ATTIC SUPPURATION

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SINCE few of us may claim a comprehensive or even a competent knowledge of attic suppuration, this choice of subject need cause no surprise. There is the implication that previous enquiry has been at best inconclusive, and such has been the impression of my reading. In consequence, though influenced by previous reading, the major part of this paper was first constructed from observation and experience, in order to see where I stood before turning directly to the literature. The terms of reference are narrow, yet it may be useful to begin with a study of lesions which are not now suppurating, and later to discuss things which do not appear to suppurate at all.

The surgical anatomy of the attic, in my experience, provides three spaces in which *limited* pathology or its consequences may be observed.

1. The small region anterior to the head and neck of the malleus.

2. The mid-region, of minor importance if only Prussak's space be involved, but otherwise if disease includes the superior lateral recess above. (Note that the flaccid membrane may vary greatly in size and shape—the posterior fold may be long and prominent, the anterior almost absent, or vice versa; and again one membrane may be tall and narrow, another broad and short.)

3. The narrow but high posterior recess external to the body of the incus.

All three regions may, of course, be involved simultaneously, and indeed the whole epitympanum through to the inner wall.

Inactive Lesions

In considering the evidence of past damage to the attic we must note that a positive history will usually depend on three factors: (*a*) rapid pressure changes of such degree as to cause pain, (*b*) a discharge sufficient in quantity as to reach the surface, (*c*) some impairment of function, ***which in childhood will seldom be noted in the absence of a bilateral lesion, with deafness severe enough to attract parental attention.***

Now routine examination reveals many examples of past damage wherein cure was clearly spontaneous, and neither patient nor parent can recollect previous advertised aural disease. Some of these perforations and erosions are well covered, some are bridged or lined out

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by thin epithelial sheets, some remain partly or wholly open. Many give rise to no subjective complaint and normal hearing is a common finding. To this group must be added an unknown quantity, arising from the observation that a lesion occurring just in front of the external process of the malleus may heal without discernible trace.

In some cases there is evidence of soft-tissue repair only, in others of an additional bony erosion. In the anterior space one may seldom be certain of past damage in the absence of an erosion ; in the mid-region the appearance of the epithelium alone is frequently conclusive ; whilst in the posterior attic a high upper segment scar may be enough to indicate previous adjacent epitympanic activity. These, then, are the visible scars of silent bygone battle—or ambush if you will.

Observation, therefore, suggests that the epitympanum is often the seat of some curiously insidious process tending to spontaneous arrest, a disease frequently unadvertised in the active phase, and it may be that in general we are only consulted in those cases which fail to limit themselves, those further infected, or those whose apparent onset is dramatized by a sudden acute otitis, this latter possibly an intercurrent thing. This insidious process must frequently belong to childhood, and I suspect that it is essentially a response to infection.

Active Lesions

Consider next that routine inspection makes it certain that visible active attic disease, especially when lying anterior to the neck of the malleus, may be physically and functionally symptomless. We know that this activity may be quite painless in onset, and I have heard a patient tell me that one month ago she suddenly became deaf in the left ear and lost taste in the same side of the tongue, a syndrome apparently due to an extensive attic cholesteatoma. Then there is the common story of the child whose " acute " otitis began say three days ago, the discharge being already fœtid. Beyond all this is the occasional affront to the pride of every experienced otologist at the finding of extensive cholesteatoma above an intact membrane which appeared not very much out of the ordinary. I recall a doctor, already qualified a few years, whose sole previous symptom was of right deafness known since the age of 15. Pre-operatively he showed only a red full upper membrane and, except for the meso-tympanum, the whole middle-ear cleft was later found to be packed with cholesteatoma.

Turn now to some of our past experiences. Until recent years large numbers of acute mastoid suppurations were treated surgically. The operation of simple cortical mastoidectomy was a good one and in the main gave encouraging results ; but certain reservations attached to those cases discharging by way of the attic, and especially suspect, were

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those presenting a mucosal herniation. A proportion of these membranes healed at once and remained so, and some continued to suppurate; others might heal quickly enough but the patients returned in a few weeks, a few months, or much longer, showing a blown-up wound but perhaps no otorrhœa—a lesion which often seemed to yield very well to simple re-drainage of the cavity. Such patients might return more than once, and then one day we found the mastoid cavity full of cholesteatoma. Occasionally, both attic and post-aural wound did well at once, and continued to be satisfactory through months of observation. Two, three, five years later, the patient returned with a painless fœtid otorrhœa from the same antero-superior site as was originally involved. The recent story might cover only a day or two, but already the tiny gap showed granulations and squames. By contrast with this type of case, there were far fewer relapses and delayed surprises in another large group presenting at first sight the much angrier picture of a big red pulsating posterior segment surmounted by a streaming nipple perforation but free of a hanging attic. It may be doubted whether a combination of tympanic and retro-adital drainage was always really effective in relation to the attic, and particularly its anterior space. Moreover, when dissecting finely cellular clefts, both in the cadaver and in fenestration cases, I have often noted bars of pneumatic bone almost shutting off various segments of the epitympanum, and these may be of considerable importance in the presence of mucosal swelling.

Occasionally one sees a case of negative pressure syndrome exhibiting a grossly indriven flaccid membrane long after the meso-tympanum has returned to the state of an air-containing cavity. Such conditions may exist without obvious evidence of a notable inflammatory factor, and the finding is not inconsistent with a normal-looking tense membrane. So it may be that in certain anatomies there is a much restricted communication between the meso-tympanum and the anterior and middle attic spaces, and that under unfavourable conditions, even air exchange, let alone drainage, is difficult.

Dormant Lesions

This rapid survey of silent healed lesions on the one hand, and active or insidious processes on the other, may be left in suspense while we look at what may be called the dormant group. Here the routine examination of an uncomplaining patient reveals a trace of moisture in front of the external process, or a small adherent crust whose removal uncovers a minute smear of sticky pus, or perhaps a small herniation here today and gone tomorrow, or even a clean dry seed of pearly cholesteatoma which some day we find to have burrowed a little cave of its own in the sloping anterior attic wall. It has been my experience that such of these

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lesions as are shallow yield frequently to minor topical therapy, and in those containing a readily accessible ball of cholesteatoma, a suction clearance has often given gratifying results, more reliable perhaps when healing was by a lining of epithelium rather than by a covering sheet.

And here comes a forthright interpolation, intended to be provocative.

An adequate study of these finer points, particularly in relation to the evidence of past disease, requires the use of a *good* electric auriscope, and I suspect that to attain the same accuracy of definition with the Siegle speculum demands artistic handling of such an order as to make the rest of aural surgery a mere trifle. I do not understand the inordinate resistance of some of my colleagues to an instrument so manifestly useful. I began my career with head-mirror and simple speculum, using also the Siegle instrument, and later added the electric auriscope. Now all three are used, and I have no prejudice in the matter.

But to return to my argument. So far, three hypotheses seem to emerge :

1. The attic spaces are relatively inaccessible.
2. Satisfactory arrest of inflammatory processes is common enough where drainage has been good and presumably not very prolonged.
3. There is a tissue-reaction in the epitympanic region suggestive of a peculiar instability.

All this, nevertheless, goes only so far and in hesitant fashion at that, and there remains a challenge to the instinct of curiosity, more questions unanswered than otherwise. It gives me no satisfaction to say that we recognize these things when active, because it does not follow that recognizable activity is the whole process either in extent or in time, or even that it is not merely a fortuitous addition. I feel certain that pathology may exist, and sometimes over a long period, which I cannot detect, and improvement in detection on scantier evidence is to me the real challenge.

In a recent paper on chronic otitis Daggett (1949) erroneously credits me with writing of chronicity " from the start ". I admit discussion of an insidious process, something which may be construed as chronicity when first seen, and it is this characteristic which is stressed now. Whether I accept chronicity from the start, or reject a few weeks as a definition of chronicity, is not germane to the present argument. I do suggest, however, that the problems of attic disease are not to be dismissed on the easy assumption that we are only required to treat manifest acute otitis " properly ", and I think this still holds despite the considerable alteration in the character of much of acute aural disease during the past ten or fifteen years.

In support of the points already submitted I would offer for consideration the common picture of a quiescent postero-superior perforation

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with cholesteatoma in the lateral recess above ; and also the everyday finding of a clearly chronic partly-closed attic disease in one ear alongside a lined-out attic erosion in the other, the latter being symptomless at all stages.

A brief review of two illustrative cases may be helpful. Ten years ago I saw a patient of 40 confined to bed with right earache. The anterior attic showed a red acute polyp or herniation. Eventually he submitted to a cortical mastoid operation which yielded complete healing and restoration of hearing. Five years later he returned complaining of some wet brown stuff from the same ear which now showed a few sheets of keratinizing epithelium in a moist epitympanic erosion amounting to a nice attico-aditostomy, through which the ossicles could be easily viewed. A year later he returned to discuss some reverberations in the opposite ear, with some plopping and moisture of about three months' duration. This left attic showed an anterior polyp with a moist pulsation, and I failed to control the process by topical treatment which included a suction clearance. After fifteen months of ups and downs, the ear sometimes moist and sometimes dry, the patient agreed to submit to a post-aural attico-antrostomy, which revealed a cholesteatoma, mainly dry, extending from the anterior attic to the aditus, with the head of the malleus and the incudal body separated by a sandwich of the mass. Removal of the incus and excision of the malleus head, assisted by a reverse tympano-meatal flap, yielded complete healing in three months.

In this case the right lesion seemed acute in onset and healing was objectively satisfactory after cortical operation. At some stage within the next five years a symptomless erosive process removed the whole outer attic wall. In the left ear, the disease was insidious in onset—and incidentally I would now almost insist on much earlier opening of the attic.

The second case concerns a lady of 50 who complained only of a year's deafness in the left ear which showed a dry anterior attic cholesteatoma. At operation this extended throughout the epitympanum and into the antrum. Complete epithelization ensued ten weeks after an endaural radical. After a year of routine inspection, she casually mentioned a little moisture from the right ear, and two weeks afterwards I was able to define a minute gap in Shrapnell's membrane. A small ball of cholesteatoma was delivered by suction clearance, and healing was not only rapid but so complete as to leave no discernible scar. This position has been maintained now for several months and the hearing is excellent.

Pathology

Genuinely acute attic pathology gives me no bothersome intellectual problem, physical inaccessibility being the prime difficulty, a factor of

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much less significance with modern blood-borne therapy provided that the management is early and adequate, and assuming that the organism is sensitive, as it usually is. My concept of subacute pathology in which tissue changes are presumed not to be irremediable, differs only in veering away from medical measures unless these show a clearly rapid and reliable effect, towards uncovering surgery which, even should it fail quickly to control discharge, at least prevents destructive ossicular erosion. The full-blown chronic lesion, especially when exacerbated, presents an obvious enough picture of irreversible mucosal disorganization and often of osteitis. I do not know either the microscopic or the macroscopic picture of those unadvertised progressive changes which hide behind a normal-looking fundus and which eventually present with some recognizable abnormality discovered accidentally or otherwise. Is this smouldering process always a residue of an acute infection, however undisturbing to the patient, and if so, is the silent stage a direct consequence of continued bacterial infection? I do not know, but one may recall a comment by Tumarkin (1938) concerning " the natural manifestations of varying degrees of infection *or other irritation* (italics mine) acting on the pavement epithelium— ", very nicely put but it really means he does not know either!

It is difficult to treat of our subject today without re-opening the question of cholesteatoma or cholesteatosis. It is obvious, of course, that this material, though generated most often from the epitympanum, is not exclusively so. On this score I agree with Dan McKenzie (1931) but must differ with his notion that " the material is concentrated in one quarter or region ". I need only recall a predominantly granulating pathology in which the sole cholesteatoma mass in the attic lay inside the œdematous mucosa lateral to the incudal body, but in which there were also several larger masses enclosed in granulations filling the mastoid segment; and again another case in which there were both dry and wet collections in large numbers of small cells, many of these cells *still retaining their bony septa*. Last November, I suggested that there was no essential difference between the predominantly granulating lesion on the one hand, and the chiefly cholesteatomatous on the other, since all grades may occur together (1949). I find myself with less and less doubt about the genesis of the clean dry sterile masses we sometimes see, despite vague uncertainties arising from contemplation of such things as lingual leucoplakia, laryngeal keratosis, and of some external meatal keratoses, which, by the way, may on occasion be associated with bony erosion without any demonstrable pressure factor. Almost against my will, the idea that there is no such thing as cholesteatoma has gradually formed, if by the word we mean some quite separate pathological entity. In short I approximate to the view of A. R. Tweedie (1931), namely, that cholesteatoma is merely one tissue reaction of the epitympanic

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lining to some past sepsis or irritant, and at the same time remind you of his neat phrase "a bottle-neck cavity". It is true that over the years I have entertained some wild theories, epidermoid and epidermal, primary and secondary, congenital and invasive, and a host of others, only to revert to the views of my predecessors and betters; but in so doing I have succeeded in becoming more catholic than Pope Tumarkin himself, by abolishing the cholesteatoma doctrine altogether. A further paraphrase of McKenzie's adaptation of William of Occam's logic might read: Why assume that there are two varieties of cholesteatoma when none will do? And yet the word or something like it must still be used for clinical purposes.

Conclusion

Though it has been my chief purpose this morning to treat of the general rather than the particular, there must be some attempt at drawing together these extremely tenuous strings.

The anatomical remoteness of the attic spaces must be considered important, and it seems clear that cellular metaplasia, in response to infection or irritation, may go as far as to form a reproductive keratinizing type.

Attic disease may be insidious in its development, and may be already an established chronic lesion when first manifest. On the other hand it may proceed to complete resolution without subjective complaint.

The detection of those cases in which resolution is apparent rather than real offers a challenge to our clinical acumen, and it must be recognized that enclosed ossicular erosion may proceed without much evidence.

Therapeutically, we must aim at complete cure in genuinely acute lesions, at the conservation of something useful in those middle ears not yet too far gone, and at the alleviation of further discomforts and risks in those beyond this stage.

Finally, it may be noted that nothing has been said of intracranial matters, but this is because I feel that middle fossa complications are not specifically related to the epitympanum alone; and if I am open to the accusation of evading the operative surgical issue, it is because something approaching my current view has already been publicized. Any one contribution to this discussion must have its limits.

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