The postero-superior quadrant*

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Cholesteatoma has left a legacy of intracranial complications, persistently discharging ears and deafness. Our main efforts have always been to control established disease, to save life, to produce healed dry ears and only latterly to restore function.

As with many other diseases, early diagnosis provides a better chance to eradicate disease and preserve function. Better still, would be to prevent the development of cholesteatoma formation by recognizing and eliminating the causes.

It is now generally accepted that the majority of cholesteatomata are due to a herniation inwards of parts of the drum into the middle-ear cleft to form a sac in which collects the keratotic debris of stratified squamous epithelium. One of the common sites of invasion is the postero-superior quadrant of the membrana tensa.

The postero-superior quadrant is bounded in front by the handle of malleus, and above by the posterior fold of the malleus adajcent to which lies the chorda tympani. Immediately deep to this quadrant lie the incudo-stapedial articulation and the oval window niche.

The blood supply of this portion of the tympanic membrane has a different architectural pattern but the histological structure is apparently similar to other parts of the membrana tensa.

The middle-ear cleft is aerated through the eustachian tube but within the middle ear itself the airways are narrowed particularly at the level of the umbo and promontory and the incudo-stapedial joint. Proctor (1964) has shown that ventilation of the epitympano-mastoid segment occurs in some patients mainly through the anterior and posterior isthmus which lie on either side of the long process of the incus. In the fully pneumatized mastoid there is free communication between air cell systems, whereas in the sclerotic mastoid with absent cells ventilation of the epitympano-antral segment is via the two narrow isthmuses. The poorly pneumatized mastoid approximates to the sclerotic state in that although air cell systems have developed, free communication between groups of cells does not always exist.

For a long time cholesteatomatous ear disease has been associated with a poorly pneumatized mastoid. The explanation probably lies in the

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behaviour of Shrapnell's membrane or a thinned postero-superior quadrant under the influence of negative pressure changes developing in the epitympano-mastoid segment when the normal channels of aeration are obstructed over long periods. The fewer the aeration channels the more readily they become obstructed by oedematous mucous membrane, viscous or gelled mucoid exudate, or adhesions—the products of inflammation.

The stress is on inflammation and not infection. The mucous membrane of the middle-ear clefts seems to function only satisfactorily in air, as does the endothelium lining the bony air cavity of the fowl's humerus (Beaumont 1966). In the infant absence of aeration leads to failure of pneumatization, and at all ages produces an inflammatory response. The exudate which is a product of the inflammatory response will of course be sterile. If secondarily infected then organisms may be cultured. Therefore, I would suggest that although the initial cause of restricted ventilation of the tympanum may be an infective lesion of the upper respiratory tract, the inflammatory response in the middle ear may be non-infective and due to the physical trauma of absence of air, and the mucous membrane is only likely to recover if completely re-aerated and only then if it has not sustained irreparable damage.

The stigmata of exudative otitis are collapse of Shrapnell's membrane and thinning of the postero-superior quadrant. The pathogenesis of attic cholesteatoma through the stage of collapse and pouch formation has long been understood, but a similar grasp of the pathogenesis of postero-superior cholesteatoma through the stages of thinning, collapse, pouch formation and herniation is more recent.

The development of a hearing loss in attic cholesteatoma tends to be late in the natural history of the disease for the sac has to be fairly extensive to damage the ossicular chain. On the other hand in postero-superior quadrant cholesteatomata a hearing loss develops early for the long process of the incus and superstructure of the stapes are adjacent and vulnerable. Indeed, they are frequently destroyed by the aetiological exudative otitis before a true pouch has developed.

The postero-superior quadrant of the drum is not alone in being subject to thinning by loss of its lamina propria, but it seems to be the most frequent site. Other areas of the drum may undergo thinning and the trauma of frequently inserting grommets is a contributory factor. Surgical trauma, however, cannot be regarded as the usual cause of postero-superior quadrant thinning.

Loss of the lamina propria is not usually seen in the early stages of exudative otitis; it usually becomes apparent in recurrent or long-standing cases. The pathological mechanism is ill-understood but thinning occurs above the narrowing of the intratympanic airway at the promontorial level. Obstruction of this channel causes loculation and persistence of inflamma-

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tion in the oval window niche with subsequent thinning and ultimate loss of the overlying lamina propria and destruction of the long process of the incus and superstructure of the stapes to produce a permanent loss of hearing. The anterior part of the tympanum may become re-aerated if eustachian tube function recovers and yet the remainder of the cleft beyond the obstruction may fail to ventilate.

More commonly reventilation of the middle ear either naturally or by insertion of a grommet empties the loculus but at a subsequent occasion a recurrence of obstruction at the narrowed and perhaps increasingly narrowed intratympanic airway causes the thin quadrant to collapse on to and drape the incudo-stapedial joint.

Occasionally instead of collapse one sees ballooning of the thinned postero-superior quadrant in the natural state. Presumably this results from an air lock.

At the stage of thinning and early collapse of the postero-superior quadrant, the most important factor in treatment is to re-establish and maintain adequate aeration of the tympanum. Provided adhesions have not occured to the long process of the incus and promontory the drum may regain its tension and function normally. Tympanosclerotic patches may even be deposited in the thinned lamina propria without interference with function but they may be the only indication that tympanosclerotic plaques are also being formed in the oval window niche.

With ossicular destruction recovery of aeration may lead to the membrane regaining tension but now there is a permanent loss of hearing. The hearing loss may be slight and negligible if the membrane heals down to the head of an intact stapes. Surgery may be needed to restore hearing by ossicular reconstruction but should not be attempted until one is absolutely sure that satisfactory aeration has been maintained for at least one to two years, particularly in a child.

Tympanomanometry is useful at this stage to distinguish between the excessive mobility of ossicular loss and the fixed chain of tympanosclerosis.

Persistence of negative pressure in the epitympano-mastoid segment together with the development of adhesions may cause the thinned collapsed area to form a pouch. Desquamation of the surface layers of the drum is now important for although the surface of the thinned collapsed membrane shows surface desquamation, as this does not accumulate, little harm results. However, once a pouch forms the accumulation of keratotic debris within the pouch, which by definition has a narrow neck, converts the retraction pouch into a pulsion pouch so increasing its hernation within the middle-ear cleft.

The pouch may herniate upwards towards the epitympano-mastoid segment on either side of the long process of the incus or deep to the body of the incus if the long process and stapes have been destroyed either by

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osteitis or the invading cholesteatoma matrix. Occasionally it herniates between the incudal body and the chorda tympani.

Sometimes the pouch extends mainly backwards and medially into the sinus tympani, downwards towards the hypotympanum, and forwards deep to the handle of the malleus into the anterior tympanum and eustachian tube. Occasionally all these routes are involved in an individual patient producing a purely tympanic cholesteatoma. (Fig. 1.)

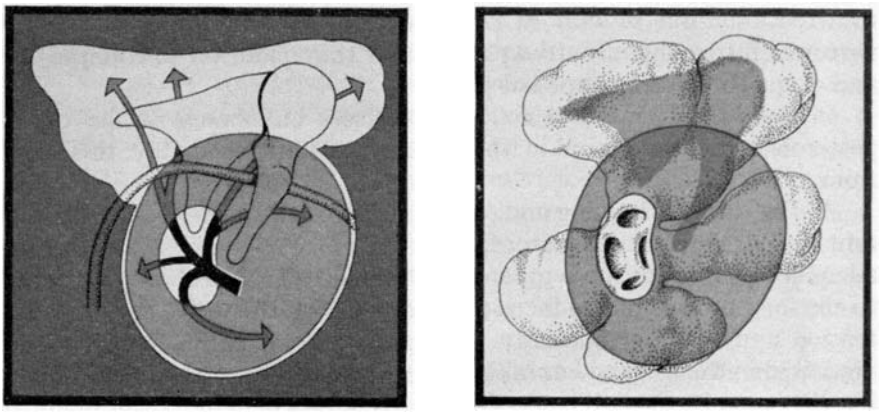


FIG. 1.
a) Directions of extension of a postero-superior pouch.
b) Type of cholesteatoma that is sometimes formed.

The degree of pouching and collection of keratotic debris within the pouch varies from patient to patient and each case must be carefully assessed as to future management. Increased production of keratotic debris may be stimulated by surface wetting or the presence of a serous or catarrhal otitis.

The thin collapsed membrane may or may not be adherent to the medial wall. If not, under anaesthetic this quadrant may be ballooned out by diffusion of gases into the middle ear, or it may even be sucked out by direct aspiration with a suction tube. The surface of the ballooned-out segment shows cracking of surface keratin and desquamation; and frequently a serous or catarrhal otitis is present. The aspiration of exudate and maintenance of aeration by a grommet may allow a full recovery. If it is not blown out and has to be sucked out the outcome is usually less satisfactory. However, maintenance of tympanic ventilation either spontaneously or by grommet may permit the tympanic membrane to regain normal tension and function even though thinned.

Cutting off the pouch to leave a large gap does not necessarily prevent healing of the perforation and re-formation of a pouch. A permanent perforation may be produced and would be preferable to a pouch, for ventila-

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tion of the middle ear is immediately better, the mucosa may recover and at a later date a surgical repair can be undertaken.

If the pouch is not blown out or cannot be sucked out it is adherent to the medial wall. Surprisingly if the pouch has a wide neck the appearance resembles that of a simple perforation, particularly so if the surface epithelium retains its migratory potential and becomes self-cleansing. However, if the pouch is narrow-necked or extends deep into the sinus tympani keratotic debris accumulates and once infected causes an osteitis of the bony annulus to produce granulations and polypi. Occasionally the bone of the annulus is eroded so widening the neck of the pouch and the keratotic debris is spontaneously extruded.

Removal of granulation tissue and polypi and aspiration under anaesthetic may control the lesion temporarily but it frequently recurs particularly with an adherent pouch and surgical removal offers the best solution.

The sinus tympani has a superficial and deep portion, the two being separated by the facial canal and pyramid (Fig. 2). The superficial part immediately deep to the annulus is the facial recess; the deeper portion communicates with the hypotympanum below and runs up the posterior wall of the tympanum to end blindly behind the stapes. The sinus tympani may communicate with the mastoid air cells in the well-pneumatized bone but in the relatively poorly-pneumatized, the type usually associated with cholesteatoma formation, the sinus tympani is separated from the mastoid air cells by a solid block of acellular bone and tends to be relatively shallower. However, its depth is variable and its extends backwards and medial to the labyrinthine block.

Provided the sac is limited to the sinus tympani and accessible parts of the tympanum, it can be dissected out through an endaural or tympanotomy approach. To reach the depths of the sinus tympani it is necessary to elevate the annulus and perhaps remove bone external to the facial canal (Fig. 3). The only time one is sure that the whole sac has been rémoved is when it is possible to elevate it in one continuous intact sheet. This is easier if the superstructure of the stapes has been destroyed but can be extremely difficult if the stapedius and stapes are intact. Once the sac is cleared, Silastic sheeting should be placed over the bared bone and the drum repaired. The bone removal required is little more than that needed for stapedectomy and therefore loss of bone does not jeopardize the drum repair. Failure to remove the whole sac may lead to early breakdown of the graft, or if the graft takes initially an enclosed cholesteatoma may be produced to cause a late failure. Whenever there is any doubt about sac removal, ossicular repair should be left to a later stage, and it is wise to forewarn the patient prior to surgery that more than one stage repair may be needed.

A self-cleansing wide-necked pouch is usually associated with a hearing

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FIG. 2.
Drawings of thin horizontal cuts across the tympanum to illustrate the sinus tympani at different levels.
a) above the stapes.
b) at the level of the stapes.
c) at the level of the round window

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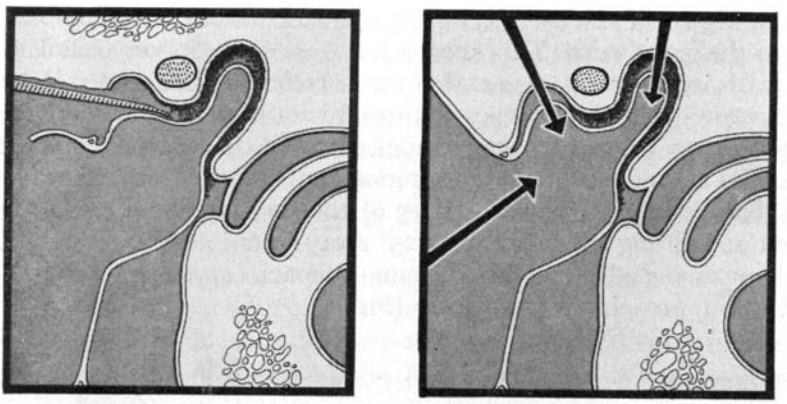


FIG. 3.
Dissection of a pouch in the sinus tympani by the transmeatal route.

FIG. 4.
Diagram showing the approaches to the sinus tympani; *viz.* the facial sinus approach as used in Combined Approach Tympanoplasty, the route deep to the facial nerve and the anterior approach.

loss and surgical repair of the membrane and ossicular reconstruction may be indicated. This is usually best done as a two-stage procedure. Seeing an apparent simple perforation the temptation is to do a simple onlay graft. In a normal central perforation the squamous epithlium usually extends for 1–2 mm. on the medial surface and is readily removed when freshening the edges by removal of 2 mm. at the edge of the perforation but one must be sure that a pouch is not overlooked particularly in postero-superior lesions.

In many of the postero-superior perforations it would seem that the suppurative otitis media which destroyed the drum occurred in a patient who had previously developed a pouch. Stratified squamous epithelium is frequently seen to extend on to the deep surface of the malleus and the medial surface of the anterior part of the drum, the chorda tympani seems closely wrapped by it, and fragments of squamous epithelium are also found in the sinus tympani, hypotympanum and promontory. I now take the view that in all postero-superior quadrant perforations the annulus must be elevated and the tympanum carefully inspected before placing an onlay graft. Even if an inlay graft is used it is still essential to look for and remove any remains of a pouch which may be present. Any ossicular repair required is better left to a second stage.

Pouches of this type are frequently associated with cholesteatoma or cholesterol granulomatous types of lesion in the epitympano-mastoid and of course this must be treated at the same time.

It is worth while considering the approaches to the sinus tympani (Fig. 4). The combined-approach tympanoplasty opens up the facial recess only

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but the depths of the sinus tympani could be reached from the mastoid deep to the facial canal. This approach is only of use in very cellular mastoids with extensive disease and is not a recommended route. However, the anterior approach offers possibilities. Victor Goodhill reminds us that a microscope can be used through a range of 270° if the surgeon is prepared to move. Furthermore he uses an anterior approach for stapedectomy and finds that he gets an excellent view of the sinus tympani by facing the patient and tilting the table up to 25° away from him. It is worth noting that he uses angled eyepieces. Such an approach can be readily used with combined-approach tympanoplasty (Fig. 5).

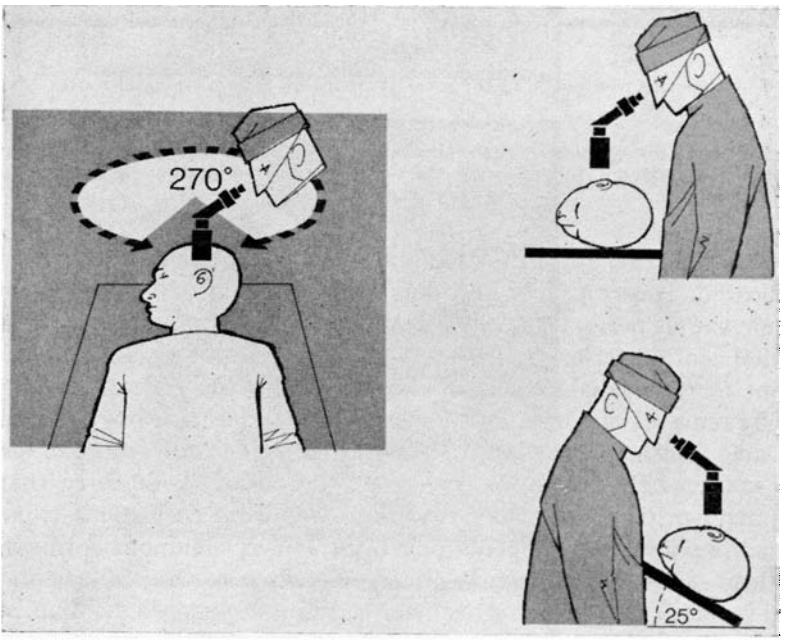


FIG. 5.

The anterior approach to the sinus tympani as advocated by Victor Goodhill.

Having talked about the pathogenesis of cholesteatoma it may be necessary to keep all cases of serous and catarrhal otitis under prolonged surveillance or we must pick out those at risk for careful follow-up and timely surgical intervention. Perhaps some of the patients at risk are those with poorly pneumatized mastoids, persistent recurrence or prolonged attacks of serous otitis and those with thinning of postero-superior quadrants and attic erosions. However, some will escape the net and between visits will develop a cholesteatoma.

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Finally, having argued that many cholesteatomata follow pouch formations I wish to illustrate metaplasia in the middle-ear mucosa (Fig. 6). Progress from columnar epithelium to cholesteatoma or vice versa can be seen. These findings emphasize the potential of the basal cells of the mucous membrane; they can develop into ciliated epithelium or stratified squamous epithelium according to environment and stimulus.

Professor Rannie comments on the cholesteatoma removed from the epitympanum as follows:

The material shows in one histological section through the specimen a transition from fully cornified stratified keratinizing squamous epithelium with entrapped desquamated squames (Fig. 6a) to non-keratinizing transitional epithelium. At one end the keratinizing epithelium overlies the

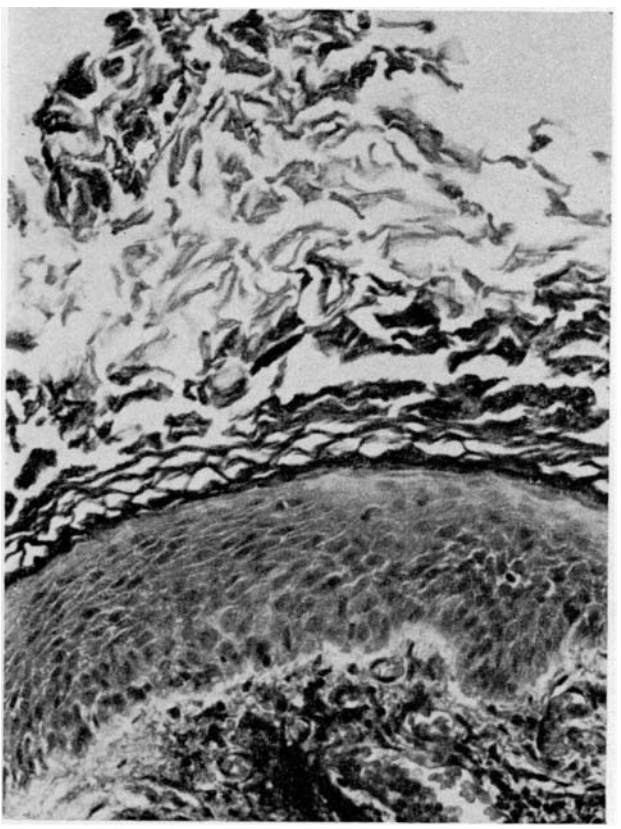


FIG. 6a.

Wall of cholesteatomatous sac showing keratinizing squamous epithelium with trapped desquamated squames.

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remains of the malleus which shows evidence of resorption, and this epithelium is in direct continuity with stratified squamous epithelium with a prominent granular layer, which is overlying chronically inflamed granula-



FIG. 6b.

Keratinizing squamous epithelium with pronounced granular layer. Note plasma cell infiltrated in underlying tissue.

tion tissue with intense infiltration by plasma cells (Fig. 6b). This in turn is in continuity with a transitional type non-keratinising epithelium covering chronically inflamed granulation tissue, again densely infiltrated by plasma cells.

This transitional epithelium varies from one or two cells in thickness to six to seven cells (Fig. 6c) and eventually shows typical ciliated epithelium of upper respiratory type (Fig. 6d).

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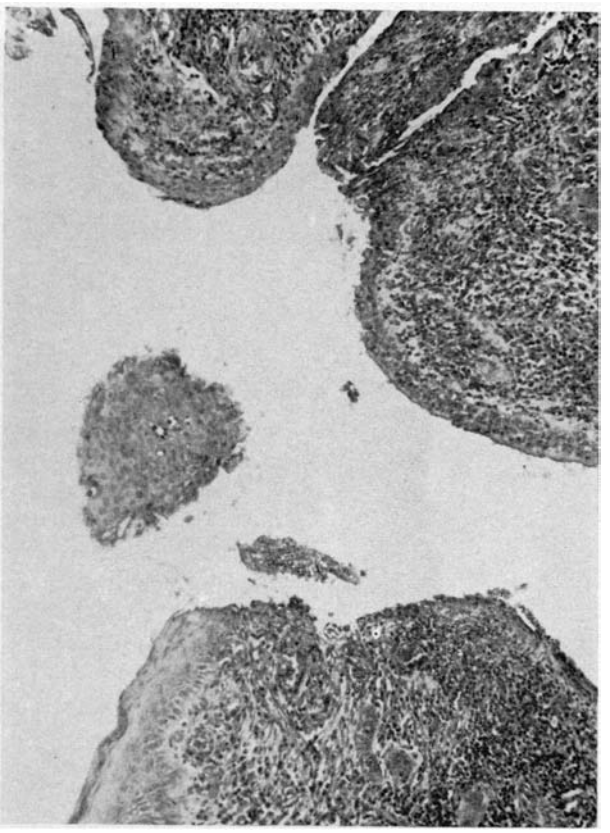


FIG. 6c.

Transition from keratinizing squamous epithelium as seen in Figure 6b to transitional type epithelium covering chronically inflamed granulation tissue with dense infiltrate containing many plasma cells.

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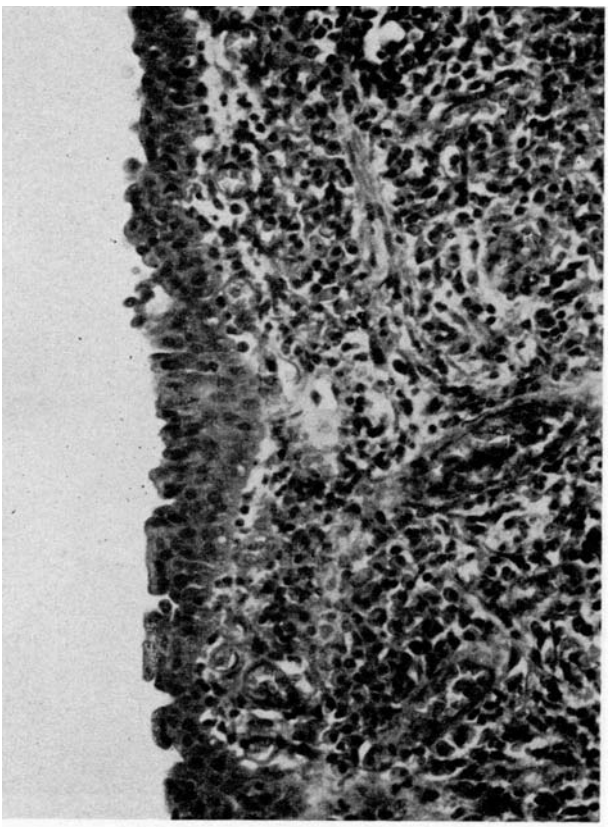


FIG. 6d.

Transitional type epithelium in continuity with that in Figure 6c covering chronically inflamed granulation tissue.

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