EXPERIMENTAL PRODUCTION OF CHOLESTEATOMA

It was only natural that efforts to prove the nature of cholesteatoma should be made by means of animal experiments. Berberich claimed to be able to produce it by irritating the external auditory canal with tar and similar products. He maintained that chronic external otitis was a potent predisposing cause of cholesteatoma. Not only is this contention contrary to general clinical experience, but it seems apparent that the lamellated masses which he produced were not true cholesteatoma. In any case, the production of such a condition can tell us nothing about the middle-ear cholesteatoma. McKenzie has pointed out that it is not unknown in man. (I have never seen a case and so cannot discuss it.) It seems very remarkable that an epidermis which so rarely (if ever) produces cholesteatoma in its normal situation, should develop this

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amazing capacity as soon as it enters the sacred confines of the attic. Certainly it may be argued that in the outer ear the exfoliated surface layers are not confined as they would be in the attic and therefore the "perle" does not accumulate. In this respect it is very instructive to examine the behaviour of a radical mastoid cavity lining, in which healing has remained imperfect. Such a cavity is capable of producing typical cholesteatomatous débris indefinitely, and we note two curious facts:—

(1) That the formation never arises from the true skin obtained by the plastic operation on the posterior meatal wall.

(2) That it arises from the apex of the mastoid or the aditus, or some part of the original cholesteatomatous lining. *But this area is no longer confined.* It is in free communication with the external world. Why, then, does it go on producing cholesteatoma even though the immediately contiguous skin flap obstinately refuses to do so? Must we not conclude that the two tissues are entirely different in origin?

Milstein has attempted to produce cholesteatoma experimentally in cats and dogs. His technique consisted in laying skin flaps into the mastoid, and deliberately traumatizing the drum and the bony cavity. The resulting mélange invariably became infected, and it is not surprising that cholesteatomatous appearances were produced. It is not easy to deduce anything as to the pathogeny of cholesteatoma from this work, especially when we read the somewhat lax criterion which Milstein sets.*

The underlying principle in these experiments is none the less valuable. "Can we produce cholesteatoma by burying true skin in the mastoid cavity?"

We may obtain some information on this point from an operation which I have called the "quadruple flap operation". †

Briefly, this starts with a skin incision AB in the post-aural

* " sans nous attarder a mentionner les caractères et les symptômes bien connus du cholestéatome chez l'homme, nous devons néanmoins signaler que tous les agglomérats possibles constitués par les cellules, en voie de désagrégation d'un epithelium faisant preuve d'un accroissement exagéré, agglomérats qui representent des tumeurs composées de masses epitheliales entremêlées de pus et dont le signe distinctif est la coloration bleue que les éléments keratinisés prennent avec l'hématoxyline, sont considérés par nous, chez les animaux, comme des formations cholestéatomeuses."

† I need hardly say this procedure was not undertaken as an experiment in cholesteatoma, but as a contribution towards the rapid epithelization of the mastoid cavity. Its detailed technique, indications and results, will be the subject of a later contribution.

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sulcus (or even encroaching on the back of the auricle). This is supplemented by incisions BCD as in Figure 11 and the resulting pedicle is dissected subcutaneously and turned down. The deep tissues are then elevated as a flap with its pedicle at the upper margin, Figure 12. The bony operation is completed and finally the meatal plastic is performed by splitting the posterior wall down its centre, as in Siebermann's flap.

The upper part of the meatal wall is turned upwards and sutured to the temporal fascia and to the deep tissue pedicle, Fig. 13. The lower half is turned over the facial ridge and sutured to the original skin flap, *which is thus drawn into the mastoid cavity*. The operation is completed by suturing AB to CD so that the skin flap is buried except for its pedicle at P (Fig. 14). I hoped in this way to obtain rapid epithelialization of the cavity, and I was prepared to deal with the pedicle at a later date by dissecting it up. In actual fact this has not been necessary, as in no case has it given the slightest trouble. Nevertheless, in two patients a curious complication occurred, which resulted in a condition closely similar to Milstein's experiments.

Namely, owing to intense post-operative œdema, the upper and lower halves of the meatal wall became adherent, *so that the skin flap became truly buried in the cavity. Here, surely, we have the conditions for cholesteatomatous formation*. In point of fact, both these cases were children with typical chronic perforating epitympanitis (secondary cholesteatoma), nevertheless neither of them shows the slightest evidence of cholesteatomatous degeneration in the pedicle. (We should easily recognize any such débris emerging at P which is now the orifice of a narrow duct leading into the mastoid, exactly as a perforation in the pars flaccida leads into the attic.)

It is apparent, then, that true skin embedded in the mastoid does not undergo cholesteatomatous degeneration, even though chronic epitympanic infection is already present. (I may add that both bones were of the usual diploetic type, so that the conditions were completely favourable to cholesteatomatous formation.)

Admittedly, we have not produced an exact copy of the natural conditions under which cholesteatoma occurs, nevertheless the similarity is striking, and I cannot help feeling that we are justified in accepting these two cases as *evidence against the epidermic theory*.

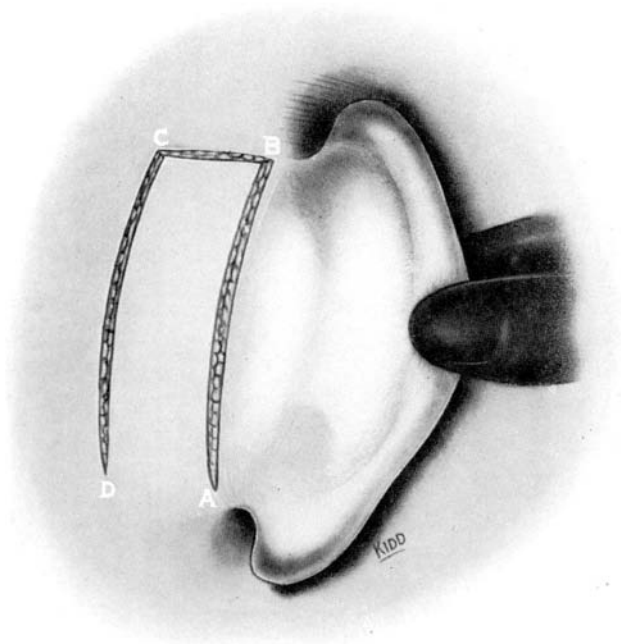


FIG. 11.

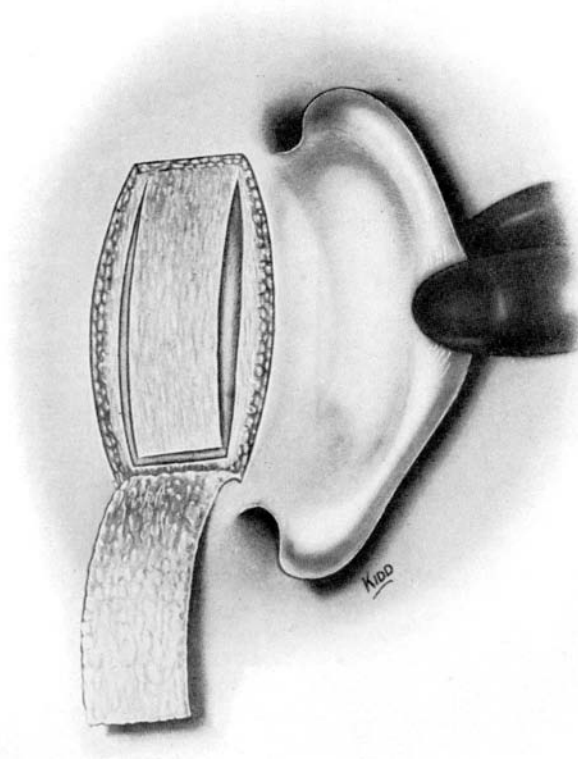


FIG. 12.

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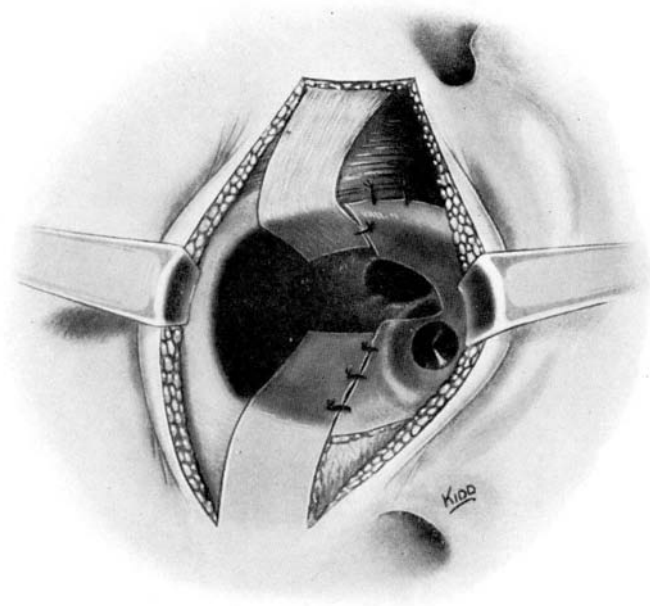


FIG. 13.

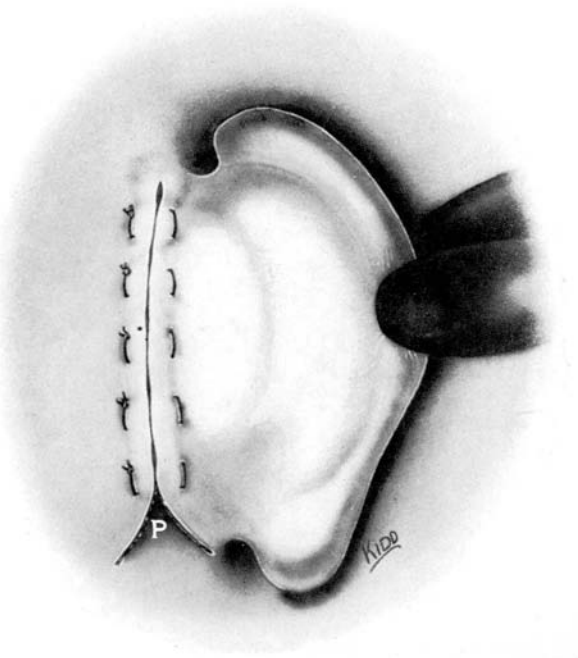


FIG. 14.

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PRACTICAL IMPLICATIONS OF THE THEORY

We have been led to the conception of a definite type of temporal bone in which the process of pneumatization has failed, and the result is an acellular bone. The lining of the attic and antrum in these cases seems peculiarly prone to infection.

The conception is by no means devoid of practical implications. To begin with, it accords with, and indeed fully explains, a clinical observation which has been made repeatedly. Namely, that in the majority of radical mastoid operations, the general mastoid structure is "sclerosed". Unfortunately, the erroneous belief is still widely held by otologists that this sclerosis is a post-inflammatory reaction, and the bone must therefore be regarded as unhealthy.⁴⁰ Such a belief cannot be refuted too emphatically. Admittedly, exacerbations of infection can lead to caries and necrosis of the ossicles, and of the osseous wall of attic and antrum, nevertheless it is a matter of constant observation that chronic perforating epitympanitis can exist for years without causing any gross bone disease, and without damaging the ossicles. This is proved conclusively by three observations :—

(1) That a very considerable number of these cases can be cured by conservative means.

(2) That even when a mastoid operation is performed, the surgeon finds no need to curette the cavity.

(3) An experienced radiologist can frequently state quite emphatically, before the operation, that there is no evidence of osteosclerosis.

It has been pointed out repeatedly that scrupulous respect for the cholesteatoma lining frequently results in a rapid "epithelialization" of the cavity. In other words, there is no underlying osteitis. Not only is the bony mass healthy, but the mesotympanum and the pars tensa are in the majority of cases unaffected. We are led irresistibly to the conclusion that :—

(1) Radical operations entailing widespread removal of healthy mastoid bone, healthy ossicles and healthy drum are emphatically contra-indicated.

(2) Conservative operations such as Heath's, in which the tympanic ring is preserved, are just as unsatisfactory. The healthy mastoid bone is unnecessarily removed whilst the

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essential drainage of the epitympanum is not achieved, because the perforation is still confined by this same bony ring. Furthermore, if caries should occur, it is only too liable to attack this very spot. In other words, the bone which has been left is the only part the removal of which is imperative.

(3) Conservative operations of the Bondy type, i.e. the trans-mastoid atticotomy, are certainly more satisfactory. These operations are coming more and more into favour (Howarth and Bateman³⁷). They have the great virtue that removal of the mastoid bridge and outer attic wall provides adequate drainage of the attic. Nevertheless, an outstanding disadvantage still remains, namely that much healthy mastoid bone has to be sacrificed. Further disadvantages will emerge when we contrast this with the alternative operation of trans-meatal atticotomy.

Before describing this latter operation, we may properly digress to discuss the conservative treatment of epitympanitis. This is begun essentially with a careful X-ray of the temporal bone. Information is thereby obtained as to the possible presence of a chronic retrotympanitis or pretympanitis, such as would indicate more heroic measures. Usually the report will be "diploetic mastoid", less commonly "compact ivory mastoid", and sometimes "poorly pneumatized". In addition, the radiologist should report as to the presence of post-inflammatory sclerosis. In general it may be said that whilst practically no case need be denied the chance of conservative treatment, yet the outlook is poorer in those cases in which pneumatization is more or less complete, and in which inflammatory sclerosis has extended into the mastoid bone. This is not to be interpreted as in any way opposed to the thesis that pneumatized bones are much less susceptible than acellular bones. We reconcile the two statements on the pathological grounds that although infection rarely invades the pneumatized bone, yet when it once gets a grip of such a bone, it is able to spread much more widely into the retrotympanic region where it is less accessible to conservative measures.

In addition, the surgeon will attempt to decide by means of a careful history whether he is dealing with a case of acute perforating epitympanitis or a chronic case of much longer standing.

The determining factor in any case is the presence or

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absence of a polyp. It is clear that drainage of the epi-tympanum is impossible when the perforation is blocked by a polyp. Such a case will "suppurate" indefinitely.

The essential destruction of the polyp can be achieved in many ways. In a few cases, especially those of recent onset, the frequent application of boric-iodine powder will desiccate the tissues and result in a cure. Usually it fails, and more active measures will be required. The direct application of chemical caustics is favoured by many authorities, but in my opinion it is much inferior to the electrical methods. In zinc ionization, which is the standard treatment for "chronic ears", the chemical effect is obtained by the electrolytic deposit of zinc ions which combine with tissues to form zinc albuminate. Now the density of this ionic deposition in any given area will depend on the density of the current passing through that area. Ordinary untreated skin has a considerable resistance owing to its natural greasiness. Granulation tissue, on the other hand is a very good conductor. When, therefore, we fill the external auditory meatus with a zinc solution and pass a current through it, the greater part of the current traverses the granulation tissue in preference to the healthy skin. Thus the really dense deposition of zinc ions occurs exactly where we need it. This explains the outstanding success of zinc ionization in suitable cases. That is to say, in chronic otorrhœa without gross bone disease, and associated with a drum defect large enough to permit the zinc solution to reach all the diseased tissues. It is clear that some cases of epitympanitis will not fulfil the first proviso, and that very many will not fulfil the second. Even when contracted by strong cocaine and adrenalin, the polyp is still seen to be almost strangulated by the margins of the perforation. Under these circumstances, zinc ionization merely attacks the external surface of the polyp. It is not feasible to eradicate such a polyp even by repeated treatments, because in between the treatments it grows again (see Fig. 2, plate facing p. 692).

A more drastic method is required, and this is supplied by the method of bipolar electrolysis. For this purpose the author has designed a special electrode consisting of two zinc needles, six inches long, closely bound together, but well insulated by shellac. The blunt ends of the needles are connected to the usual source of direct current, whilst the sharp ends, which are left free of insulation for a distance of an eighth

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to a quarter of an inch, are plunged into the polyp. In this way a current is passed from one needle to the other through the polyp. Two to three milliamps can be passed for ten minutes or more without risk or discomfort to the patient. The polyp turns greyish-white, and not infrequently comes away with the needle. In other cases it disintegrates within a few days, leaving a fistula through which subsequent treatments can be carried up to the attic.

In cases in which this method fails, it may be supplemented by small incisions into the margins of the perforation so as to facilitate access, or the needle points may be more widely separated and plunged through the drum on either side of the perforation.

In this way not only is the polyp destroyed, but the perforation is widened.* Conservative treatment along these lines will be rewarded, in a considerable percentage of cases, by complete cure, as the following figures show.

Seven cases of acute perforating epitympanitis were all cured. The duration of treatment varied from one to nine weeks. Out of thirty-eight cases of chronic perforating epitympanitis, twenty-six were cured and twelve resisted treatment. It will thus be seen that, despite the notoriously bad reputation of this type of otitis media,³⁹ it can be treated conservatively with a considerable measure of success. All these cases had been discharging for many years, several for over twenty and a few for much longer.

Let us now go on to the problem of those cases which resist treatment. As I have already pointed out, the belief that caries of the ossicles or of the bony wall of the attic antrum is the cause of this chronicity is far from correct. Admittedly, pressure of accumulated squames can lead to necrosis of ossicles and invasion of adjacent vital structures. On the other hand, a careful X-ray frequently reveals an acellular mastoid, and the absence of true inflammatory sclerosis. In such circumstances, the surgeon is entitled to assume that the disease is limited to the soft tissues of the attic antrum space, and to plan his treatment accordingly.

A consideration of the arguments on p. 751 leads us to the deduction that the essential point of the surgical attack is to

* Bipolar needles are, of course, well known, but the particular virtue of this double zinc needle is that it can be bent and twisted so as to enter any fistula, however awkwardly placed.

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remove the outer attic wall, with the minimum disturbance, either to the ossicular chain or the tympanic membrane.

This conclusion was reached by McKenzie for identical reasons, although it appears that he never carried his ideas into practice. He says, "Treatment is suggested—namely, in the case of disease limited to this small area and not getting well to remove the thin plate of bone by punch forceps from the meatus so as to provide free exit for the contained debris. As the cholesteatomatous lining may be left *in situ*, if other symptoms which indicate wider infection are absent, the mastoid route of access is unnecessary. Consideration of this aspect of our subject, however, raises a wider question. In the past when dealing with cholesteatoma the attention of the surgeon has been focused upon the supposed cause of the cholesteatoma, the suppuration of the middle ear, and his efforts have been applied to getting rid of this disease. But, as we have shown, middle-ear disease may be entirely absent. How many hearing ears have been destroyed by this misconception?"

This operation of trans-meatal atticotomy has, so far as I know, never been performed in England. Certainly there is no published reference to it. Similarly it has received no support in America. Shambaugh³⁰ has roundly condemned it as follows: "This operation, similar to ossiculectomy with removal of the outer attic wall through the meatus, has the same disadvantages: limited visibility, limited accessibility and therefore increased danger of facial nerve injury or stapes luxation. The post-aural route has no disadvantages to compare with the increased hazard of the canal route."

In this case he is criticizing Kecht's³² article on trans-meatal radical mastoidectomy, and it is very unfortunate that neither Kecht nor Shambaugh attempt to differentiate between atticotomy and mastoidectomy. The latter may indeed be a somewhat ambitious operation to undertake *via* the straitened meatal approach. Not so with atticotomy! The writer, even on the limited experience of nine cases, feels entitled to reject Shambaugh's criticism as invalid. Even in a narrow meatus the accessibility is quite adequate. The drum and the attic structures are visualized much better than in the trans-mastoid operations. They are, in fact, completely and constantly in sight throughout the operation. Consequently the risks of facial nerve injury or of ossicular subluxation are correspondingly diminished.

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On the Continent it has been enthusiastically reported by a small number of surgeons,* but roundly condemned by the majority^{34, 35, 36}. Neumann indeed said, "Es is verpont von einer radikal operation durch den Gehorgang uberhaupt zu reden."³³

Before attempting to evaluate the operation of trans-meatal atticotomy, we must distinguish sharply between it and the better known, although obsolete, operation of ossiculectomy. We must also distinguish it from the more extended procedure of trans-meatal radical mastoidectomy. It is true that in ossiculectomy the removal of the ossicles was frequently supplemented by a limited nibbling of the outer attic wall by means of special punch forceps. Nevertheless, it is quite clear that much greater importance was attached to the removal of ossicles than to the bone-nibbling.

In atticotomy the situation is completely reversed. The drum and ossicles are meticulously conserved, but the attic wall and the posterior-superior wall of the external auditory meatus are removed by means of hammer and chisel sufficiently to drain the whole of the attic antrum space.

There should be no need to emphasize further the essential and profound differences between these two operations.

In the trans-meatal radical mastoidectomy, the surgeon extends the scope of his operation and is prepared to remove not only drum and ossicles, but also to follow any extension of the disease into the mastoid and adjacent structures. This may be regarded as an extended ossiculectomy and not really as an extension of atticotomy. I shall show that the somewhat serious objections which have been raised to trans-meatal radical mastoidectomy do not really apply to the minor operation of atticotomy.

Let us now consider the technique of a trans-meatal atticotomy. We will assume that our patient has had a scanty offensive otorrhœa for many years, his drum is intact except for a small perforation in Shrapnell's membrane through which a small polyp protrudes. X-ray reveals a diploetic mastoid

* Lempert has recently described a series of trans-meatal operations of ascending order of magnitude³⁷ based on the excision of a mobile endaural antauricular window through which he is able to expose the various tympanic and para-tympanic structures. These impressive and heroic procedures have nothing in common with atticotomy, being designed for conditions entirely different from epitympanitis.

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with no evidence of inflammatory sclerosis. Conservative treatment as above outlined has failed, and we intend to remove enough bone to drain the attic antrum space and give us enough room to facilitate application of medicaments to the inflamed lining.

The operation is performed under local anæsthesia, partly because it is useful to have the patient's co-operation when the chisel approximates too closely to the facial nerve or labyrinth. Above all, however, the injection of novocaine and adrenalin provides the essential hæmostasis without which the operation would merely be a dangerous groping in a pool of blood. This subcutaneous anæsthesia is supplemented by surface application; the ear being filled with strong cocaine and adrenalin solution for half an hour before the operation. In addition, premedication with morphine and scopolamine (or similar drugs) adds much to the general smoothness of the procedure.

Instruments are of the simplest. Adequate illumination, slotted aural specula, fine gouges and curettes. A triangular flap of skin is removed from the posterior-superior wall of the meatus, the base of the triangle being situated at the posterior-superior margin of the drum. This exposes the fistula with its contained granulation tissue which stands out very distinctly from the adjacent white bone.

Hammer and gouge are then taken to remove the offending wedge of bone.

The close proximity of the facial nerve and external semicircular canal make this a procedure of considerable nicety, in so cramped an operation field. Nevertheless, it can be carried out with precision, especially with regard to the preservation of the ossicles and drum. The resulting cavity is gently cleansed, but not curetted. The patient stays in hospital for a day and then, apart from attending hospital for dressings, can resume his normal life.

In comparison with the major mastoid operations, transmeatal atticotomy is a minor procedure. Except in very nervous patients and young children it can be done under local anæsthesia, especially if supplemented with some form of basal hypnotic. The shorter time spent in hospital is noteworthy. Some patients stay in only a day; apart from complications, no patient need stay in more than three days. After-treatment is enormously reduced. There are no soft tissue flaps to be watched, no post-aural tissues to heal and,

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owing to the small size of the cavity and its accessibility, the discharge is minimal and easily controlled.

Above all, however, the original lining of the attic antrum space is preserved, in accordance with the dicta of most authorities. This is an outstanding advantage of atticotomy in comparison with the trans-mastoid operations, in which a mass of healthy bone is removed so that the resulting large cavity, inadequately lined by soft tissues, continues to suppurate for many weeks, if not months.

The close supervision of ossicles and drum has already been mentioned as a further valuable feature of trans-meatal atticotomy. In this respect the trans-mastoid operation is markedly inferior.

What of the possible disadvantages of the operation?

(1) Admittedly, local anæsthesia is essential in order to secure hæmostasis. There is, however, no reason why it should not be supplemented by general anæsthesia in nervous or young patients.

(2) Danger to the facial nerve and semicircular canal is minimal in skilled hands. The technical difficulties are naturally much increased in patients who have a narrow external auditory meatus.

(3) The operation has been declared to be unsuitable for cases in which the disease has advanced beyond the confines of the attic antrum space. This is probably correct. In many cases, the main essential is to lay open the cavity widely so as to save the patient's life, or to search for a focus which might later threaten life. The patient's comfort, appearance, and even his hearing are of secondary importance.

Trans-meatal atticotomy can have no place in such a problem. It has been maintained that it is quite feasible to expose the outermost confines of the tympano-mastoid tract *viâ* the meatus—in other words, a trans-meatal radical mastoidectomy. The author is unwilling to accept this as justifiable. Such an operation can claim none of the special advantages of atticotomy, and seems to imply considerable risk of damage to vital structures.

Naturally, it is not easy to decide in a given case whether the minor operation will be adequate or not. The decision will be based on a careful consideration of the history and symptoms, and also on the X-ray findings. In any case, however, as I shall show, there is every reason to expect that

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even should the atticotomy not produce a dry ear, yet it can result in a considerable improvement in hearing and provides sufficient drainage of the diseased area to abolish the threat of fatal complications. We must remember that radical operations frequently achieve no more than that.

Apart from this, however, I am quite satisfied that a preliminary atticotomy makes any subsequent trans-mastoid operation much simpler, especially in cases in which it is essential to preserve the hearing. (Case XII.)

The removal of the bridge in a trans-mastoid approach is comparatively simple, but to advance further and remove the outer attic wall is undoubtedly more difficult. At this stage, damage to the drum and dislocation of ossicles are likely to occur, with disastrous effect on the hearing.

The appended case histories are not included with the idea of providing any statistical confirmation of the value of atticotomy. They do, however, illustrate several points which have already been mentioned.

NINE CASES OF TRANS-MEATAL ATTICOTOMY

CASE VIII. Miss E. S. Bilateral otorrhœa since childhood. Right ear practically useless. Attic perforation. Left ear is much the better, although there is a large central perforation as well as an attic perforation. Operation: Right transmastoid attico antrotomy.*

This major operation was undertaken at a time when the writer was ignorant of the possibilities of trans-meatal atticotomy. Despite a carious fistula into the external semicircular canal, the operation cavity healed perfectly. The posterior-superior quadrant of the tympanic ring was removed, but the ossicles and drum were left intact. The result was a considerable restoration of hearing. (Audiogram I.)†

* It is the writer's practice in a case such as this, to operate first on the worse ear in view of the fact that post-operative deterioration of hearing is not unknown. If this occurs after an operation on the patient's "good ear", the result is clearly tragic. It is cold comfort to tell such a patient that she would in any case have lost her hearing eventually, through the persistent otorrhœa. The alternative—that is, to start on the worse ear—has this great advantage that if the operation should be really successful, considerable hearing will be restored to that ear and, at a later date, it is justifiable to risk an operation on the other ear. Of course, if this first operation should fail, the decision with regard to the second ear has to be made. Such a problem arose in Case XII.

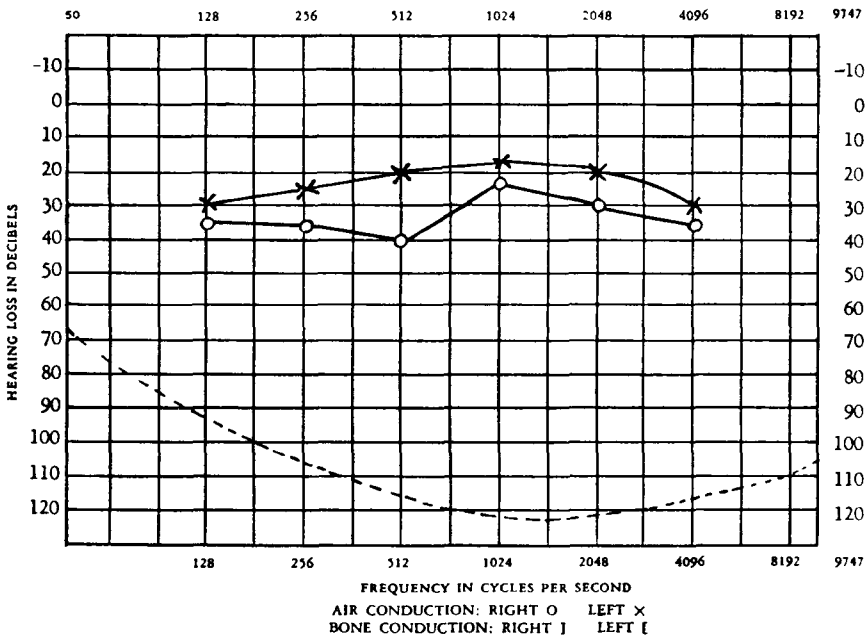
† Unfortunately pre-operative audiograms were not taken in this and several other cases, but it is sufficiently instructive to remember that this ear was regarded by the patient as practically useless.

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The patient at that stage declared that the right ear was equal to, if not better than, the left ear.

Some months later, as this improvement was clearly permanent, the question of operation on the left side was raised, but the patient pointed out that as her hearing was now quite adequate, she was reluctant to undergo the discomforts of another major operation. At that point the alternative of a trans-meatal atticotomy was raised, and in view of its minor nature, the patient agreed to it.

This operation, the first in the writer's experience, was performed



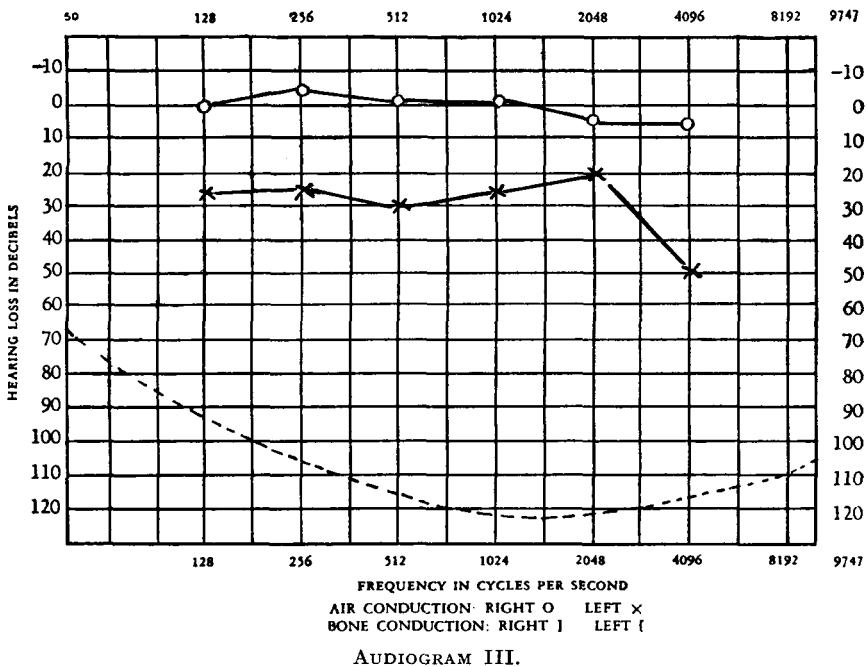
AUDIOGRAM I.

under imperfect conditions, with inadequate premedication, and before its completion a facial palsy supervened. The operation was immediately abandoned. Despite this, and despite the fact that the otorrhœa persisted (although much diminished), the patient's hearing improved considerably, so that once more the left ear is the better one. (Audiogram I.) Incidentally, the facial palsy disappeared completely within half an hour.

CASE IX. Was also undertaken with imperfect technique and inadequate premedication; had to be abandoned because the patient developed vertigo. This man has been lost sight of.

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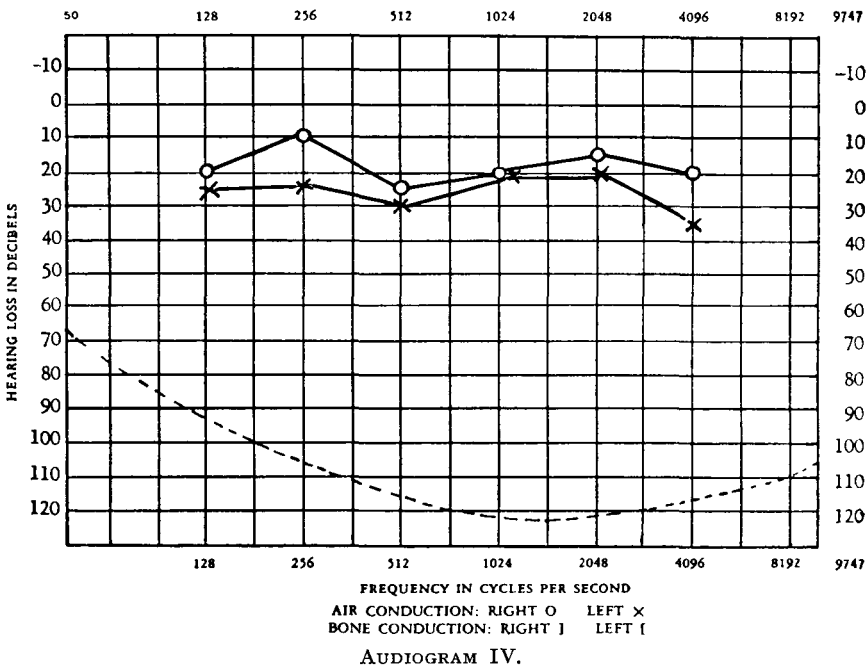
CASE X. Mr. J. R. R. had complained of left otorrhœa for over fifteen years. There was a small polyp protruding from a fistula in Shrapnell's membrane. Conservative treatment, as outlined above, improved his hearing but failed to cure the otorrhœa. A limited atticotomy was performed on January 20th, 1938, and the operation cavity was enlarged on March 10th, 1938. In neither case did the patient stay in hospital overnight. The hearing has, if anything, improved, but although the attic is well drained, and no more polyps are forming, a little discharge continues to appear. (Audiogram III.)



CASE XI. Mrs. E. E. Came under treatment in December 1936, on account of subacute otitis media with polyp formation. Owing to the failure of conservative treatment, the mastoid was explored and found to be practically normal except for a little granulation tissue in the antrum attic. It is quite clear that this was a case of subacute perforating epitympanitis, although the significance of the findings was not realized at the time. Probably the correct operation would have been a trans-mastoid attic antrotomy, or better still a trans-meatal atticotomy. Instead the writer merely did a simple mastoidectomy (Schwartz). Within three months

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the patient was complaining of severe otalgia associated with polyp and crust formation in the pars flaccida, obviously resulting from the failure to drain the attic. Conservative treatment merely gave temporary relief, and so finally a trans-meatal atticotomy was performed. This has resulted in a dry ear and relief from pain. (Audiogram IV.) The writer regards this case as quite important. It is obvious that after the failure of the Schwartze, the next step in the ordinary way would have been either a radical mastoidectomy or a trans-mastoid atticotomy. In the trans-meatal atticotomy,

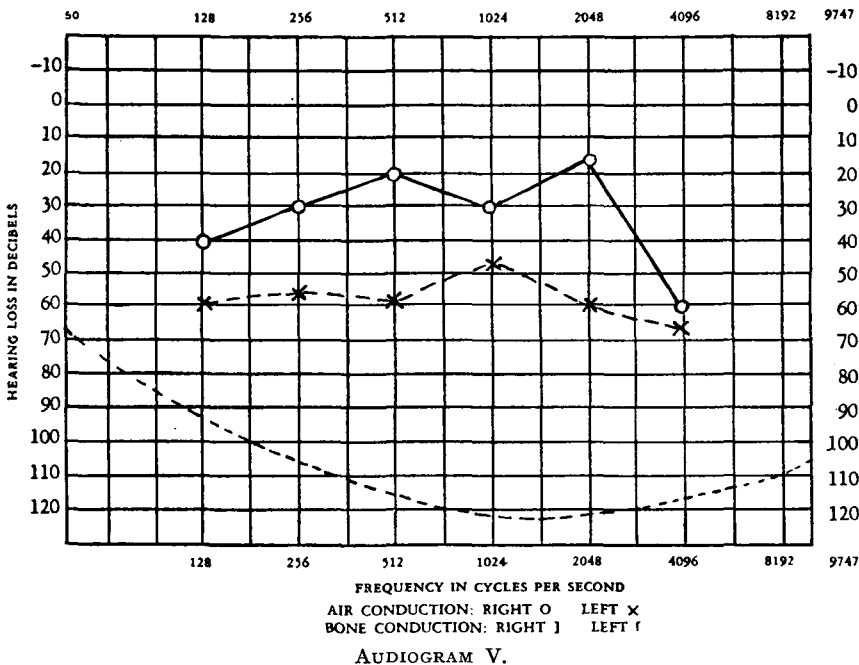


however, we now have a procedure which is entitled to equal consideration and indeed in most cases should be given preference, if only because of its comparatively minor character. Incidentally, this case was the first of the series to be completed in a technically satisfactory manner.

CASE XII. F. B., æt. 18, had had bilateral otitis media since early childhood. The left ear was almost useless. The right ear could hear conversation in a raised voice. Both drums were much thickened, so that outlines of the malleus were invisible. Both had a perforation in the pars flaccida, but the left drum, in addition, had

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a large postero-inferior perforation. In accordance with the principles outlined above, I explored the left mastoid, and found it extensively diseased. There were, in fact, no recognizable ossicles and a radical mastoidectomy was performed. The result was definitely bad. Despite careful after-treatment the operation cavity never completely healed, and the hearing if anything deteriorated. The position was rendered more desperate by the fact that a progressive deterioration of the right ear set in. Clearly if nothing was done, the boy's hearing was doomed quite apart from



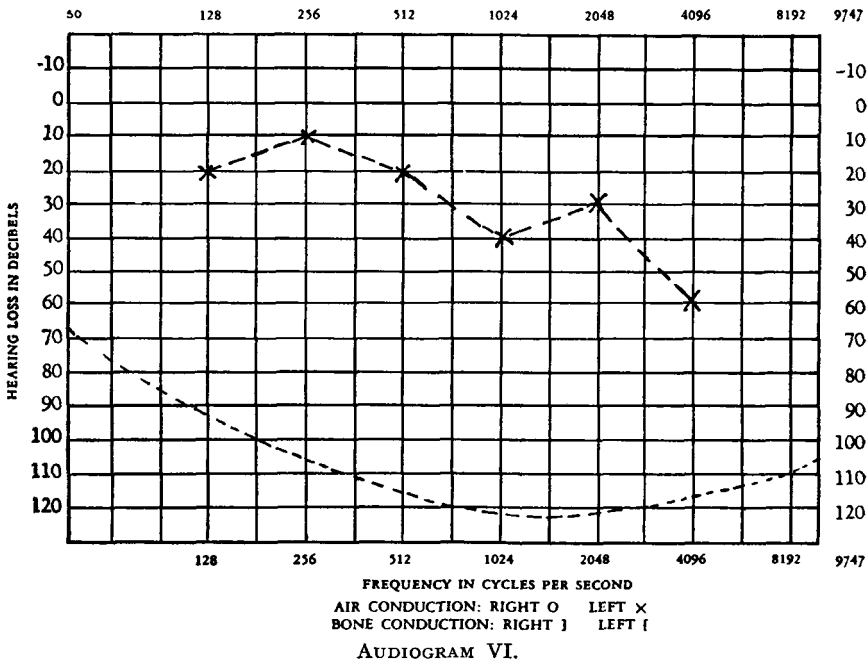
the possibility of intra-cranial complications. On the other hand, a major mastoid operation on the right ear, whilst dealing with the threat to life, might conceivably precipitate a profound deafness such as would otherwise not have developed for some time.

Under these circumstances a trans-meatal atticotomy was undertaken: it was felt that the delicacy and precision of the operation entitled one to expect no loss of hearing after it. This was found to be the case. After the post-operative œdema had subsided, the hearing was found to be no worse; if anything it was better. The otorrhœa diminished but little, even though the exposed attic region healed up quickly. The main source of the

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discharge was clearly the mastoid itself. The inner tympanic wall (as visualized over the upper edge of the drum) remained cedematous and congested. The operation was, however, far from a failure. The marked improvement in the condition of the tympano-attic structures enabled the writer to undertake a subsequent mastoid exploration in which those delicate structures were in no way disturbed.

The meatal plastic, for instance, was simplicity itself. A diseased tract of bone, running down to the apex, was dealt with,

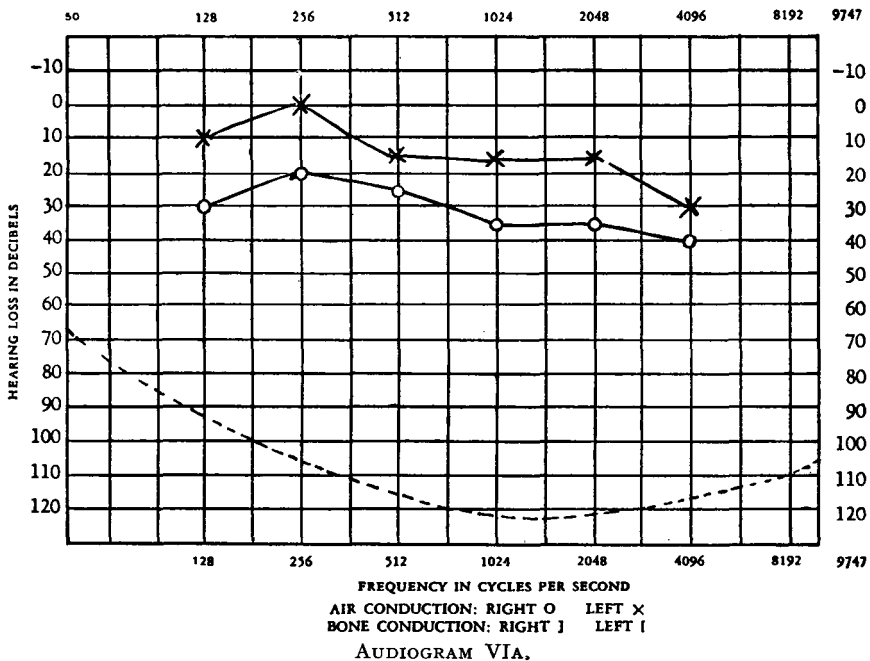


and the patient now declares that he can hear better than he can ever remember. (Audiogram V.) This, in the writer's opinion, is a particularly valuable function of trans-meatal atticotomy, namely as a preliminary procedure, undertaken because of the desperate need to save hearing, in cases in which the other ear is useless. Even should it fail to cure the bony disease, such an atticotomy does half the work so that a subsequent retro-auricular exploration can be undertaken without fear.

CASE XIII. Miss W. P. Complained of deafness and giddiness. Right drum intact but right ear deaf owing to progressive middle-ear

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catarrh. Left ear—otorrhœa about ten years. Typical posterior-superior perforation with polyp formation. Resisted conservative treatment. This problem was similar to Case XII, interference seemed urgent owing to the giddiness, but the patient could not leave her work for the two or three weeks required for a major mastoid operation. Trans-meatal atticotomy was performed on June 29th. The attic structures are now well exposed, and show no evidence of granulation tissue. The giddiness has disappeared completely. This patient is still under treatment and promises to be a complete success, although



she does not find any dramatic improvement in her hearing. Audiogram VI shows that her average hearing loss is only about 20 db., but that unfortunately she goes down 40 db. in the most important region, i.e. circa 1,000 d.v. Here again, if the otorrhœa should persist, a mastoid exploration can be undertaken without any interference with the drum-ossicle apparatus.*

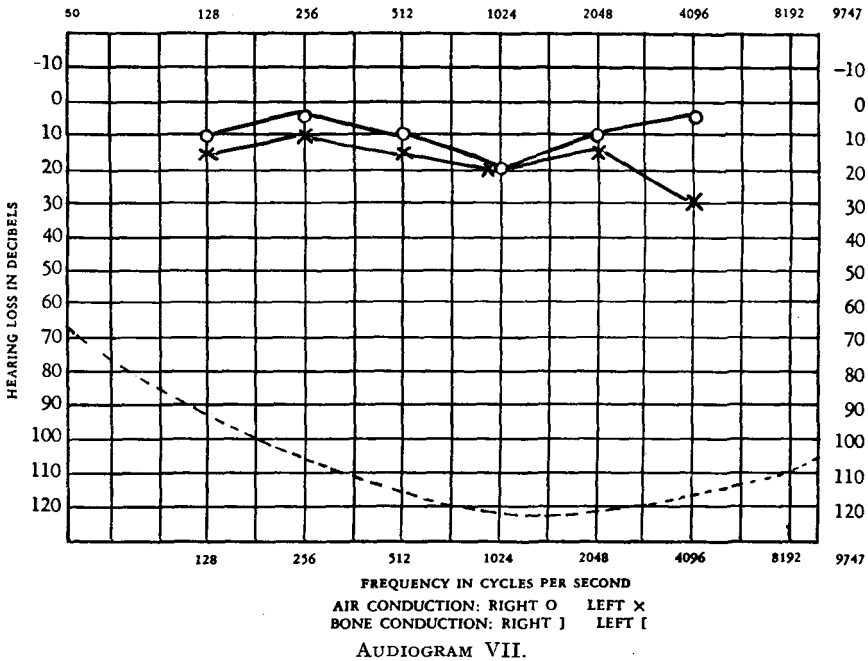
CASE XIV. William B., æt. 16. Left otorrhœa since childhood. Both mastoids "compact ivory bone". Left drum shows both

* Three weeks after the above was written the ear dried up completely. The patient can hear much better, as is shown by the later Audiogram VIA.

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central and superior perforations, trans-meatal atticotomy followed by a distinct improvement in hearing. Operation cavity practically dry within a few weeks. (Audiogram VII.)

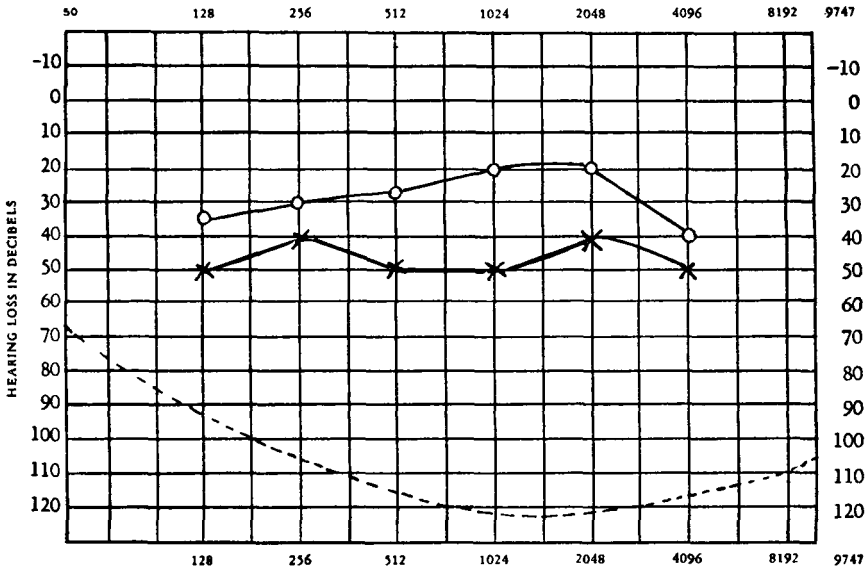
CASE XV. K. M. P., æt. 52, attended hospital because of chronic right otorrhœa of over twenty years' standing. Both mastoids were diploetic. Patient declared that the left ear had never given him any trouble and that his hearing was fairly good in it. Conservative treatment failed, and an atticotomy



was undertaken. The small operation cavity healed rapidly and a most gratifying improvement in hearing occurred. This was the more welcome in that, for no apparent reason, the left ear suddenly developed an acute epitympanitis complete with polyp. Prompt treatment resulted in a fairly rapid healing, but to the patient's (and my own) dismay, the hearing did not return proportionately. To-day, this man, a ship's captain, depends for his hearing on the right ear which he had regarded for many years as practically useless. (Audiogram VIII.)

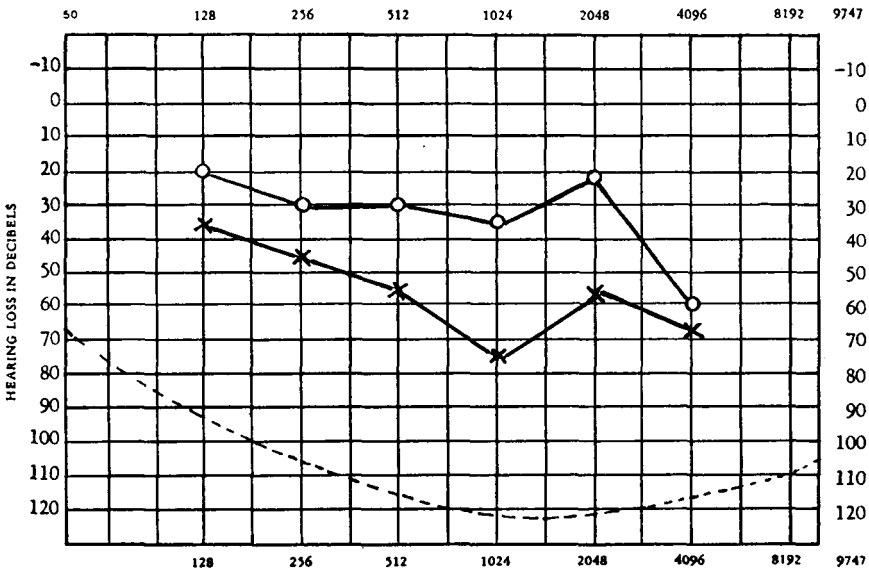
CASE XVI. (Audiogram No. IX.) This patient consulted me in great distress. His left ear had been deaf for nine

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FREQUENCY IN CYCLES PER SECOND
 AIR CONDUCTION: RIGHT O LEFT X
 BONE CONDUCTION: RIGHT] LEFT [

AUDIOGRAM VIII.



FREQUENCY IN CYCLES PER SECOND
 AIR CONDUCTION: RIGHT O LEFT X
 BONE CONDUCTION: RIGHT] LEFT [

AUDIOGRAM IX.

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years and showed a healed posterior-superior perforation. He could not recollect ever having had otorrhœa. The right ear began to go deaf about four years ago but did not "run" until after he had it syringed about eighteen months ago. Both mastoids are diploetic. The usual polyp was present. Conservative treatment over a period of several months failed to cure him, and finally operation was advised. The patient declared that he would lose his job if he was away for two or three weeks, and therefore an atticotomy was performed. This was rendered more difficult than usual by the fact that his external auditory meatus was much narrower than normal. He left hospital after three days, and resumed his work. Post-operative deafness lasted only four days, and now, although the ear is by no means dry, he thinks his hearing is better than before. The attic is well exposed and there is every prospect of curing this case.

To sum up. These nine cases had all resisted the most persistent conservative treatment for anything up to six months, and would normally be subjected to one or other of the major mastoid operations according to the personal preference of the operator. The operation described here was performed with practically no dislocation of the patient's daily life. Several were done as out-patients and none stayed in hospital more than three days. We may reject Cases VIII and IX, which were done with imperfect technique, although, even so, Case IX has improved functionally very considerably. Out of the remaining seven, one (Case XII) had to have a subsequent mastoid exploration. Here again the writer attributes the dramatic functional improvement to the fact that the operation was done in two stages. Of the remaining six, one (Case XIV) has been done only three weeks but shows every promise of complete healing. His hearing is already definitely improved. Three are quite healed and have much improved hearing. Two are still discharging and may require further surgery.

It seems justifiable to claim, therefore :—

(1) That trans-meatal atticotomy can cure certain cases of otorrhœa which have resisted the most persevering conservative treatment.

(2) The operation is a comparatively minor procedure.

(3) That, contrary to Shambaugh's dicta, it is not to be brushed aside as inadmissible, owing to excessive difficulties and hazards.

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(4) That, even when it fails, a subsequent mastoid operation will be found much easier than usual and more likely to achieve functional improvement.

It would be quite unjustifiable to draw final conclusions as to the value of this operation from such a small series of cases. Nevertheless, there are many noteworthy and instructive features. The earlier cases certainly suffered because of inadequate technique, but in skilled and experienced hands the writer is satisfied that trans-meatal atticotomy can always be completed satisfactorily without danger, or indeed excessive discomfort, to the patient. (So far, the more extensive operation of trans-meatal mastoidectomy has not been attempted.)

The ideal end result is a dry cavity (which does not form crusts) together with an intact drum ossicle mechanism and improved hearing. That this can be obtained in some cases is quite clear even from this small series. The functional results in such cases are likely to be much better than would be obtained from any other operation. It will naturally be asked—In what percentage of cases could such a result be obtained, and, as a corollary, how does the trans-meatal operation compare in that respect with trans-mastoid atticotomy? Further information must naturally be sought in a much larger series of cases; nevertheless, the mere comparison of groups of figures must not be allowed to loom too largely. The fallacies inherent in such a statistical approach are notorious, and the author prefers to base his advocacy of the trans-meatal operation on the following facts.

(1) It is a comparatively minor and quite safe operation.

(2) It enables the drum ossicle mechanism to be treated very carefully so that better functional results are to be expected.

(3) Even if it fails to produce a dry cavity, it makes a subsequent mastoid exploration much simpler.

For these reasons, the author has come to the conclusion that in chronic perforating epitympanitis, if conservative treatment has failed, the operation of choice is a preliminary trans-meatal atticotomy. The only reservations are that there should be no evidence of intra-cranial complications, and also—as is usually the case—the mastoid should be of the acellular variety.

The position may be put rather differently as follows, the

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author recommends, for the above type of chronic epitympanitis—a *two-stage operation*. The first stage being a trans-meatal atticotomy, which, in a considerable proportion of cases, will result in a complete cure. In those cases in which suppuration persists, the second stage must be undertaken, namely, a mastoid exploration.

ACKNOWLEDGEMENTS

The author is deeply indebted to Dr. P. Whitaker for his most valuable assistance in the radiological problems.

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Der Verfasser glaubt, dass die verschiedenen Typen des Cholesteatoms auf die gleiche Ursache zurückgehen und dass sie in einzelnen Formen des Warzenfortsatzes aus den normalen entzündlichen Reaktionen des Schleimhautnages im Epi-, Retro- und Prae-Tympanalraum entstehen. Diese entzündlichen Reaktionen gehen mit der Bildung von Schollen und Polypen einher. Die Annahme des Verfassers ist eine modifizierte Metaplasietheorie, die nach seiner Ansicht die verschiedenen Formen in einfacher und logischer Weise zusammenfasst.

L'auteur croit que les différentes formes de cholésteatome ont une même origine, et qu'elles dérivent des réactions inflammatoires normales de l'épithélium pavimenteux de la région épi, retro et prétympanique dans certaines formes de mastoïdes. Ces réactions inflammatoires sont associées avec la formation de squames et de polypes. L'auteur propose une modification dans la théorie de la métaplasie qui groupe les diverses entités d'une manière simple et logique.