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Ossicular fixation*

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THIS title was chosen as the subject is essentially practical. Much has already been said and written about ossicular fixation in its varied forms and therefore this address is only a selective summary of personal experiences without the labour of collecting, bringing up to date and presenting a mass of statistics. That is not to decry the importance of evaluating results but my interest has always been the cause of failure or in the technical solution of the problems one meets in the individual patient.

Ossicular fixation may occur as a congenital anomaly, follow trauma, be associated with acute and chronic inflammation and its sequelae, and be seen in hereditary bone disorders such as otosclerosis, Van der Hoeve syndrome, and Paget's disease. Obviously swollen mucous membrane, exudate, masses of granulation tissue and cholesteatoma, the products of inflammation, may interfere with ossicular movement but they are best treated according to the needs of the disease itself. The only two inflammatory sequelae of interest are bony ankylosis and tympanosclerosis.

In congenital ear abnormalities bony ankylosis of a deformed or virtually normal ossicle to the walls of the epitympanum or the oval window commonly occurs. Apart from the gross facial, pinnal and general deformities with which they are associated they may occur as isolated abnormalities and only the age of the patient, the length of history, a minor pinnal abnormality, an abnormally shaped external canal or the absence of the malleus handle recognized on otoscopy may indicate that a congenital fixation exists but it does not indicate the extent or site of fixation. An absent malleus handle has been recognized on three occasions during the past thirty-five years and in none of these cases was there a significant hearing loss.

Fractures of the squamous or petrous temporal bone may displace bony fragments so that they interfere with ossicular movement (Fig. 1) and if left the fixation becomes permanent because of fibrous or bony ankylosis. If recognized early surgical relief may be obtained by elevating or removing the fracture fragments. At a later date the ankylosis is dealt with according to its needs.

After acute otitis media the head of the malleus and body of the incus separately or together may become ankylosed to the walls of the epitympanum. The extent of the bony ankylosis is variable but may only

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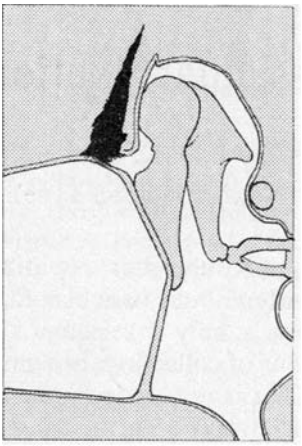


FIG. 1

Diagram to illustrate how a displaced fracture of the outer attic wall may fix the ossicular chain.

involve the suspensory ligaments or mucosal folds. The size of the epitympanum relative to that of the ossicles varies enormously and is a well recognized surgical anatomical feature. Surprisingly often the clearance between the ossicular mass and the epitympanic walls, in particular the outer attic wall, is minimal even when a mobile chain is present. The attic space itself may be capacious in places but in others it is narrowed by mucosal folds, ligaments, bony spicules, and the openings of cells almost abut against the ossicular chain itself. In such circumstances, often but not always associated with a radiologically acellular or sclerotic mastoid, a localized osteitis accompanying an otitis media may lead directly to bony ankylosis during the healing period.

Paget's disease (osteitis deformans) may fix the stapes but more often crowds the ossicles in the epitympanum, so limiting movement. Frequently this ossicular fixation is later accompanied by cochlear damage due to osteitis deformans in the petrous bone itself. As the disease is progressive surgical intervention is of only temporary value.

Otosclerosis is a well recognized clinical and histopathological entity involving the otic capsule which leads to fixation of the footplate of the stapes. Sometimes it is accompanied by ossicular bony fixation in the attic, possibly due to another cause or 'disuse' for there is no convincing evidence that otosclerosis occurs in the malleus or incus.

The Van der Hoeve syndrome (osteogenesis imperfecta) may be accompanied by stapes fixation, in practice similar to that of otosclerosis.

Tympanosclerosis (a well documented sequela of otitis media) fixes the ossicular chain but may be a histopathological entity quite distinct from bony ankylosis and is readily recognized during surgical exploration of the ear. Simply stated it is a hyaline degeneration of scar tissue which occurs in

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the submucosa, in the ossicular ligaments, and in the lamina propria of the tympanic membrane. The hyaline degeneration may undergo calcification and be reorganized into lamellar or haversian bone but there are nearly always areas of hyaline degenerative disease which have not become so organized. Surgically the tympanosclerosis appears to be white, avascular and laid down in sheets within the submucosal layer, even within the matrix of a cholesteatoma, or it is a degenerative change within the tendons of the tympanic muscles and sometimes is seen as blocks within the tympanic airways between mucosal folds. Commonly such blocks are seen filling the epitympanum and oval window niche. Rarely do they occur in the round window region and the Eustachian tube orifice. The blocks tend to occur in *cul de sacs* and may have a different origin from, although frequently accompanied by, submucosal sheets. Perhaps the calcium salts are deposited in organized gelled mucus or perhaps they are directly attached to the mucopolysaccharides of the mucus, so by-passing the stage of organization and hyalinization. Clinically the blocks and sheets seem to have a different stratification which suggests a different method of formation. Occasionally the whole ossicular chain is embedded in a tympanosclerotic mass.

It is frequently asked, does tympanosclerosis re-form, although many regard it as the end result of disease. However, at surgical exploration hyaline degenerative changes and bony reorganization are seen at different stages of development and later progressive degenerative changes may occur in regions not showing recognizable advanced changes at the time of surgery. Therefore surgical removal does not mean that all the damaged mucosa has been removed, nor do we know whether surgical intervention itself which damages the tissues may encourage degenerative changes to recur over a period of years. Certainly the surgical results in many cases of extensive tympanosclerosis are disappointing. It is not surprising that tympanosclerosis occurs in areas repaired by fascial grafts and in homograft tympanic membranes for it is usually found in damaged or dead tissue. Tympanosclerosis may require an entirely different solution to bony ankylosis.

Patients with hereditary bone disorders and congenital abnormalities without atresia of the meatus have intact tympanic membranes. Most traumatic perforations of the drum associated with temporal bone fractures heal spontaneously. Both bony ankylosis and tympanosclerosis may occur behind intact drums and therefore patients with ossicular fixation present with an air-bone gap and a flat tympanogram. If there is a wide air-bone gap of 60 decibels or more then a congenital lesion may be suspected. The presence of a chalk patch may suggest that tympanosclerosis is the cause of ossicular fixation but it is no more than a clue. The cause, extent and site of the lesion can only be determined by surgical exploration, usually a tympanotomy with or without an endaural incision depending upon the amount of exposure required.

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Tympanosclerosis and bony ankylosis are often associated with a perforated tympanic membrane, for they are both sequelae of otitis media and frequently a history of recurring otitis with a permanent perforation is obtained. A tympanogram is no longer of value. If the patient has an air-bone gap of less than 25 decibels the chain is usually not fixed. If it is between 30–40 decibels, about 75 per cent have a fixed or deficient chain, so these patients must be warned that more than one operation may be required to improve the hearing. At the first stage the tympanic membrane is closed and only if freeing or reconstruction of the ossicular chain is easy is the operation completed as a one-stage procedure. If the attic needs to be widely exposed, or if a large amount of bone needs to be removed postero-superiorly to gain access for ossicular reconstruction, then the risk of graft failure in tympanic membrane repair is increased. Therefore the operation is staged, a myringoplasty first and then nine months to one year later the ear is re-explored to tackle the problem of ossicular fixation as in the case of intact drums. Closure of the perforation in many cases when ossicular fixation is present leads to a widening of the air-bone gap.

Once the middle ear cleft is closed the problems of ossicular fixation are similar and for the sake of convenience are treated in an anatomical sequence.

Fixation of the malleus

Fixation of the malleus handle may be produced by tympanosclerosis of the drum or of the tensor tympani, and by adhesions of the umbo to the promontory. An isolated case of tympanosclerosis of the tensor tympani has never been seen; it has usually been associated with more widespread disease. The malleus handle may be fixed to the anterior sulcus by tympanosclerosis which has infiltrated the annulus tympanicus and sometimes has become organized as bone (Fig. 2). This is often discovered at myringoplasty and the tympanosclerotic plaque is removed, perhaps without damaging the underlying mucous membrane, so freeing the malleus and a simple repair of the tympanic membrane gives a satisfactory hearing result. Fixation of the umbo to the promontory may be by mucous membrane adhesions sometimes infiltrated by tympanosclerosis. Surgical separation of the umbo from the promontory is maintained by placing a silastic sheet in the middle ear and a formal myringoplasty is done.

Fixation of the head of the malleus is usually due to bony ankylosis or tympanosclerosis. Bony ankylosis may be limited to involvement of the suspensory ligaments or it may be a massive fixation of the head of malleus and body of the incus to the attic walls. Tympanosclerosis by involving the ligaments may partially or totally fix the chain in the attic and occasionally masses of chalk fill the attic and embed the ossicular mass. Ankylosis of the malleo-incudal joint alone does not interfere with the axis of rotation of the ossicular chain and therefore causes no deafness.

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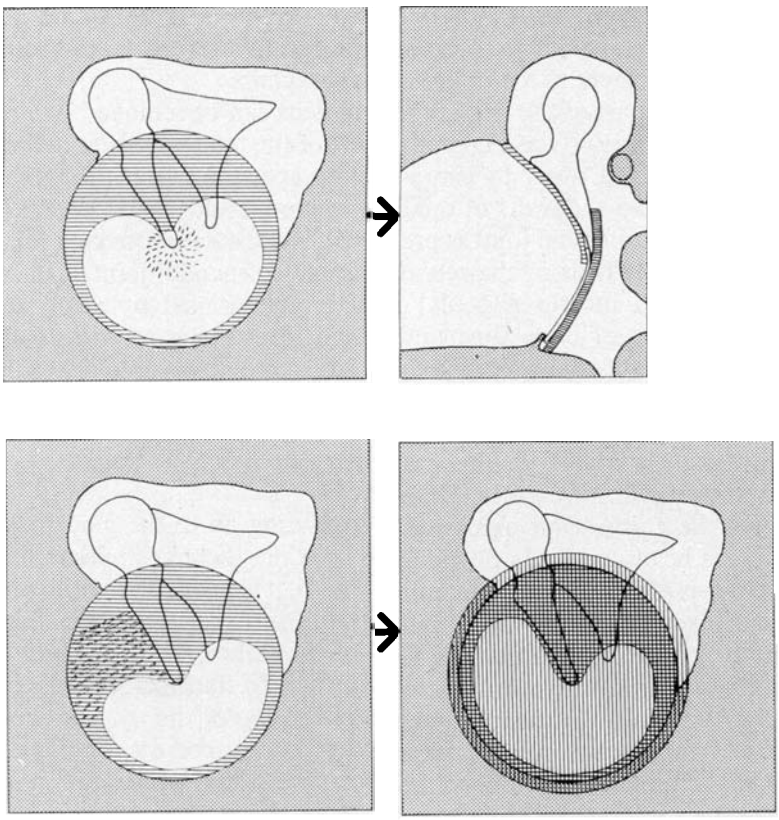


FIG. 2

Fixation of the malleus handle.
Above: Fixation of the umbo solved by myringoplasty and silastic sheeting.
Below: Fixation of the handle by tympanosclerosis of the anterior part of the tympanic membrane.

Ankylosis by involvement of the spheno-mandibular ligament may be of congenital origin as this ligament is the remains of the first branchial arch and if it is not fully differentiated may lead to congenital fixation. Infrequently incomplete differentiation is seen as the ligamentum folianus (Fig. 3), which is a bony spicule extending from the anterior spine of the malleus head to the anterior tympanic fissure within the spheno-mandibular ligament. Whether the ankylosis is congenital, or acquired and merely involves the ligaments, the ossicular chain can be freed through a limited atticotomy (Fig. 4). However, the attic should be enlarged anteriorly and superiorly so that a wide enough space is created between the ossicles and the walls of the tympanum.

In the small celled or sclerotic mastoid the epitympanum may be very small and it becomes impossible to enlarge the attic itself, so the ossicular

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mass must be reduced. It is most important before drilling is commenced that the incudo-stapedial joint be subluxated to prevent transmission of ossicular displacement to a mobile stapes footplate.

The anterior part of the head of the malleus can be removed leaving the malleo-incudal joint intact. The upper part of the malleus head and incudal body can be drilled away to leave enough space between the remaining ossicular mass and the walls of the epitympanum and, provided the lower cog of the malleo-incudal joint is preserved, a satisfactory locking joint can be obtained. Ankylosis of the remaining malleo-incudal joint surface and the reconstituted incudo-stapedial joint is encouraged by using a bone paste—a mixture of bone dust and blood. Preserving an attic ossicular mass with an intact chain seems a more satisfactory physiological solution than transposing an incus.

All bone dust created by drilling should be carefully washed out prior to reconstitution of the chain in case it gives rise to a source of osteogenesis and ankylosis at undesirable sites.

When the ankylosis is extensive or extending on to the medial side of the malleus head and body of the incus or when there are masses of tympanosclerosis filling the epitympanum (Fig. 5), it is probably best to remove the incus, excise the head of the malleus and transpose the incus between the handle of the malleus and the head of the stapes. This is best done by using the two drill hole technique of Marquet so that fixation is accurate (Fig. 6). Anatomical variations in the relationship of the pyramid and the posterior tympanic wall to the stapes may prevent correct fixation to the stapes head and therefore it becomes necessary to 'key' a piece of modified

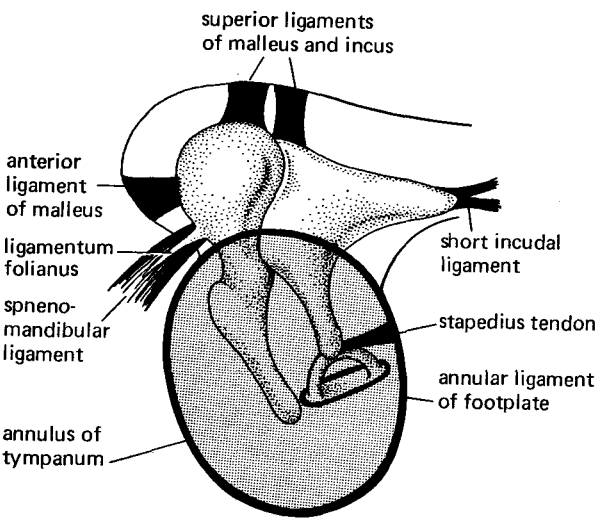


FIG. 3

The suspensory ligaments of the ossicular chain. Note the position of the ligamentum folianus.

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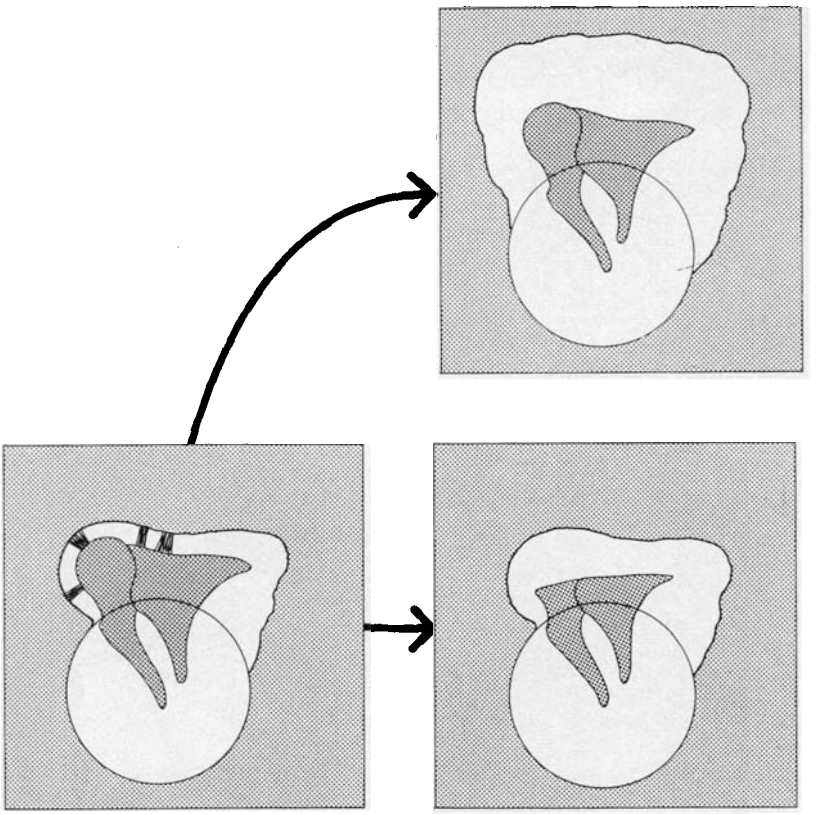


FIG. 4

Fixation of the head of malleus.
Above: Solution by enlargement of the epitympanum.
Below: Solution by reduction of the ossicular mass.

bone between the handle of the malleus and the head of the stapes (Fig. 7). Simply grooving the body of the incus for the stapes head and placing the short process on the handle of the malleus is unsatisfactory as the drum often becomes attached to this transposed ossicle and, as the drumhead regains its tension, elevates it from the head of the stapes so that only a fibrous non-functional union remains (Fig. 8). Therefore what appears to be a success is often only a short-term result.

When the oval window niche is deep and the head of stapes barely projects beyond the level of the facial canal, a modified incus preserving the lenticular process can be attached to the malleus handle by a deep groove or hole and the lenticular process position maintained by sterispon and bone paste (Fig. 9).

Histoacryl glue is avoided because of its toxicity, the difficulty in application and perhaps more importantly it prevents a true bony ankylosis at the points of junction between the transposed and normal ossicles.

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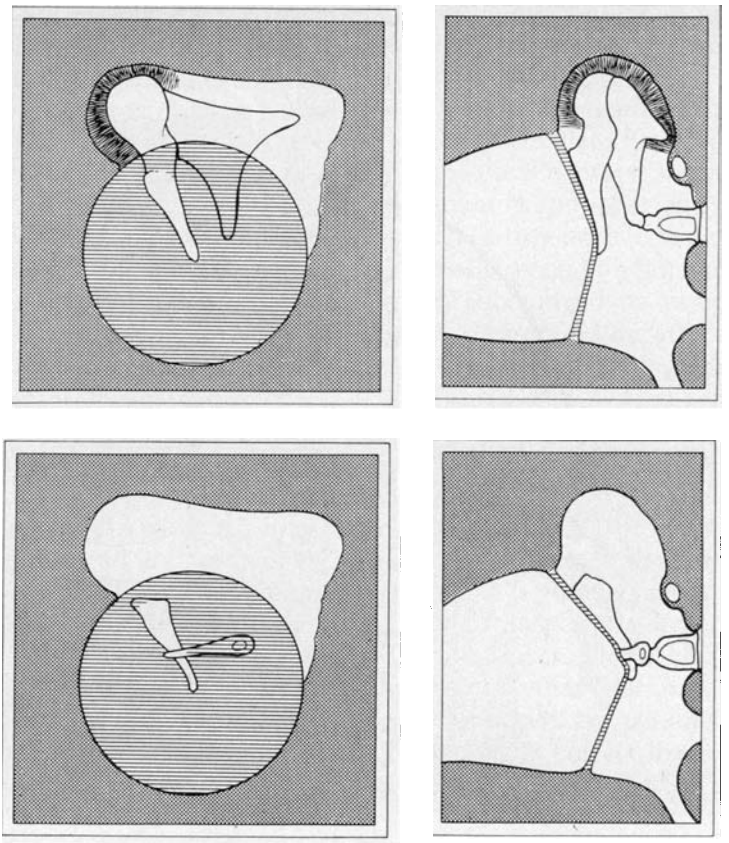


FIG. 5

Extensive tympanosclerosis of the epitympanum treated by incudal transposition.

Autograft or homograft bone are preferred for reconstructive surgery as bone is easy to reshape and readily becomes ossified when repositioned. However, the bone graft must only come in contact with ossicles being rejoined and must not contact the tympanic walls, otherwise the reconstructed ossicular chain will refix. Experience has shown that homograft cartilage eventually tends to disintegrate and the long-term results are more disappointing. Artificial implants are not used for ossicular reconstruction for they do not encourage bony fixation at the points of contact, and indeed may produce bony necrosis, are rejected and if they do work it is a very short-term solution.

Occasionally the malleus is fixed by a mass of bone extending from the anterior wall of the external auditory meatus and tympanic cavity directly to and incorporating the head and handle of the malleus (Fig. 10). The patient in whom this occurred insisted that it was an acquired disorder but it is more likely to be of congenital origin.

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In another case the whole malleus was fixed by a mass of tympanosclerosis replacing the drum itself and fluid was present within the narrowed adhesive middle ear cleft (Fig. 11). Merely lifting the tympanic membrane demonstrated extensive disease and as nothing further could be done the

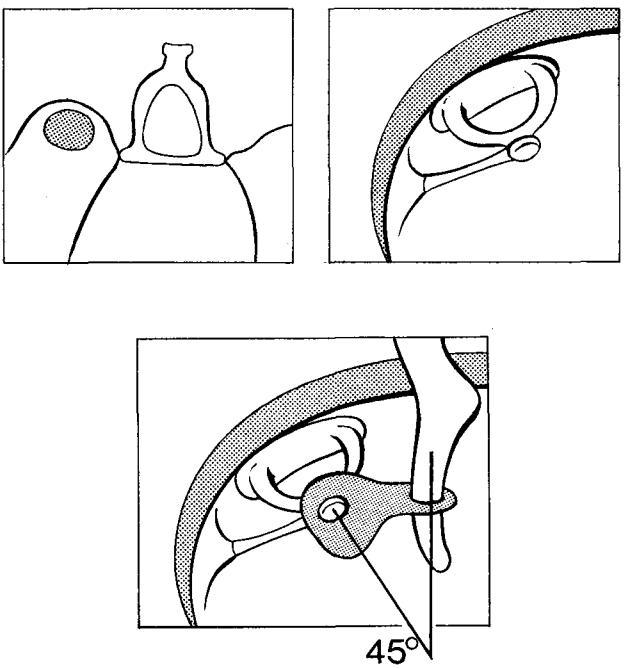


FIG. 6

The two hole technique of Marquet for incudal transposition when anatomically suitable.

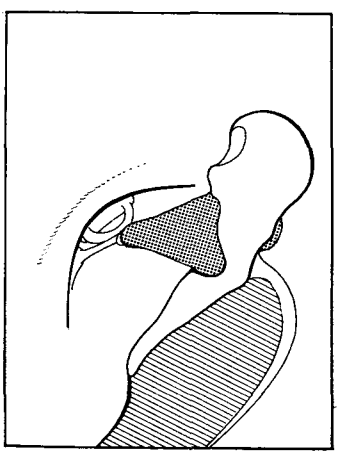


FIG. 7

'Keying' of remodelled incudal body between malleus handle and head of stapes.

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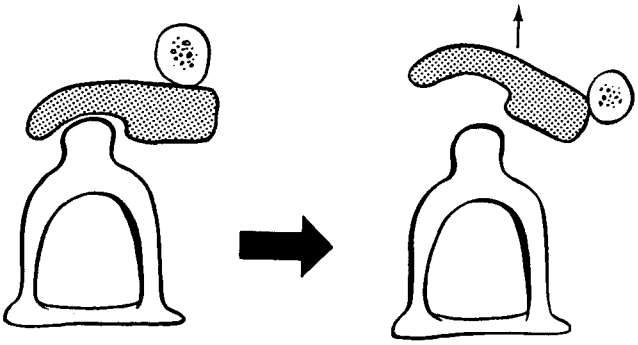


FIG. 8
An unsatisfactory ossicular reconstruction which in the long term frequently leads to non-functional fibrous union between the head of the stapes and the transposed incudal body.

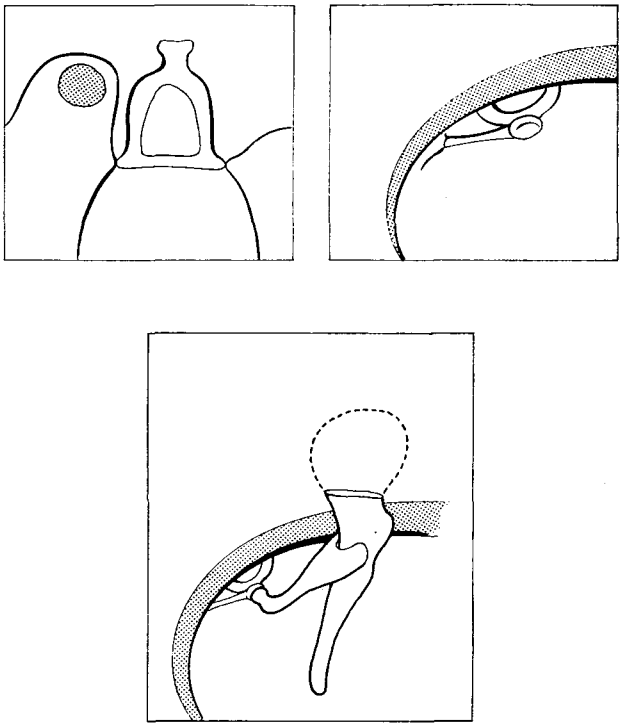


FIG. 9
Ossicular reconstruction using the long process of the incus when the oval window niche is deep.

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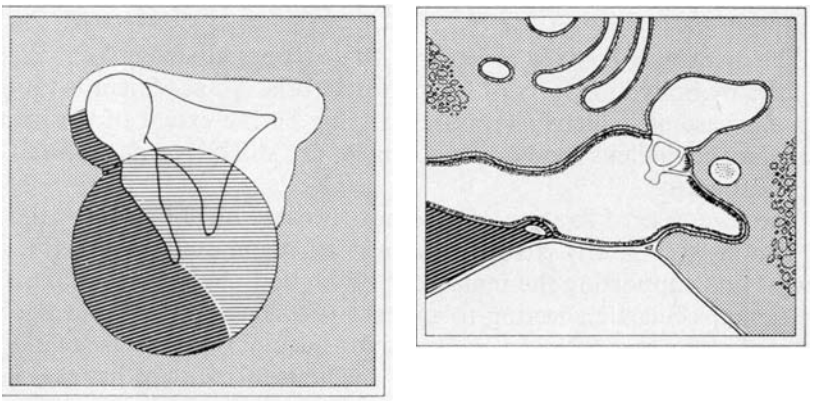


FIG. 10
Massive bony fixation of the malleus.

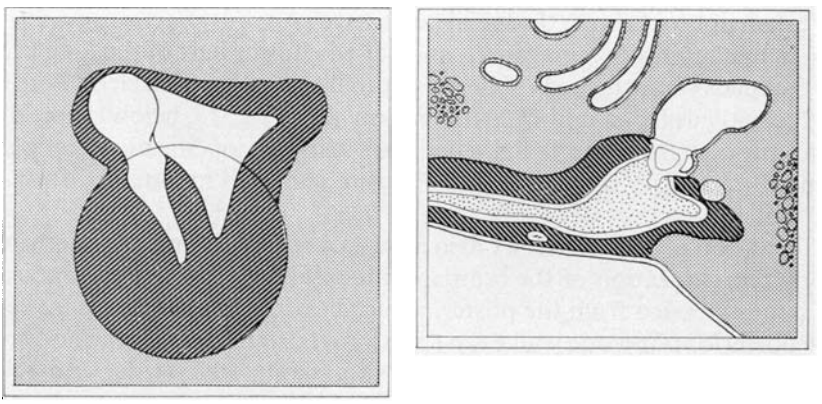


FIG. 11
Tympanosclerosis involving the whole tympanic membrane and medial wall of the mesotympanum accompanied by a serous and adhesive otitis. Note the position of the facial nerve in relation to the tympanosclerotic mass.

flap was replaced but unfortunately the patient developed a facial paralysis. At re-exploration the mass of tympanosclerosis which involved the whole tympanic membrane had extended around all the tympanic walls including the sinus tympani and the facial nerve lay immediately beneath and partly surrounded by this mass. Although the facial nerve was decompressed and found intact recovery did not occur over a period of several months. It is therefore always worth remembering that tympanosclerosis may cover an exposed facial nerve and disturbance may stretch and permanently damage the underlying nerve.

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Fixation of the incus

The incus may be fixed together with or separately from the malleus in the attic by bony ankylosis or tympanosclerosis. Management is the same as in the case of malleus fixation depending on the extent of the fixation. The removed malleus head or incudal body can still be remodelled and used as an autograft.

The rare case of fixation of the short process by ankylosis of the fish-tail ligament (Fig. 12) is readily solved by merely cutting off the short process and supporting the remainder of the body by a piece of Sterispon, and perhaps Silastic sheeting to prevent re-ankylosis to the medial wall. Such ankylosis may follow a cortical mastoidectomy or the use of a homograft monobloc when the homograft incus itself falls against the medial wall of the tympanic cavity; the treatment in each case is the same.

When the long process of the incus is fixed to the facial canal by tympanosclerosis or an adhesion (Fig. 13), division of the adhesion and permanent separation by Silastic sheeting has worked but when the fixation is more extensive it is better to remove the incus and reposition it. Care must be taken not to damage the facial nerve itself.

In one interesting congenital anomaly the lower part of the long process of the incus was attached to the facial canal and only connected to the body of the malleus by an undifferentiated bony bar (Fig. 13, below). Freeing the long process from the facial canal and insertion of Silastic sheeting prevented refixation for the patient's hearing gain was maintained for a long period.

Fixation of the incus may also arise as a result of tympanosclerosis and bony reorganization of the tympanic mucosal folds. In one patient a mass of bone extended from the posterior surface of the handle of the malleus to the posterior tympanic wall deep to an intact drum in which the body of the

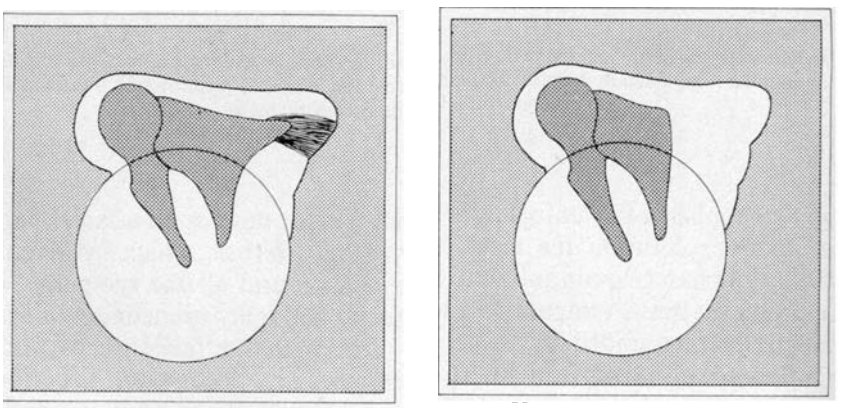


FIG. 12

Fixation of the incudal body by tympanosclerosis of the short ligament solved by excision of the short process.

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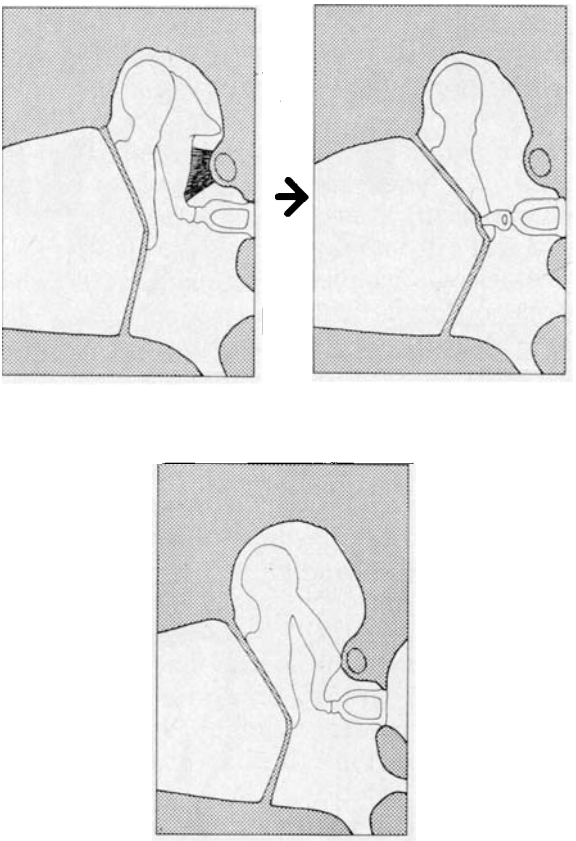


FIG. 13

Above: Fixation of the long process of the incus to the facial canal by tympanosclerosis treated by incudal transposition.

Below: An unusual congenital anomaly—the normal tip of the long process was attached to the facial canal and was only attached to a mobile malleus by an undifferentiated bony bar.

incus was incorporated (Fig. 14). The epitympanum was full of typical tympanosclerosis and the stapes appeared to be mobile. In such extensive cases of tympanosclerosis the patient often gets a better result wearing a hearing aid than is obtained by surgery although today a monobloc homograft transplant may be equally satisfactory when the whole or part of the stapes is mobile (Fig. 15).

Fixation of the stapes

Fixation of the stapes usually occurs at footplate level but fixation may occur at the level of the crura and capitulum.

Traumatic ankylosis of the stapes to the posterior tympanic wall in the neighbourhood of the pyramid has been seen (Fig. 16). The fracture line

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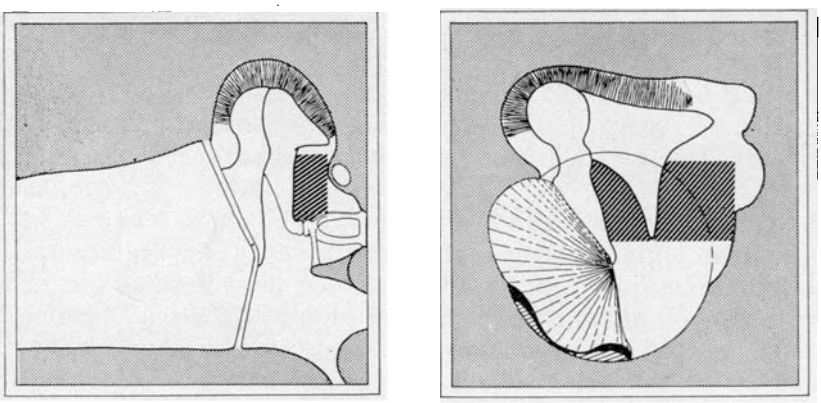


FIG. 14

Diagram to show ossification of the incudo-malleal mucosal folds embedding the incus. (In the lecture this was illustrated by an original coloured slide). The epitympanum was full of tympanosclerosis.

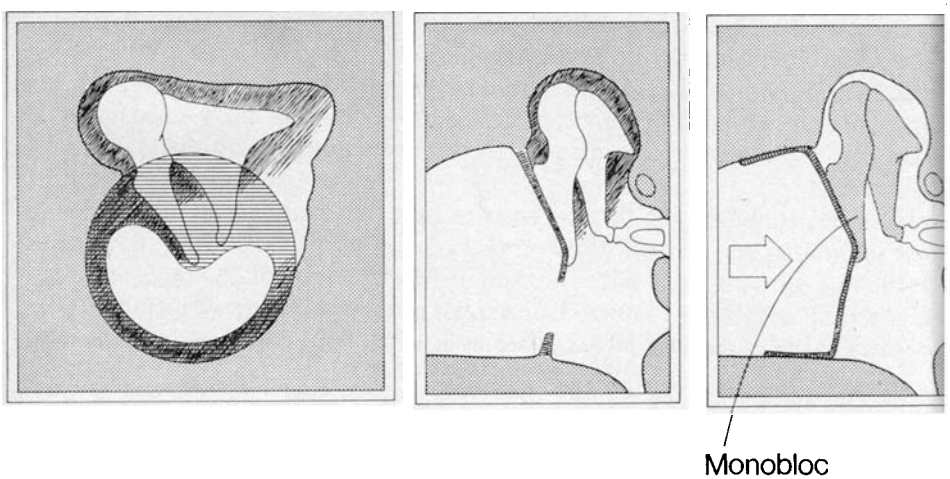


FIG. 15

The use of a monobloc homograft transplant when there is massive tympanosclerosis associated with a mobile stapes.

passed through the posterior wall of the tympanum and the lower fragment of the petrous had been displaced forward to contact the head of the stapes. Freeing the stapes was difficult as the facial nerve itself had to be exposed.

The head and crura may be fixed by tympanosclerosis of the stapedius tendon; removing a segment of the infiltrated tendon frees the stapes.

Tympanosclerosis of mucosal adhesions stretching from the promontory or the facial canal to the crura can fix the stapes, leaving a normal

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mobile footplate beneath; removing the adhesions frees the stapes. Otosclerosis growing from the promontory may fix the crura or even grow between the stapedial crura to the upper rim of the oval window niche but never have I found a mobile footplate beneath it, even though the footplate looked thin and almost normal.

Fixation of the stapes at footplate level is due most commonly to otosclerosis, less so to tympanosclerosis, infrequently due to congenital abnormalities, the Van der Hoeve syndrome or Paget's disease.

Otosclerosis and Van der Hoeve syndrome are best treated by stapedectomy provided that the patient is audiologically suitable. A technically well performed stapedectomy results in closure of the air-bone gap. The subject has been amply discussed but a few pertinent remarks may be permitted.

The chorda tympani can always be preserved even if this means dissecting the nerve from its canal. The stapedius tendon can be preserved in some of those cases where the stapedius tendon is wholly or partially attached to the lenticular process (Fig. 17). The footplate should whenever possible be perforated prior to removing the crura, so reducing the difficulties of dealing with a floating footplate. If a floating footplate does occur and you are not experienced in removing one, leave it for it will refix and the ear can be re-explored later. Fiddle with it and the footplate may be lost in the vestibule and even without such a dramatic occurrence fiddling produces a sensori-neural hearing loss. Furthermore if otosclerotic bone overhangs the footplate remove it before tackling the footplate fixation as the overhang markedly interferes with the ease of removing fragments.

I prefer to make a small circular hole in the centre of the footplate so that it will just accept the piston—the pot-hole technique (Fig. 17, c and d). Even if such perfection is not always attained the opening into the vestibule should be as small as is technically possible. As many perilymph fistulae



FIG. 16

Fracture across the facial canal with displacement of the lower fragment producing ankylosis of the pyramid to the stapes.

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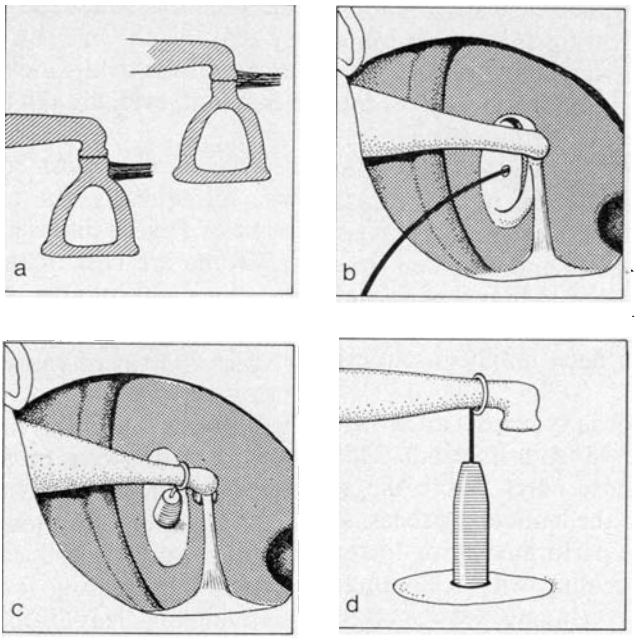


FIG. 17

The 'pot-hole' technique of stapedectomy.

(a) A variation in attachment of the stapedius tendon to the lenticular process or stapedial head.
(b) Perforation of the footplate prior to removal of stapedial superstructure.
(c) Preservation of stapedius tendon.
(d) The 'pot-hole' merely to accept the piston.

result from a sheering motion at least the degree of sheer is limited by the size of the opening. If the whole footplate is inadvertently or deliberately removed then the window must be sealed by vein, fascia or fat. This will prevent an immediate perilymph leak if correctly done but of course in time the sealing material thins and sheering movement of the piston may tear it.

An overhanging facial canal (Fig. 18) may prevent the use of a solid piston or even a wire-teflon piston if the barrel should abut against the canal. This problem can be overcome by shortening the barrel of a long wire-teflon piston so that it is of the correct length (Fig. 19). The shortened barrel can then fit into the oval window and the wire just clears the canal. If the whole footplate has been removed a wire-fat piston does equally well. However, when there is a large bulge of the facial nerve itself this is not possible (Fig. 20). Occasionally when the anterior part of the footplate and the anterior crus are just visible this portion can be removed and the wire-teflon piston fixed to the handle of the malleus. Unfortunately the bulging facial nerve still prevents free movement. Therefore it is probably better to abandon the operation in such cases. If the patient is insistent

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upon a surgical solution, and the majority are not, then the other ear may be anatomically different and as a final resort in suitable cases the lateral semicircular canal can be fenestrated—something I have not done for otosclerosis in twenty years as a hearing aid seemed preferable.

A late cause of failure in stapedectomy is fixation of the head of malleus or body of incus, perhaps the result of healed old disease or ankylosis



FIG. 18
The overhanging facial canal.

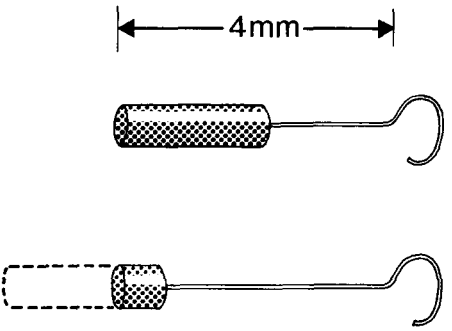


FIG. 19
A modified wire-teflon piston with shortening of the barrel.



FIG. 20
The bulging facial nerve.

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initiated by displacement of a bone chip at the time of stapedectomy. The important factor at re-exploration is to remove the piston first before freeing the other ossicles.

Revision stapedectomy for conductive hearing loss can lead to very satisfactory results but in some patients the operation may have to be repeated on several occasions often successfully. However, final defeat arises when the handle of the malleus disintegrates. It is interesting that patients who have at one time had a successful hearing result from stapedectomy so much prefer the quality of hearing attained compared with that provided by a hearing aid that they very much wish to have the chance of regaining it by surgery.

The question of the second ear operation often arises. Despite the fact that many surgeons advocate that the second ear should never be operated upon the view is taken that this is the patient's choice provided that the implications have been fully explained to them. Some patients need binaural hearing to follow their occupation successfully, and others are insistent upon it, although many patients are quite content with one good hearing ear particularly if the hearing level is within 15 db of normal. In my view the second ear is worth while doing but I tend to leave a variable period of time between the first ear operation and the second ear operation to be sure in so far as it is possible that the hearing in the first ear is stable and lasting. Most stapedectomy hearing results have been very pleasing but a proportion of patients, approximately 10 per cent, have had troubles over the years, most of which are correctible, a few not. As the patient's life expectancy post-stapedectomy depends on their age at the time of operation there is a need for some very flexible guide-lines. If a teenager, then perhaps twenty years should be the time interval, in the third and fourth decades a ten-year interval, in the fifth decade five years and in the sixth and seventh decades one to three years. These rules have been broken on many occasions as they must vary according to individual needs.

Occasionally a rapid deterioration in the unoperated ear with widening of the air-bone gap with or without apparent or real deterioration in bone conduction levels does occur. It seems reasonable to explore such an ear and the impression gained has been that further deterioration has been prevented but these cases are relatively uncommon and no definitive answer can be given.

Congenital fixation of the stapes footplate is due to failure of full differentiation of the otic capsule. It may be accompanied by other congenital abnormalities of the ossicular chain such as undifferentiated stapedial crura. In cases of partial differentiation where a stapes is recognizable or almost normal, stapedectomy is done. However, as congenital fixation of the stapes may be accompanied by a patent aqueductus cochlearis, if the small opening into the vestibule made before crurotomy or partial stapedectomy produces a free flow of perilymph fluid it is wisest to cover the small fistula with fascia or vein, plug it in position with Sterispon

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and abandon the operation. On such occasions it has been possible to obtain a very successful result with stapedectomy but the incidence of 'dead' ear is higher.

Total failure of differentiation of the otic capsule may be accompanied by an almost normal pinna and external ear canal but at tympanotomy an abnormal incus and rudimentary stapedial crura lying on the promontory are found. In one patient the ear with the greatest pinnal deformity had a total failure of differentiation of the otic capsule and in the other ear, which had an equal degree of conductive deafness, apart from an expected abnormal incus and stapes a stapedial artery was found. The patient manages very well with a hearing aid.

Tympanosclerosis may fix the stapes at footplate level by involvement of the overlying mucous membrane or by infiltration of the annular ligament. The pot-hole technique of stapedectomy is ideally suited and it may seem easy to perforate a thin footplate. However, the pressure required to perforate the footplate readily displaces the footplate into the vestibule. Therefore it is even more important that the footplate should be perforated before fracturing the crura, and the mucosa must not be widely stripped off the footplate for it helps to retain fixation during perforation. If the whole footplate has to be removed together with the crura for ease and safety then the oval window must be sealed by fascia, vein or fat. The end results are rarely as satisfactory as occurs in otosclerosis for the mucosal folds and suspensory ligaments are stiffer and frequently a 20 db air-bone gap has to be accepted post-operatively.

A massive tympanosclerotic infiltration of the oval window niche presents a more exacting problem. Careful stripping of the layers without displacing the stapes is impossible and by the time the footplate is exposed the whole stapes is very wobbly and has to be totally removed and no doubt in many patients a sensori-neural loss or dead ear has already been induced. Furthermore as the tympanosclerosis in these cases is more extensive the post-operative mucosal healing is probably delayed; no wonder that after stapedectomy in many patients the hearing is made worse.

Statistics are no guide to the management of such cases for so often the extent of the disease and difficulties of surgery are not reported—it is a matter for individual judgement according to the individual problem.

Occasionally the whole of the middle ear cleft is filled by masses of tympanosclerosis which not only fix but totally embed the ossicular chain. If localized to the attic careful removal of the masses of tympanosclerosis may free the chain and produce a pleasing hearing result but if the whole chain is involved a fenestration can be done in selected cases or a hearing aid prescribed. A fenestration was done in six cases with reasonable results but as they did not all last for a long period I would probably now have given them a hearing aid.

Each and every case of ossicular fixation presents an individual problem and each must be treated according to its merits. However,

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patients who have ossicular fixation only, do very well with a hearing aid. There is no harm done or shame incurred if it is your judgement that an operation should be abandoned because of the attendant risks to the patient's hearing.

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