EXPERIMENTAL STAPEDECTOMY: THE INTERNAL EAR CHANGES

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THE changes in the internal ear which occur after stapedectomy in the experimental animal have so far received scant attention in the literature. For this reason the effects of stapedectomy on the human internal ear remain somewhat speculative though light has been thrown upon the problem by the contributions of Bellucci and Wolff (1958), (1960). Singleton and Schuknecht (1959), Schuknecht and Tonndorf (1960), Schuknecht and Oleksuik (1960) and Harris (1961).

The present paper describes the post-operative changes in the internal ears of eighteen cats and the relevance of these changes to human stapes surgery is discussed.

The results of these experiments supplied material for an earlier paper (Colman, 1960) which dealt with the appearances of successful grafts across the oval window after removal of the stapes and made no attempt to describe the results of an imperfect operation or of failure. The animals were usually killed at sixteen to eighteen weeks by intravital perfusion with Ringer's solution followed by Heidenhain-Susa solution as a fixative. As indicated in the earlier paper the types of graft were skin, mucosa (advocated by Tato, 1960), vein (Shea, 1958), conjunctiva, connective tissue and adipose tissue (Schuknecht, McGee and Colman, 1960).

The table overleaf indicates the abnormalities recognized in the internal ears and shows the type of graft used in the animal concerned.

(1) *Acute labyrinthitis*

This occurred once only (animal Mm 1), with a mucous membrane graft. Sudden severe ataxia and nystagmus occurred on the forty-fourth day after operation. A low grade infection localized to the attic part of the middle ear was associated with graft necrosis. Infiltration by acute inflammatory cells was still confined largely to the vestibule of the labyrinth, the cochlea showed severe hydrops with early infiltration of the scala vestibuli. The posterior labyrinth was not yet involved (Fig. 1).

(2) *Sero-fibrinous labyrinthitis*

This was observed in two animals, ataxia and nystagmus were not present and graft necrosis was presumably slow (animals Vn 1, Vn 2) (Figs. 2 and 3). A vein graft had been inserted into each of these ears.

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TABLE OF ABNORMALITIES OBSERVED

Abnormality	Times Seen	Graft	Animal
(1) Acute labyrinthitis	Once	Mucosa	Mm 1
(2) Sero-fibrinous labyrinthitis	Twice	Vein	Vn 1 Vn 2
(3) Electro-coagulation injury	Three	Vein and skin	Vn 2 Vn 3 Sk 1
(4) Hydrops	Five	(The animals included above)	
(5) Saccule rupture	Twice	Mucosa and adipose tissue	Mm 1 Ft 2
(6) Blood contamination	Twice	Connective tissue and mucosa	CT 3 Mm 3
(7) Foot-plate fragments and remnants	Five	Skin, adipose tissue and conjunctiva	Sk 2 Sk 3 Ft 1 Ft 2 Conj 3
(8) Graft herniation	Twice	Skin and conjunctiva	Sk 2 Conj 1

Mm—indicates an animal with a graft of mucosa; Vn—Vein; Sk—skin; Ft—adipose tissue; CT—connective tissue; Conj—conjunctiva.

Inadequate oval window protection resulted in invasion of the internal ear by proliferating fibroblastic tissue. A sero-fibrinous exudate with many round cells, particularly macrophages was present. Acute inflammatory cells are very few or absent not only in the area of the degenerating graft but also in the vestibule. The process in each animal was beginning to involve the cochlea. Scanty fibroblasts, round cells and macrophages were present in the scala vestibuli and ballooning of Reissner's membrane was present. Loss of hair cells was nearly complete throughout the organ of Corti though the supporting elements and stria vascularis were almost normal in appearance. The vestibular labyrinth was histologically normal apart from slight exudate in the perilymph space.

(3) *Electro-coagulation injury*

Damage of this type to the labyrinthine capsule and cochlear contents was observed three times (animals Sk 1, Vn 2 and Vn 3). Changes were not found in two other animals (Sk 2 and Sk 3) in which a coagulation current was similarly used to arrest persistent bleeding from the tensor tympani muscle.

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FIG. 1.

Acute labyrinthitis. Graft necrosis and invasion of the vestibule by acute inflammatory cells. A rupture of the saccule is present (arrowed) and cells are entering at this point (×22).

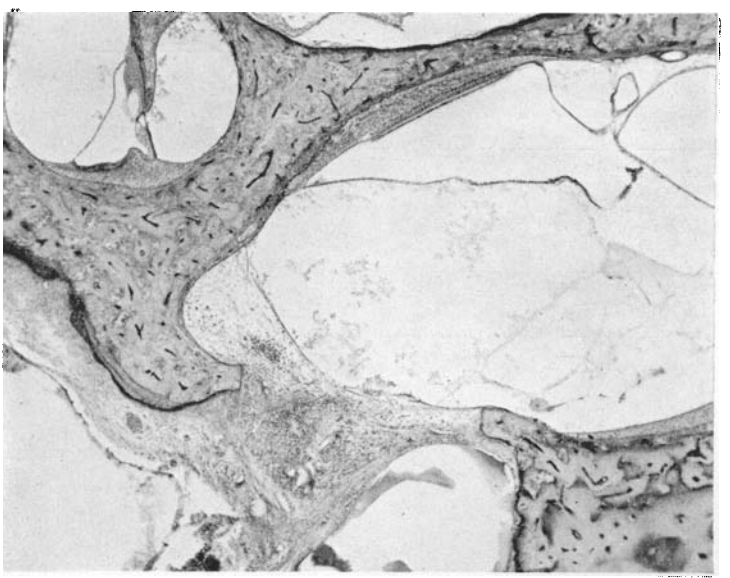


FIG. 2.

Sero-fibrinous labyrinthitis. Serous exudate, fibroblasts and round cell infiltration in the vestibule and in the perilymph spaces of the cochlea. The result of slow aseptic graft necrosis (×22).

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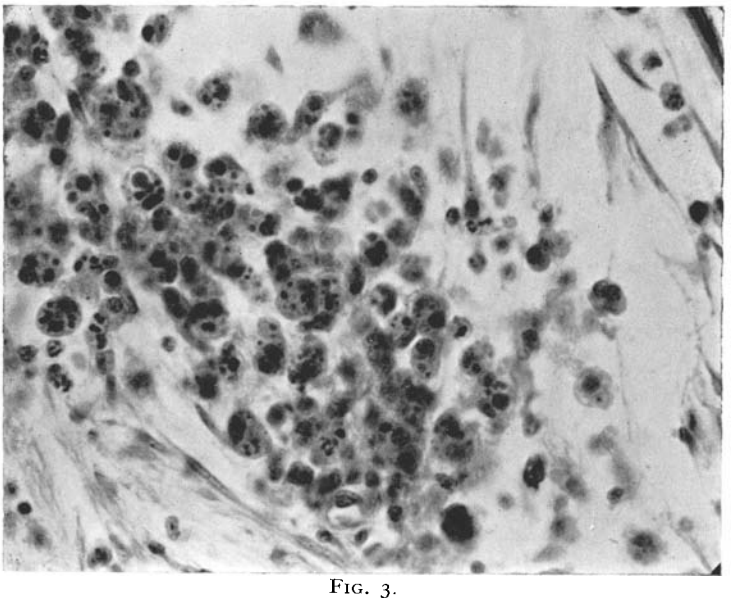


FIG. 3.
Sero-fibrinous labyrinthitis. The cellular infiltration contains great numbers of phagocytes (×450).

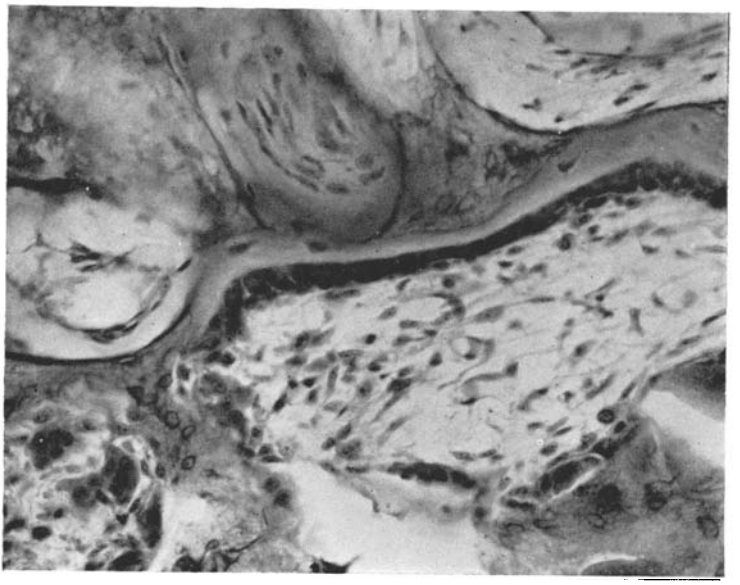


FIG. 4.
Electro-coagulation injury. Decalcified area containing loose fibroblastic tissue and lined along its upper edge by a layer of osteoblasts. A mantle of metachromatically stained bone lies above this layer (×300).

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That the bony lesion exactly followed the vascular channels is indicated by its irregular pattern and diminished severity around outlying vessels. Distant foci sometimes appeared to have no continuity with the main lesion unless traced carefully in serial section.

The bony lesions were characterized by decalcified areas filled with loose fibroblastic tissue and lined by closely packed osteoblasts (Fig. 4). The presence of mantles of metachromatically staining bone surrounding these areas was notable as well as the higher degree of cellularity of this bone. Many large, multi-nucleated giant cells having the appearance of osteoclasts were present (Fig. 5). Areas of predominantly osteoblastic and predominantly osteoclastic activity were often in close relationship with one another. The overall picture was in many respects suggestive of certain features occurring in human otosclerosis.

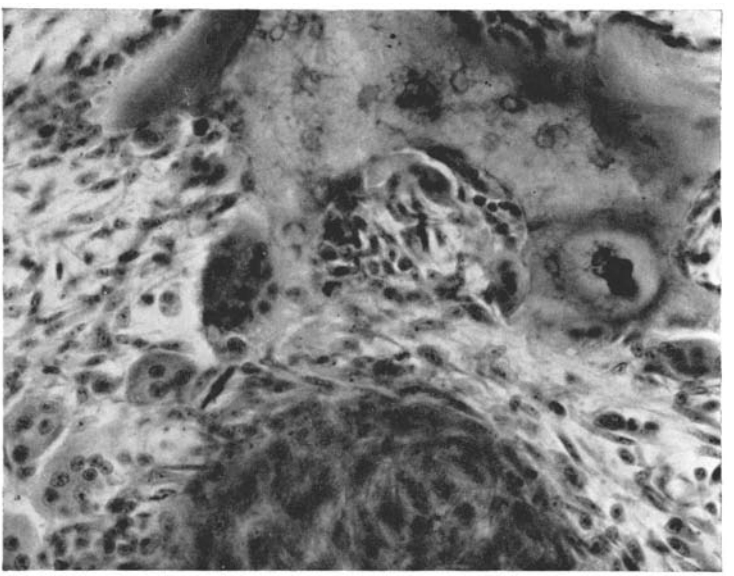


FIG. 5.
Electro-coagulation injury. Area of predominantly osteoclastic activity.

Associated changes in the cochlear contents consisted of a diffuse necrosis involving all structures contained in the scala media.

(4) *Hydrops*

This occurred in varying degree in five animals but only as part of an extensive cochlear degeneration under the three headings just described. As well as the obvious bulging of Reissner's membrane there was swelling of the cells of Deiters and Hensen and vacuolation in the stria vascularis.

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(5) *Rupture of the saccule*

The lesion was observed in two animals (Mm 1, Ft 2) and was in the same situation in each, directly beneath the foot-plate. The underlying macula did not show any evidence of mechanical injury. Infection complicated the picture in the first animal (Fig. 1) but in the second the internal ear was otherwise entirely normal.

(6) *Blood contamination*

Blood cells were found in the labyrinth on two occasions (animals CT 3 and Mm 3) though the operation in one of these was thought to have been bloodless. The blood cells were concentrated in the scala vestibuli and scala tympani of the apical and middle turns of the cochlea and were absent elsewhere (Figs. 6 and 7). The cochlea of one of these animals was



FIG. 6.
Blood contamination. Many blood cells are present in the cochlea at the three points arrowed (×22).

graphically reconstructed as in the method for plotting hair cell damage (Schuknecht, 1953). The spatial distribution of the blood cells could thus be accurately represented, they were confined to the adjacent parts of the upper two cochlear turns and concentrated in the same part of the lumen of each scala (Fig. 8).

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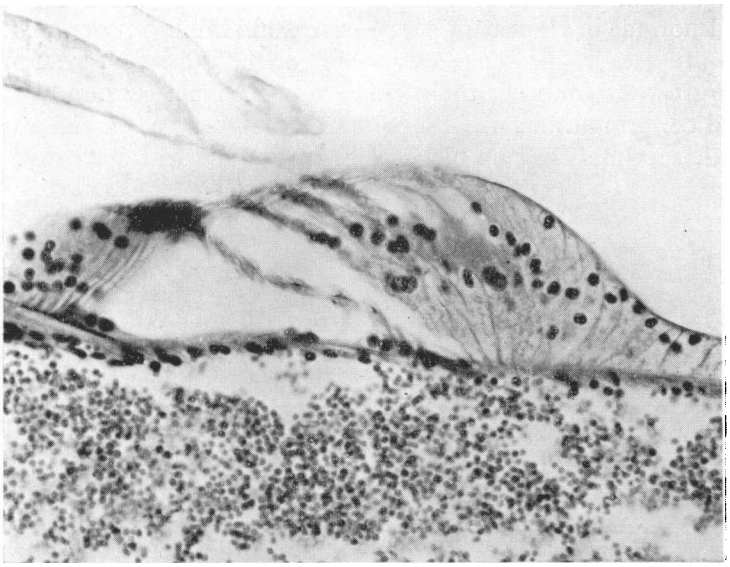


FIG. 7.
Blood contamination. Apical turn of cochlea. The organ of Corti is normal (×300).

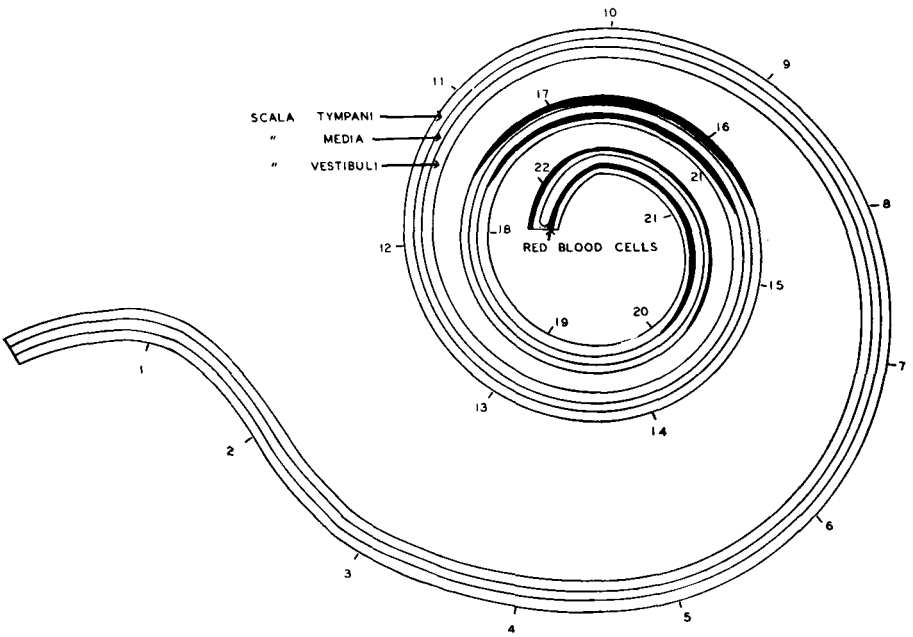


FIG. 8.
Blood contamination. Graphic reconstruction of cochlea to show distribution of blood cells (shaded area). The numerals indicate the distance in millimetres measured from the basal end of the organ of Corti. In this animal it was 22·5 mm. long.

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In both animals red cells only could be detected. Individual cells appeared normal in respect of shape, size and staining properties and no reaction to their presence was detected. Phagocytes containing hæmosiderin were found in the cochlea of one animal (Fig. 9).

Blood contamination was thought to have occurred in the operations on six other animals but no blood cells were found histologically.

(7) *Foot-plate fragments*

Careful examination never revealed the presence of foot-plate fragments inside the labyrinth even in those animals in which pieces of bone seemed to disappear deep into the vestibule at the time of stapedectomy. In such animals, spicules of bone were found adherent to the vestibular surface of the graft and bound to it by a strand or a covering of endothelium. Foot-plate remains at the window margins (Fig. 10) were also sometimes present. Such fragments and remains were not associated with any excessive fibroblastic response nor any evidence of osteogenesis and there was no tendency for new bone to grow across the window from them.

(8) *Graft herniation*

In two animals (Sk 2 and Conj 1) exposure of the perilymph to middle-ear mucosa occurred due to herniation of the graft from the window margin posteriorly. The mucosa in each case appeared normal and still possessed cilia and goblet cells. The mucosa merged with endothelium on the deep surface of the graft and at the posterior margin of the oval window without making any attempt to replace it (Fig. 11). Changes in the organ of Corti present in one of these animals were characteristic of coagulation injury.

(9) *Damage to the organ of Corti*

The organ of Corti was abnormal in six of the eighteen ears. The animals and any obvious associated pathology were as follows:

(*a*) Sk 1 electro-coagulation injury.
(*b*) Mm 1 acute labyrinthitis.
(*c*) Vn 1 sero-fibrinous labyrinthitis.
(*d*) Vn 2 coagulation injury and sero-fibrinous labyrinthitis.
(*e*) Vn 3 coagulation injury.
(*f*) Conj 1 coagulation injury (but graft herniation also present and stapedectomy considered to have been traumatic).

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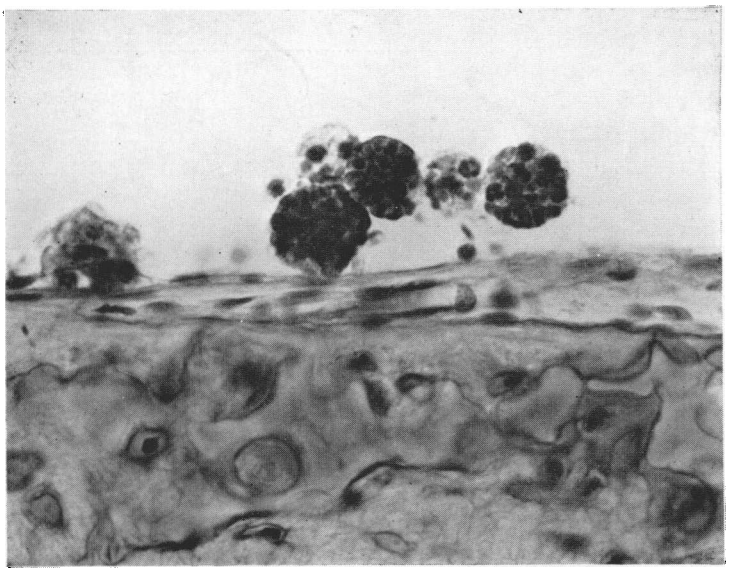


FIG. 9.

Blood contamination. Phagocytes laden with blood cells (×600).

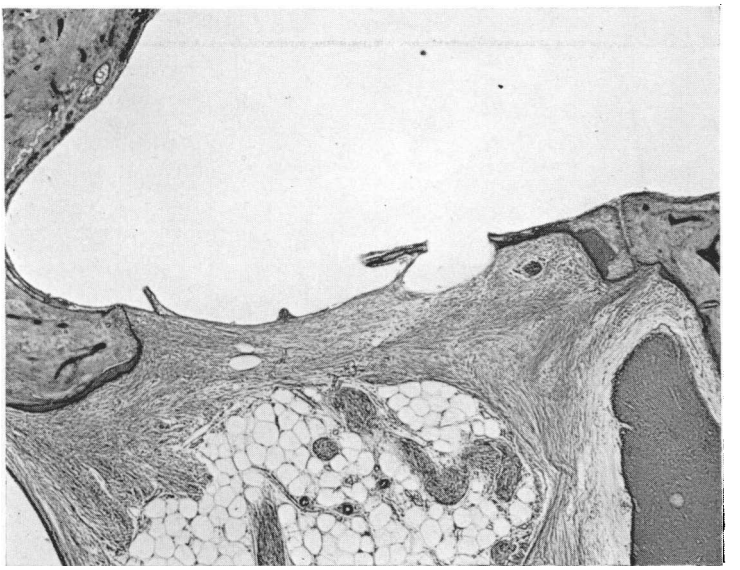


FIG. 10.

Footplate fragments. Two spicules of bone as well as a remnant of footplate at the posterior edge of the window are adherent to the graft. Clearance was thought to have been complete in this animal though one fragment was known to have fallen into the vestibule (×40).

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(10) *Vestibular neuro-epithelial damage*

The vestibular labyrinth appeared notably resistant to injury. Even in the cases of sero-fibrinous labyrinthitis, the neuro-epithelium still appeared histologically normal. The neuro-epithelium structures were abnormal only in two ears: degeneration of the saccular macula was present in the animal with an acute labyrinthitis and in one animal with an electro-coagulation injury.

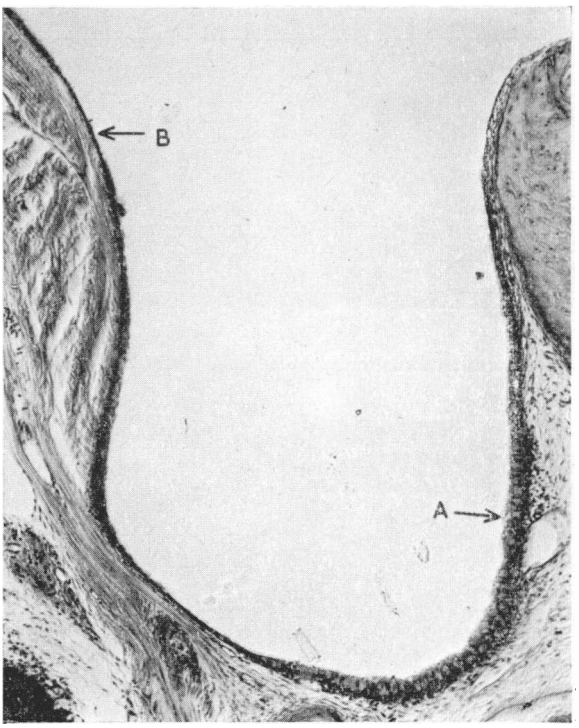


FIG. 11.

Graft herniation. The perilymph is in contact with goblet-containing mucosa (A). The graft is covered by normal endothelium in the ordinary way (B). (×85).

The combined abnormalities in the internal ear for each animal are thus as follows:

Skin grafts

Sk 1 electro-coagulation injury, hydrops, organ of Corti damage.
Sk 2 graft herniation. Otherwise normal.
Sk 3 normal.

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Mucous membrane grafts

Mm 1 early acute labyrinthitis, hydrops and diffuse degeneration of organ of Corti. Rupture of saccule and degeneration of saccular macula.
Mm 2 normal.
Mm 3 blood in cochlea.

Vein grafts

Vn 1 sero-fibrinous labyrinthitis with hydrops, degeneration in organ of Corti and saccular macula.
Vn 2 sero-fibrinous labyrinthitis, electro-coagulation injury, hydrops, degeneration in organ of Corti.
Vn 3 electro-coagulation injury, hydrops, degeneration in organ of Corti.

Conjunctival grafts

Conj 1 graft herniation, damage to organ of Corti, electro-coagulation injury.
Conj 2 normal.
Conj 3 normal.

Connective tissue grafts

CT 1 normal.
CT 2 normal.
CT 3 blood in cochlea.

Adipose tissue grafts

Ft 1 normal.
Ft 2 saccule rupture.
Ft 3 normal.

Discussions of Findings

The prime essential of any type of graft is to provide an adequate seal across the oval window to afford immediate and lasting protection to the sense organs of the internal ear.

All six kinds of tissue examined were found capable of providing such protection, even skin which for other reasons shown earlier was entirely unsuitable. In one animal only did sudden necrosis occur and this resulted in acute diffuse labyrinthitis. This possibility must be borne in mind when in human surgery a flimsy graft such as mucosa is employed or a non-viable film such as gel foam is inserted which may result in the formation of an equally delicate membrane.

It was notable that vestibular symptoms were absent in the animals with sero-fibrinous labyrinthitis and that the histological changes were confined to the anterior labyrinth.

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This type of internal ear failure appears to be the result of a slow graft necrosis and the response is caused mainly by diffusion of cell-breakdown products. The oval window remains closed by the fibroblastic reaction and the already healed middle-ear mucosa.

This may represent the pathology in certain cases of sudden cochlear failure without vertigo, which occasionally occur in human patients after an apparently satisfactory grafting operation. The oval window diaphragm appears to be functioning satisfactorily until the sudden release of toxic products and graft debris causes havoc in that part of the ear which is most sensitive and most accessible from the oval window, i.e. the basal turn of the cochlea. If any part of the cochlea survives this invasion and the subsequent arrival of round cells, phagocytes and fibroblasts it is the apical turn and the relatively unimportant lower frequencies. This is the process which is perhaps occurring under cover of the area of exuberant repair noted in those few patients whose ears have been urgently re-explored within a few hours of cochlear failure.

Though this explanation may be applicable to those cases of human failure which occur relatively early in convalescence it is less satisfactory for those few cases of acute cochlear failure which occur later. The observation of Singleton and Schuknecht (1959) is probably relevant. In the course of their animal investigation into the effect of trauma around the stapes foot-plate they showed that a minute fistula between middle ear and labyrinth can persist as long as 290 days without producing symptoms of internal ear damage. They illustrate their paper by a histological section which shows a normal internal ear but with a strand of polymorphs just entering the vestibule. There is no reason why fistulas should not persist for 290 days in the human oval window particularly if the graft is not in perfect apposition. From the article by Singleton and Schuknecht it is evident that the fistula may be very small and difficult to see with the ordinary operating microscope. The risk is probably least with a graft such as adipose tissue which tends to mould itself exactly to the shape of the window.

Improper use of electro-coagulation was a significant cause of internal ear damage and in fact Shea (1958) has found it necessary to draw attention to the dangers associated with its use in the human ear. The injury consisted of a necrosis of all elements of the organ of Corti and stria vascularis which was most severe in those parts closer to the point of application. In injuries with necrosis of all the neuro-epithelial structures, perfectly normal looking nerve fibres were nevertheless present. These were evidently regenerated efferent fibres to the cochlea of the type first described by Rasmussen (1946), (1953). Portmann has shown that they probably end on the inner hair cells and Churchill and Schuknecht (1956) suggest they are cholinergic and inhibitory.

The most severe coagulation injuries produced changes in the

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labyrinthine capsule which, as already indicated, presented many of the features of active otosclerosis: in both conditions the picture is of bone removal and replacement, in neither is the need for bone removal obvious histologically. Presumably the bone is in some way abnormal, the passage of an electric current perhaps can interfere with vital enzyme systems and thereby disturb the metabolic processes. The changes in these animals and in human otosclerosis may represent a non-specific type of repair which can occur in response to a variety of damaging agents or inborn abnormalities.

Bellucci and Wolff (1959) have also noted otosclerosis-like lesions in some of their animals after electro-coagulation injury. They too, raise the possibility that the changes may be a non-specific response produced by widely different agents and while not claiming to have produced histological otosclerosis they believe they may have found one of the basic causative factors, *viz*: a disturbance to the vascular supply to the otic capsule. They suggest an unknown agent may produce similar results in the human otic capsule.

Labyrinthine hydrops occurred only as part of a severe labyrinthine lesion though Hohmann (1960) states it is the earliest and commonest reaction found after stapes foot-plate procedures. He found it to be present in 17 per cent. of foot-plate fractures and 26 per cent. of stapedectomies in animals operated on by various persons. It is well known that temporary depression of cochlear function is frequent in human patients soon after stapes procedures and this is probably one of the changes present. He also describes an opposite change, hypotonic atrophy, characterized in animals by shrinkage of the contents of the scala media, depression of Reissner's membrane, depression of the tentorium on to the organ of Corti and severe shrinking of the saccule. The disorder is a rare complication of foot-plate surgery and was not observed in the current experiment.

Damage to the membranous labyrinth was confined to the saccule in the two animals in which it was noted and was directly beneath the centre of the foot-plate. The close proximity of the saccule to the foot-plate renders it especially vulnerable when the foot-plate is penetrated with a needle, injury would probably be more frequent but for the tendency of the saccule to yield and fall away as does the semi-circular canal in lateral semi-circular canal fenestration. Singleton and Schuknecht found an injury of the cochlea and of the saccular macula in four of eight ears in which a saccular lesion was present and a saccular tear in every ear in which a needle fenestration of the foot-plate or annular ligament was made. It must be emphasized however that their experiment was directly concerned with the results of carefully inflicted trauma aimed at the stapes, whereas the current experiment was done as atraumatically as possible.

Apart from the hydrodynamic changes occuring in foot-plate mani-

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pulation and capable of producing damage at a distance, it is surprising that direct instrumental injury is not in fact more common. The close proximity of certain vital structures even in the larger human ear puts these parts in substantial hazard and the safety margins must constantly be borne in mind. Anson and Bast (1958) have stressed the close relationships in this region in a careful and most important study. The following measurements are of special interest.

Anterior part of oval window to saccule 0·75 to 1·6 mm.

Anterior part of oval window to internal meatus 1·75 mm.

Upper part of oval window to utricle 0·5 to 1·6 mm.

Anterior part of foot-plate to proximal end of cochlear duct 0·3 mm.

Although the corresponding distance from window must be substantially smaller in the cat, it was notable that adhesions of utricle, saccule or cochlear duct to the graft were never observed, whether the latter was satisfactory or not. It is possible that removal of the graft in a human revision operation may thus be less dangerous than Shea (1961) has suggested.

Blood was found in the cochlea of two animals but was absent from six others thought to have been contaminated. The red blood cells appear to act as entirely inert foreign bodies going wherever fluid currents and gravitational forces take them. These cells persist in a good state of preservation and survive longer than in the general circulation.

Schuknecht and Davison (1956) found many red cells in the cochlea five months after experimental head injury (i.e. slightly longer than in these two animals) but the exact time which cells can persist remains unknown. Removal of the red blood cells is undertaken by phagocytes and these are active as early as the tenth day after operation (Colman, unpublished data). The paucity of such scavenging cells indicates that blood clearance must be extremely slow when contamination is of any magnitude. The complete absence of white blood cells among so many red cells suggest that their removal is simpler and quicker but the method of removal remains unknown.

There was no evidence in this experiment that blood or its break-down products had any adverse effect on the internal ear. Hohmann (1960), in a recent animal study in which he injected blood into the labyrinth, reached a similar conclusion. House (1960) has expressed the same view in relation to human stapedectomy but Shambaugh (1960) in comparison found a relationship between bleeding and cochlear damage in human operations. The experimental evidence suggests that the relationship is an indirect one: bleeding tends to be associated with the more difficult cases of foot-plate removal and the obscured field renders manipulation less precise and deliberate, the cochlear loss is thus perhaps the result of instrumental

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trauma (i.e. a stimulation injury) and only indirectly the result of blood contamination.

The absence of foot-plate fragments from the cavity of the labyrinth is of direct practical interest. On several occasions fragments undoubtedly fell free into the vestibule and sometimes could be seen lying deeply in the vestibule with the saccular macula beneath. Other fragments of the foot-plate edge appeared to fall free but the histological appearances in these animals suggests that they merely hinged inwards on their endosteal attachments. It is clear that no harm results from the loss of fragments into the internal ear and that there is no justification for hazardous attempts at removing them. The experimental evidence indicates that lost fragments become adherent to the deep surface of the graft; they must do so whilst it is still not endothelialized as they are usually incorporated beneath the intact endothelium, which has been shown by Schuknecht (1962) to be completed at ten days. Foot-plate fragments did not appear to stimulate fibrosis or osteogenesis in the labyrinth of these particular animals and their early fixation precludes any possibility of symptoms arising from abnormal stimulation of the vestibular sense-organs. Schuknecht (1962) agrees that bone chips in the vestibule are entirely inert but on the other hand has shown experimentally that foot-plate fragments can become osteogenic if in contact with connective tissue in the oval window region. It would appear, therefore, that bony re-closure of the oval window is a greater possibility in human surgery if the foot-plate removal is not total.

Exposure of the perilymph space to middle-ear mucosa was present in two animals, the result either of inadequate positioning and support of the graft or of its actual displacement by the perilymph. Perilymph overflow was troublesome in certain animals and occurred in ears subsequently found to have a widely patent cochlear aqueduct, a not infrequent occurrence in cats. Similar overflow, sometimes amounting to severe rapid flooding of the middle ear is a well recognized, but fortunately an unusual hazard, in human stapedectomy and is probably also due to a widely open aqueduct. In these two animals with graft herniation or malposition, the mucosa showed no tendency to spread on to the deep surface of the graft, which became lined by endothelium in the usual fashion. Although the organ of Corti was damaged in one animal the features were those of a coagulation injury. The normality of the organ of Corti in the other animal does not lend support to the view of Farrior (1960) who regards mucous labyrinthitis as an important cause of severe deafness after human stapedectomy, perhaps resulting even from a single mucosal cell being buried under the graft.

As already indicated, a number of animals showed changes in the organ of Corti but in each there was sufficient evidence such as labyrinthitis or coagulation injury to explain the damage. Foot-plate removal in several

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animals was considered to have been traumatic and so in these an operative note was made to expect histological evidence of stimulation injury, the features of which render it easily identifiable. Tonndorf (1959) in his extensive studies on the hydrodynamics of the cochlea has shown by means of cochlear models that maximal displacement of the cochlear partition during manipulations at the oval window occurs in the area corresponding to the lower middle and upper basal turns. The frequency of damage in this area in experimental animals was reported by Singleton and Schuknecht in their paper on stapes trauma already quoted. Cochlear injury was present in four of their fourteen animals and was in the site predicted by Tonndorf. Damage was most severe in the outer hair cells and less severe in the inner group. The lesions were thus very similar to those of high intensity noise and head injury but they point out that the lesions of stimulation injury are less discrete than in true acoustic trauma.

The features of stimulation injury are thus clearly defined and readily recognizable and though the condition may have been obscured in some of the animals which sustained severe cochlear damage from electrocoagulation or labyrinthitis, it was expected to be manifest in certain other animals. As in human stapedectomy, the utmost care was exercised in extracting the foot-plate but removal was considered to have been traumatic in the operations on animals Sk 2, CT 2 and Ft 1 ("foot-plate fragments removed after much difficulty, at least one piece was lost completely inside the vestibule"). Yet even in these animals the sense organs were entirely normal on careful histological examination. It is also worth noting at this point that no evidence was found to suggest that loss of perilymph was in anyway damaging even though loss was severe on several occasions.

The failure to detect evidence of stimulation injury in this experiment coupled with the fact that Singleton and Schuknecht obtained it only after relatively severe trauma might suggest that the cochlea is relatively resistant to injury. Too close a comparison with human work is clearly to be avoided. The removal of a normal foot-plate in the cat is probably less damaging than the removal of an otosclerotic human one, especially one with extensive disease and in man the extreme delicacy of the organ of Corti must never be underestimated.

The absence of vestibular histological change in the presence of severe cochlear damage was of especial interest, an injury of considerable magnitude was obviously indicted on these animals but produced no vestibular degeneration and either transient vestibular symptoms or none at all.

The "damage threshold" is thus very high for the vestibular apparatus in comparison to the cochlea and it follows that trauma and manipulation capable of producing only moderate vestibular symptoms are capable of producing severe cochlear damage, an idea perhaps worth remembering when operating on the human oval window.

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Conclusions and Summary

Ideally the only response of the internal ear to the presence of a graft across the oval window is to provide it with an endothelial lining. In fact, this was the only response elicited in most animals. Of the various internal ear abnormalities described, it is evident that the majority were caused by error in the operative technique rather than the presence of a graft. Electro-coagulation injury, loss of bony fragments and blood contamination of the perilymph, for example, come into this category. In human surgery it is hoped that electro-coagulation injury does not occur. The loss of stapes fragments and entrance of blood into the internal ear are undesirable but neither complication is in itself damaging and the labyrinth is capable of dealing satisfactorily with both mishaps.

As only three operations were done with each type of graft no attempt is made to correlate internal ear complications too closely on the kind of graft implanted. But of complications due to graft failure acute labyrinthitis appears to be a danger when using flimsy grafts such as mucosa. Presumably the same risk is present after using a non-viable substance, such as gel foam across the window as this also can form an extremely thin membrane.

Sero-fibrinous labyrinthitis appears to result from slow necrosis of the graft, the release of toxic products into the basal turn of the cochlea being responsible for severe deafness whilst the window remains closed by the disorganized graft and its mucosal covering. The necrosis probably results from inadequate nutrition of the graft. Sero-fibrinous labyrinthitis occurred twice in this series, each time with a vein graft but it is likely that it can occur with virtually any kind of graft.

Saccular rupture was observed but it is not possible to decide whether it is dangerous to the cochlea or not from this experiment. Clearly it is to be avoided if possible, especially in view of the possibility of cochlear degeneration described by other authors. Evidence of cochlear degeneration from perilymph loss, from exposure of perilymph to mucosa or from stimulation injury was not encountered. The cochlea nevertheless has a very low "damage threshold" in comparison with the vestibular labyrinth and is far more liable to injury.

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