ELSEVIER

Hearing Research 76 (1994) 101–117

HEARING RESEARCH

# Anatomical effects of impact noise

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(Received 14 August 1993; Revision received 26 January 1994; Accepted 31 January 1994)

**Abstract**

Four groups of binaural chinchillas were exposed to impact noise (B-duration = 200 ms) ranging from 119 dB to 137 dB peak equivalent SPL at repetition rates of 1/s or 4/s. The duration of exposure was adjusted so that each exposure consisted of equal acoustic energy. Animals were then sacrificed immediately, 24 h or 30 days after the exposure and their cochleas subjected to scanning electron microscopy. For exposures of 119 dB or greater, there appeared to be direct mechanical damage, including large clefts between the third row of outer hair cells and Deiters' cells and fracture of tight cell junctions at the reticular lamina. There was also a progressive increase in cochlear damage over the 30 days of recovery. The patterns of cochlear pathology are compared with hearing losses and cochleograms of chinchillas previously subjected to similar exposures and with results of studies using higher level impulse noise. The results are discussed in terms of 'critical level' for impact and impulse noise.

*Key words:* Impact noise; Noise induced hearing loss; Cochlear pathology

## 1. Introduction

Impact noises are the sounds produced when two hard surfaces are forcefully brought together, such as the sounds produced during rivetting, hammering and forging. Impact noise is often an important and potentially harmful part of the noise profiles found in industries. In industrial settings, the levels of impact noises typically range from 95 to 135 dB(A) and acoustic energy is often located between 0.5 and 3 kHz (Martin, 1976). Industrial settings with both impact and background continuous noise may lead to larger and more rapidly developed hearing losses than would be predicted on the basis of the total energy in the exposure (Passchier-Vermeer, 1983; Ceypek et al., 1973).

Impact and impulse noise are not adequately covered in our current noise standards. The current Occupational and Health standards (OSHA, 1983) set the upper limit at 115 dB for continuous noise and at 140 dB for impact/impulse noise, thereby introducing a range of 25 dB that is not adequately described in noise standards. However, 115 to 140 dB is the range where many industrial impacts occur. Furthermore, the application of the equal energy principle to impulse/impact noise (Martin, 1976) is questionable (Henderson and Hamernik, 1974) because the effects of high level impulse or impact noise on the cochlea may be different than the effects of exposure to continuous noise.

One indication of the differential effects of continuous and impulse/impact noise is the non-linear recovery of hearing following exposure to impulse or impact noise. Luz and Hodge (1971) and later, Henderson et al. (1974) and Hamernik et al. (1988) reported that, after exposure to impulse noise at 155 to 160 dB, the large hearing loss begins to recover immediately; by 0.5 to 1 hour, the recovery stops and the hearing loss begins to grow again and reaches a second peak at 5 to 12 h; and finally, hearing begins to recover again to some stable level. The triphasic recovery curve has been assumed to reflect mechanical damage to the cochlea.

In another set of publications, Hamernik et al. (1984a,b) demonstrated the mechanical damage and documented the sequence of changes in the chinchilla

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SSDI 0378-5955(94)00033-M

cochlea following exposure to 160 dB impulses. Immediately after the exposure, strips of the sensory epithelium were found ripped from the basilar membrane. 24 h after the exposure, sensory cells were found swollen and stereocilia were fused and distorted and by thirty days, the pathology had stabilized with a proliferation of undifferentiated cells (probably Claudius) covering the basilar membrane in the region of damage.

Spoendlin (1976) hypothesized that noise exposures above a 'critical level' damage the cochlea by direct mechanical destruction. In his article, Spoendlin showed that in cats, sensory cell edema occurs after exposure to high level noise bursts of 120 dB for 200 ms. Since the Spoendlin report, it has become clear that the critical level varies with the waveform of the impulse/impact, i.e. approximately 120 dB for noise bursts (Spoendlin, 1976) and approximately 155–160 dB for 1 ms impulses (Hamernik et al., 1984a). The critical level for impact noise is likely to be related to the duration of the impact as well as the spectral content.

In a study on the importance of peak amplitude of impact noise in the development of hearing loss, Henderson et al. (1991) reported evidence for 'critical level' and non-monotonic recovery curves. Six groups of chinchillas were exposed to impact noise that had a duration of 200 ms and a level in the range of 107 to 137 dB peak equivalent (p.e.) SPL. The durations and amplitudes of the exposures were adjusted so that each of the experimental groups was exposed to the same total acoustic energy. For levels of 107 to 119 dB, there was little permanent threshold shift (PTS) or hair cell loss; however, as the exposure level was increased above 125 dB, there was an abrupt increase in PTS and hair cell loss (Figs. 1 and 2). For many of the exposures, the recovery of hearing was non-monotonic with the greatest hearing loss occurring at 8 h after the exposure. For the 200 ms broad band impact the critical level is approximately 125 dB. The results of the Henderson et al. (1991) study provide a reference to help evaluate for the present set of experiments. Even though there were fewer subjects used in the time-invasive scanning electron microscopy (SEM) study, the subjects had the same exposure as the subjects in the larger population study. Thus, the larger study provides a perspective on the expected mean and variance of the effect.

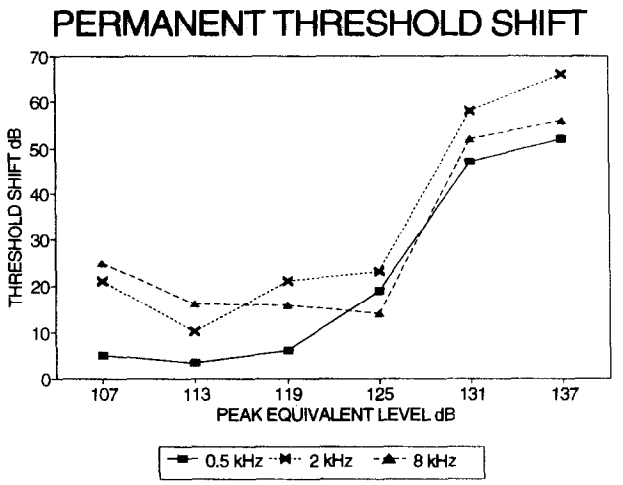



Fig. 1. The average permanent threshold shift (PTS) at 0.5, 2, and 8 kHz for groups of chinchillas exposed to impact noise ranging from 107 to 137 dB 1/s (Henderson et al., 1991).

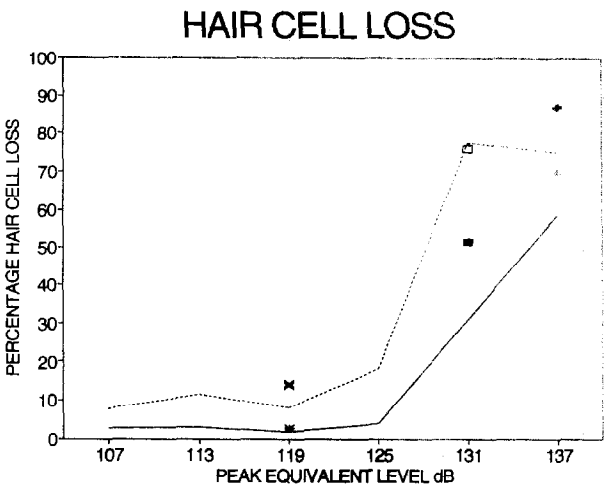



Fig. 2. Average percent hair cell loss for the groups of chinchillas presented in Fig. 1. OHC (dotted line) and IHC (solid line) in the 6 groups of animals exposed to impact noise at levels ranging from 107 to 137 dB 1/s (Henderson et al. 1991). Symbols represent data from animals in the present study. Cross (3674 OHC), Asterisk (3674 IHC), Empty Square (3614 OHC), Filled square (3614 IHC), Plus (3567 OHC) and Filled triangle (3567 IHC).

The aim of this study was to understand the cochlear pathology underlying the hearing loss from impact noise. Specifically, (a) what are the changes in the cochlea at different recovery times and do these changes parallel the non-linear recovery in hearing? and (b) are there evidences of mechanical damage following exposures to impact noise? (c) Is the 'critical level' reflected in differences in pathological changes associated with exposures to relatively low (119 dB) and relatively high level (137 dB) level exposures?

## 2. Methods

*Subjects*

Twenty-one healthy, binaural adult chinchillas were used as subjects. Six subjects served as controls and fifteen were exposed to impact noise. Each subject was free of middle ear disease.

All procedures involving care and handling of the chinchillas were reviewed and approved by both the University Animal Care Committee and by the NIOSH study section.

*Noise exposure*

One or two animals at a time were placed in small separate cages (31 × 51 × 38 cm) located below a loudspeaker and were given free access to food and water. The animals were exposed to electronically synthesized impact noise. The impact consisted of a burst of broadband noise, whose spectrum was shaped with an equalizer, so that it roughly approximated that of an impact generated by striking a steel plate with a hammer. The electrical signal was generated by multiplying (Analog Devices AD5354) spectrally-shaped noise with the output from an exponential waveform generator. The waveform generator controlled the repetition rate and envelope of the synthesized impact. The signal was amplified and transduced by a loudspeaker. The duration of the impact at a point 20 dB down from the peak was 200 ms and the nominal rise time of the signal was 12 ms (see Henderson et al., 1991). Noise levels were measured using a sound level meter (Larson and Davis, LDL 825) and a condenser microphone (Larson and Davis, LDL 2559). The microphone was positioned within the cage at the level of the animal's head.

Table 1 shows the exposure conditions. The peak level and duration of exposures/number of impacts were adjusted so that each exposure condition consisted of equal energy. In the original experiment (Henderson et al., 1991), each of these groups consisted of five subjects. In this experiment, there were at least three subjects at each level.

*Histology*

The animals were anesthetized with sodium pentobarbital and decapitated. Each bulla was immediately removed from the skull and the ossicles disconnected and removed and the round window membrane perforated. The cochlea was slowly perfused through the round window with cold 2.5% glutaraldehyde in 0.05 BES buffer (pH 7.4) and immersed in the same type of fixative for the next 24–48 h. Then, the cochleas were rinsed in veronal acetate buffer (Zetterquist, pH 7.4), post-fixed with 1% osmium tetroxide in veronal acetate buffer for half an hour, rinsed again in buffer, distilled water and 50% ethanol. The cochleas were stored in 70% ethanol.

Table 1

| Exposure level (p.e. SPL) | Rate | Duration in hours | Number of subjects [a] | [b] |
|---|---|---|---|---|
| 119 dB | 1/s | 30 | 5 | 3 |
| 131 dB | 1/s | 1.8 | 5 | 6 |
| 131 dB | 4/s | 0.47 | 5 | 3 |
| 137 dB | 1/s | 0.47 | 5 | 3 |
| Control (no exposure) | | | | 6 |

Table showing the experimental conditions and the number of subjects in each condition in the Henderson et al. (1991) [a] study and in the present [b] study.

Dissection of cochleas was performed while they were fully covered in 70% ethanol and the round and oval windows protected with boxing wax to minimize the entry of bone dust into the cochlea during bone drilling process. The excess bone was thinned closely to the otic capsule shape. The thinned bony capsule was picked away in small pieces with a sharp mini-blade beginning at the apex and proceeding toward the base for approximately one and three quarter turns. The Reissner's membrane, the stria vascularis, part of the spiral prominence and spiral ligament were removed. The apical turn of the cochlea was separated from the rest of the cochlea by first making a radial cut across the organ of Corti as far as the modiolus. The bony modiolus was picked away and the central core of nerve fibers cut with scissors to release the apical and part of middle turns. A fine tipped micropipette was used to gently flush ethanol over the organ of Corti to remove any remaining fragments. At this point, the cochlea was trimmed so that the region of 0.8 to 5 kHz was visible.

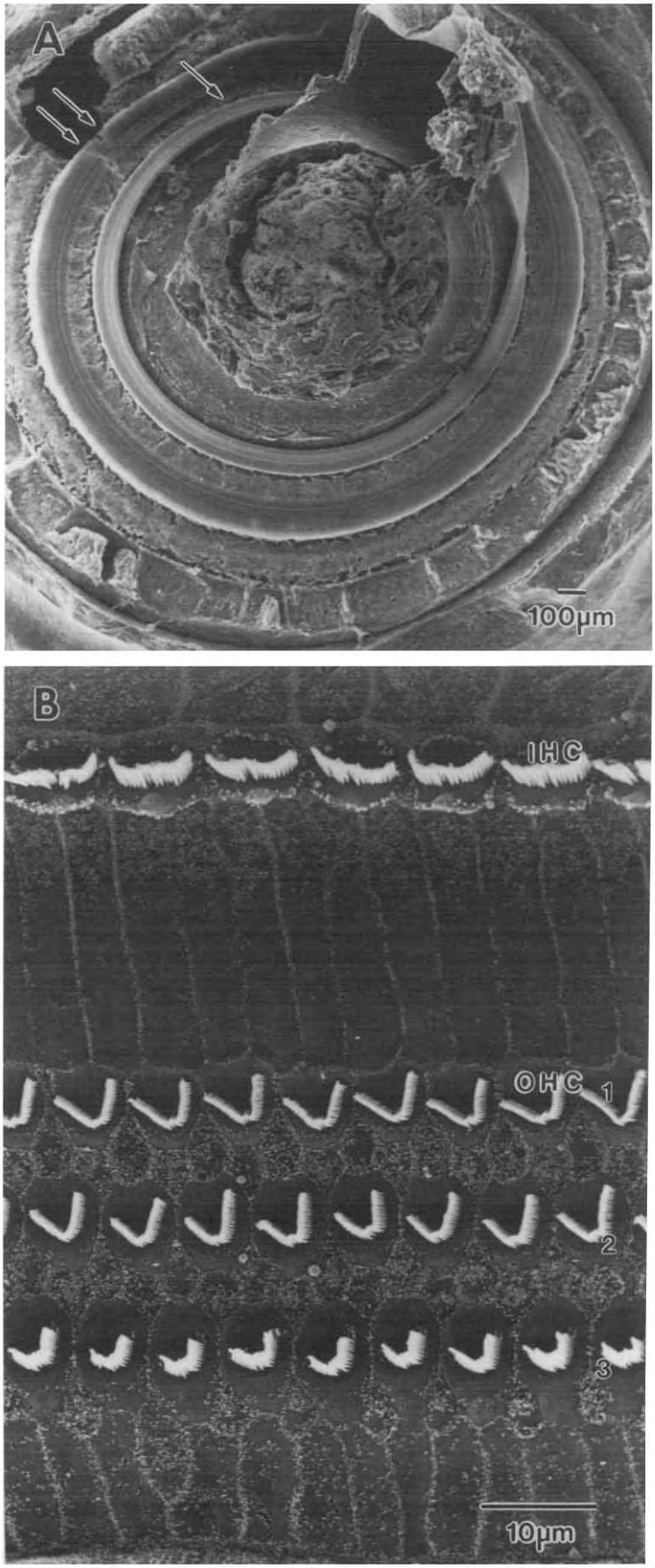
The drying procedure for SEM viewing started first with re-hydration of cochleas in order to proceed with the OTO bridging method suggested by Malick and Wilson (1975). Specimens were then rinsed in acetone and critical point dried in liquid $CO_2$. They were mounted on aluminum stubs using epoxy and coated with colloidal graphite (isopropanol base). The mounted specimens were sputter coated with evaporated gold to a depth of approximately 22 nm and examined on a field emission SEM using accelerating voltages of 15 or 25 kV. A total of 22 noise-exposed cochleas and six control cochleas were processed and viewed using a Hitachi S-800. Noise-exposed cochleas were usually processed in groups of three or four, along with a control cochlea.

*Single micron sections*

After the SEM procedure, the specimens were removed from the stub and immersed in 100% ethanol for 30 min., in propylene oxide for 2 h and gradually infiltrated with the low viscosity embedding medium, Spurr. Embedding was done in beem capsules allowing Spurr to polymerize overnight at 70°C. Serial 1 μm radial sections of the selected areas were placed on the microscopic slide and stained using 1% methylene blue/azure II mixture.

## 3. Results

Of the possible 42 ears (21 subjects), SEM was performed on 28 ears (22 experimental and 6 control). Two of the cochleas from the experimental group were re-embedded in plastic to allow 1 μm sectioning of interesting lesions. The SEM studies were focused on


A
100μm
B
IHC
OHC 1
2
3
10μm

the areas maximally damaged by the exposure, approximately 7 to 13 mm from the apex i.e., the frequency regions of 0.8 to 5 kHz (Eldredge et al., 1981).

*Control*

Fig. 3 shows low (A) and high (B) power views of a control cochlea. The low power micrograph provides a view of the 0.8 to 5 kHz region. Since one of the issues under investigation is the possibility of direct mechanical failure, it is important be able to differentiate the types of tissue artifacts that are found in non-exposed cochleas from pathologies caused by exposure to noise. The two most commonly occurring artifacts are a longitudinal crack along the margin of the spiral limbus (Fig. 3A, one arrow) or radial cracks across the organ of Corti (Fig. 3A, two arrows). The nature of cracks seen in the control samples are clearly different from those seen in ears exposed to impact noise i.e., control ears do not show longitudinal cracks along the Hensen-Deiters' cell margin or cracks between outer pillars and first row of outer hair cells (OHC). In addition, in the control animals, the tissue preparation left the stereocilia in the proper position and looking normal (Fig. 3B). Thus, the observations of stereocilia defects or cracks at the Hensen-Deiters' cell margin can be interpreted as consequences of the exposures.

### 3.1. Effect of impact noise exposures

To put the SEM results of the current experiment into perspective, hearing loss and hair cell loss from previous studies of the same exposure (Henderson et al., 1991) are presented in Figs. 1 and 2. For the subjects used in this experiment, their total hair cell loss was computed from the surface views of the SEM for regions from 0.8 to 5 kHz and from the light microscopic analysis of the region below 0.8 kHz and above 5 kHz. It can be seen that the subjects used in the 30 days post-exposure SEM studies have approximately the same hair cell losses as the average hair cell loss in the previous study. Thus the assumption is that the pathologies seen in the current SEM study are typical of the pathologies seen in the larger, light microscopic study of impact noise.

*119 dB exposures*

These exposures were at the rate of 1/s and lasted 30 h. The long duration complicates the interpretation of the data because it is difficult to estimate the time when the actual damage occurred. If the pathology is related to mechanical trauma that may occur early in the exposure, then the micrographs may reflect mechanical damage as well as subsequent degeneration. If the pathological process is related to metabolic factors, then the damage is likely to be cumulative over the duration of the exposure.

Immediately after the exposure, two areas of damage were seen in the cochlea of chinchilla No. 3657. Fig. 4A shows the 1 kHz region with a few missing OHCs. A few other OHCs exhibit stereocilia defects. The OHCs bordering the small areas of OHC lesion appear normal. The inner hair cells (IHCs) are also essentially normal. Fig. 4B shows the 2.5 kHz region where there is more extensive damage with dramatic changes in the OHC stereocilia. The IHC stereocilia are intact, but slightly splayed.

Fig. 4C and D are from the 2.5 kHz region of a chinchilla (No. 3675L) sacrificed 24 h after the 119 dB exposure. Of special interest in Fig. 4C is the 0.19 mm cleft between the outer pillar cells (OPCs) and the OHCs. The OHCs and Deiters' cells have degenerated and the IHCs show stereocilia defects. Fig. 4D is from the area adjacent (apical) to the cleft and shows a near complete loss of OHCs and sporadic loss of IHCs. The presence of the cleft in specimen in Fig. 4C is not likely to be a consequence of the degeneration over the 24-h period, instead, it may be due to the individual variability.

Fig. 5 is from opposite ear of chinchilla (No. 3675R) sacrificed 24 h after the 119 dB exposure. The basal and apical boundaries of a 0.215 mm punctate lesion in the 2 kHz region may be seen figures 5A and B respectively (see double arrows). The punctate lesion (region of cochlea between the double arrows, but not shown) involves a total loss of the IHC and pillar cells, as well as OHC. Presumably, the Hensen/Claudius cell region expands and covers the cochlear partition with simple epithelial cells (Hunter-Duvar, 1987). The inner sulcus region appears to be more active with a proliferation of microvilli as well as appearances of cells that are likely to be involved in endocytosis (see arrow). Some relatively normal looking OHC stereocilia may be seen in the area adjacent to the lesion (Fig. 5A). A few slightly disarrayed IHC stereocilia are visible in Fig. 5B. Fig. 5C shows the magnified view of distorted and injured IHC stereocilia at the edge of the lesion, seen in Fig. 5B. Fig. 5D is from a more apical lesion in the 1 kHz region, showing a damage primarily in the third row of OHCs.

The differences between the left and right ears of subjects No. 3675 are highlighted by comparing Figs. 4C and D with Figs. 5 A–D. The right ear (Fig. 5) did

Fig. 3. (A) Low power view of control chinchilla cochlea showing the typical field of SEM analysis (approximately 7.4 $\mu$m to 13.6 $\mu$m (1–5 kHz) from the apex). Note the position of minor drying artifacts (arrows). (B) Higher power view showing the status of inner (IHC) and outer hair cells (OHC) with stereocilia. 1,200 X (original magnification).

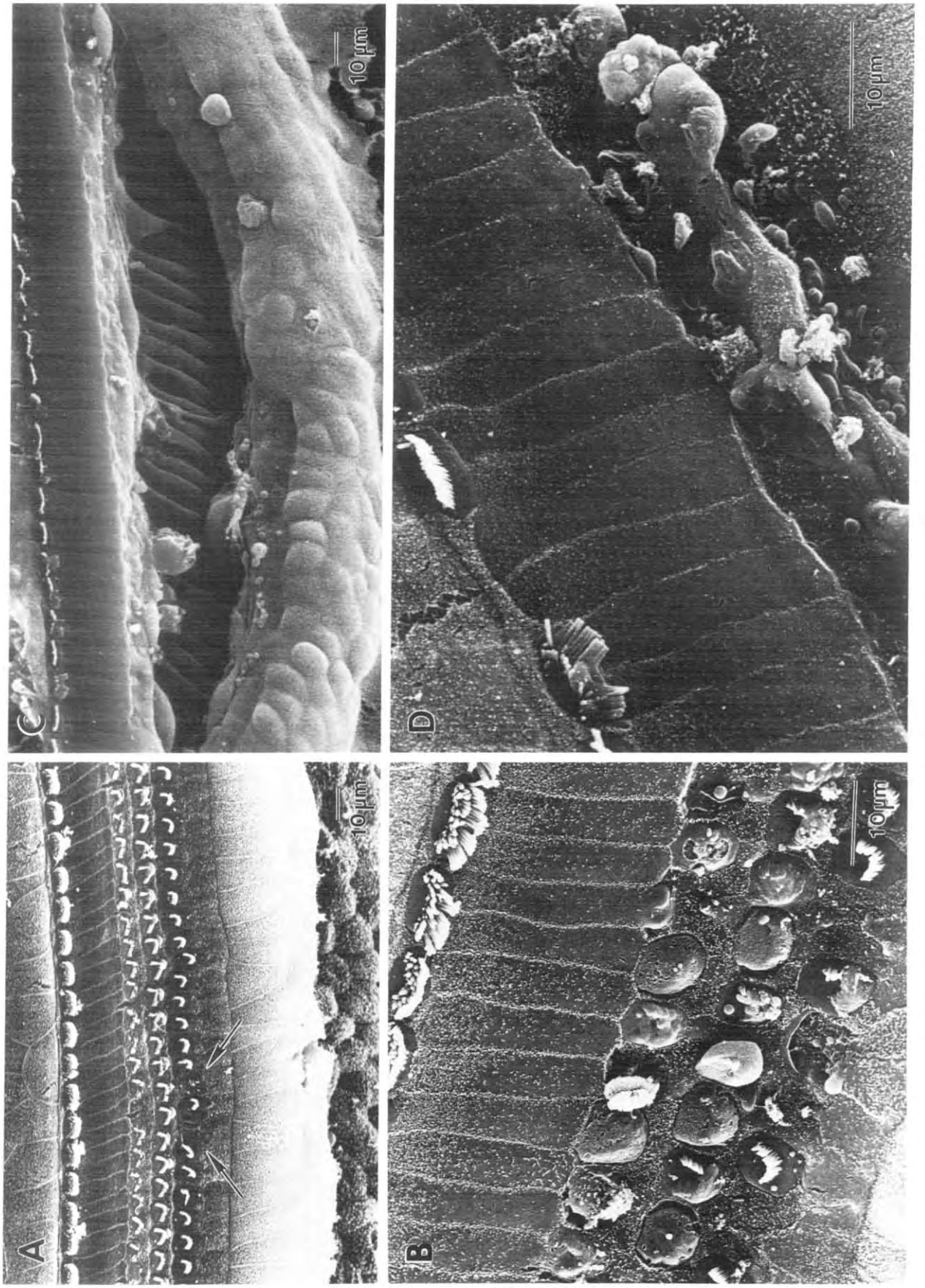



Fig. 4. Surface views of chinchilla cochlea (3657R) immediately after exposure to 119 dB impacts at 1 kHz (A) with small focal lesions (arrows) (B) the same cochlea but in 2.5 kHz region where there is more extensive OHC damage. Panels (C) and (D) are from another chinchilla (3675L) sacrificed one day after the 119 dB exposure. The 2.5 kHz region (C) has a 0.19 mm long cleft between the outer pillar cells and Deiters' cells. (D) shows the absence of OHCs and distorted IHC stereocilia at the region of the cleft.

not have the cleft between the pillars and OHCs (Fig. 4C and D), but the left ear had regions of 'mature' looking lesions that spread from the Hensen/Claudius region to the inner sulcus. The appearance of such 'mature' lesions is somewhat surprising given that Bohne (1976) has reported that scars form five days after the damage. However, the actual beginning of the scar formation and spread of Hensen/Claudius cells is difficult to pinpoint because the exposure was 30 h long and the subject was sacrificed 24 h after the exposure. Also, recent work by Raphael and Altschuler (1991) suggests that the beginning of scar formation may begin hours after damage from ototoxicity.

Fig. 6A shows a nearly normal organ of Corti from the 4 kHz region in an animal (No. 3674) sacrificed 30 days after the exposure. In contrast to the 4 kHz region, the 5 kHz region shows blister-like lesions on the top of the pillar cells (Fig. 6B). These blister-like lesions extended basalward for about 1 mm along the organ of Corti.

Fig. 2 shows that the cochleogram for chinchilla No. 3674 is close to the average hair cell losses sustained by

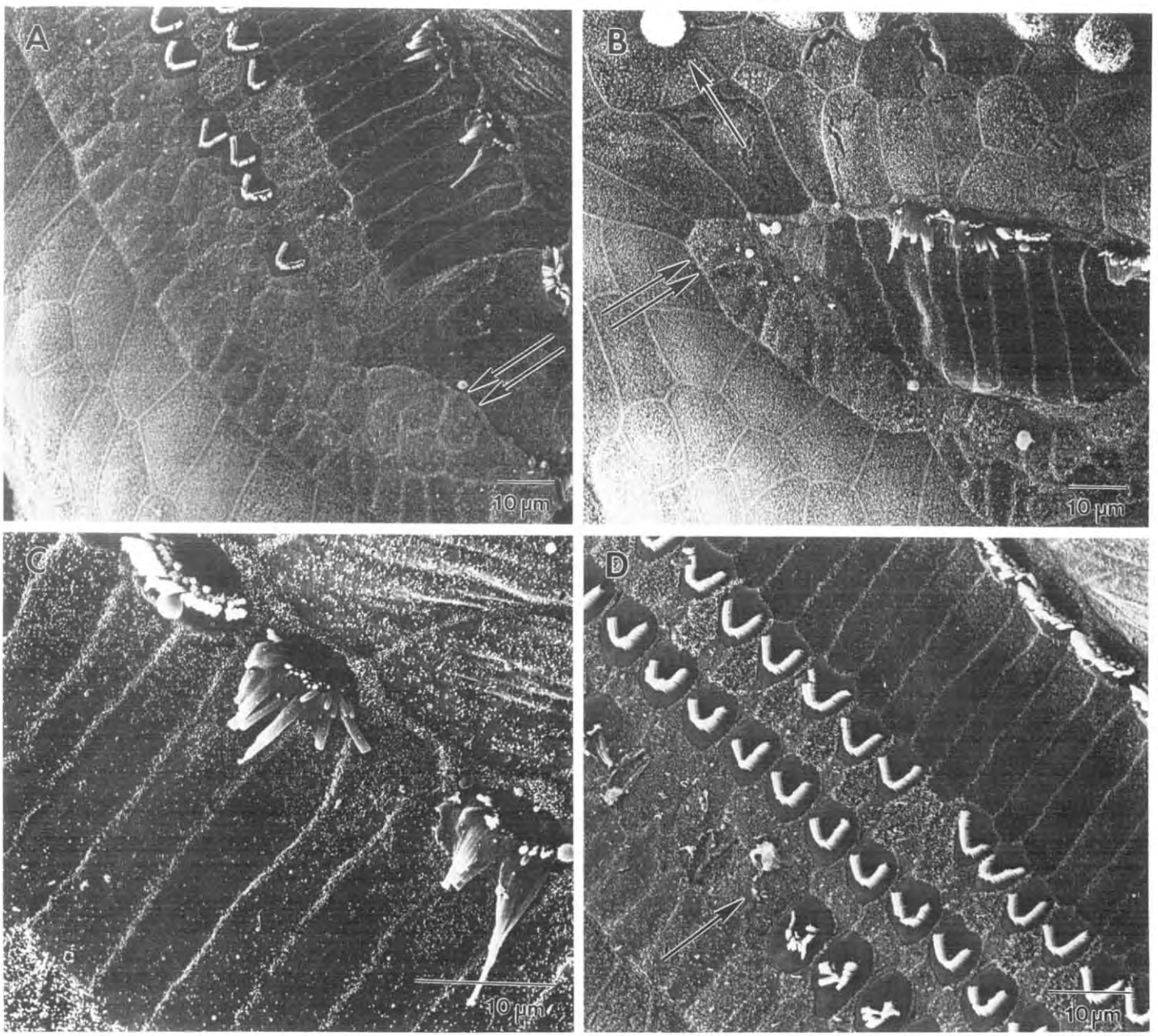



Fig. 5. Basal (A, two arrows) and apical (B, two arrows) margins of 0.215 mm lesion that involves a complete loss of the IHCs, pillars and OHCs seen in a chinchilla (No. 3675R) 24 h after exposure at 119 dB. (C) IHC stereocilia defects at the basal margin of the lesion; and (D) apical side of a lesion where damage is located in the third row of OHC. Arrow shows the complete loss of HCs.

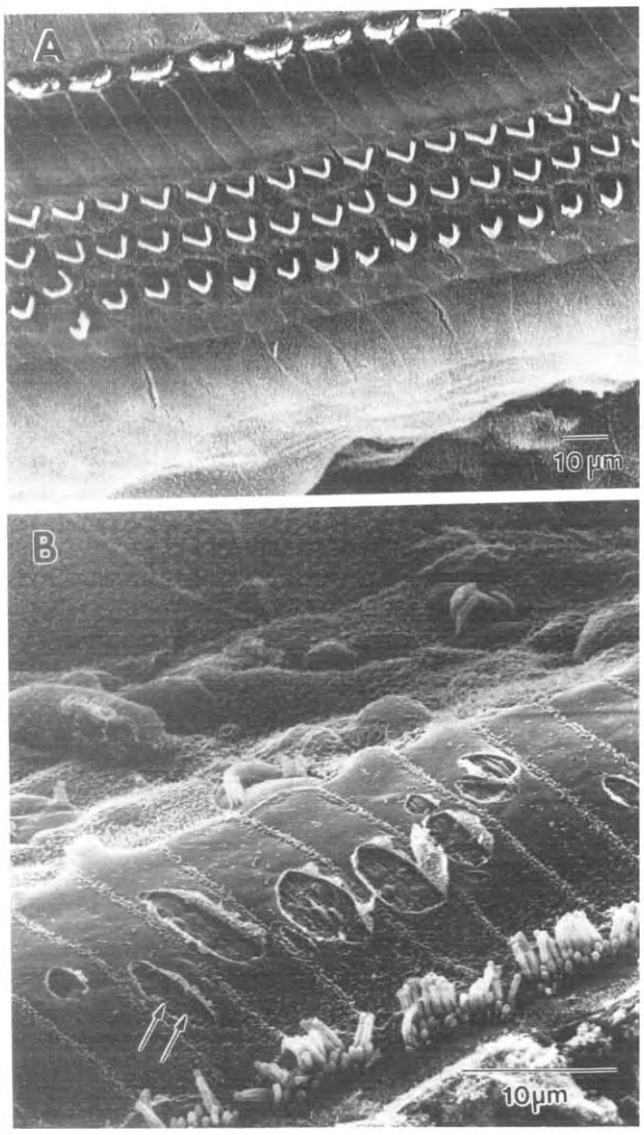



Fig. 6. Thirty days after the 119 dB exposure, (A) the organ of Corti of chinchilla No. 3674 looks normal overall, but is damaged with ruptured inner pillar heads (two arrows) in the 5 kHz region (B).

chinchillas exposed to the same noise in the study by Henderson et al. (1991). Furthermore, from Fig. 1, it is reasonable to expect that their hearing loss should be in the vicinity of 5 dB and 20 dB at 0.5 kHz and 2 kHz, respectively.

*131 dB 1 / s exposures*

Results from the study by Henderson et al. (1991) suggest that 131 dB is above the 'critical' level, with the 131 dB exposure, there is an abrupt increase in the hearing as well as hair cell loss, when compared to the 119 dB or 125 dB exposures (see Figs. 1 and 2). Exposures to impact noise at 131 dB could produce PTS of 40 to 50 dB from 0.5 to 8 kHz (Fig. 1). Given the severity of the exposure, the surface of the organ of Corti looks relatively normal immediately after the exposure (Fig. 7). Figs. 7A and B show the only clear pathological change. Although the extent of the lesion was limited to a length of 8 to 12 OHC, the damage was quite severe. The tops of the Deiters' cells appear to be 'ballooning' at the reticular lamina, tight cell junctions between the tops of the pillar cells (Fig. 7A and B, arrowhead), the cuticular plates (Fig. 7A and B, two arrows) and the reticular lamina are fractured creating an opening to the spaces of Nuel and the stereocilia of the remaining hair cells appear to be distorted. Figs. 7C and D are from tissue bordering the lesion where the most obvious pathology is a splaying of IHC stereocilia. The size of the lesion seen immediately after the exposure is likely to be an underestimation of the eventual size of the lesion if the subjects were allowed to 'recover' for 30 days.

At 24 h post exposure, the damage (chinchilla No. 3594) was much more dramatic and extensive. Figs. 8A, B and C span the region of 0.9 kHz to 2 kHz. The radial crack in Fig. 8B is a drying artifact. Figs. 8D and E are higher power views and illustrate some of the pathological changes including fused stereocilia, missing OHCs, (some OHCs are still present, two arrows) splayed IHC stereocilia and ruptures of the margins of the pillar cell and reticular lamina. There also appears to be a proliferation of microvilli covering the reticular lamina. Note the more severe and extensive pathology manifested in a subject sacrificed 24 h post-exposure compared to the small, punctate lesions found immediately after the exposure (Fig. 7A and B).

Subjects that were sacrificed at thirty days post-exposure showed a number of significant changes. First, a large extent of the basilar membrane shows missing OHCs was seen in subject No. 3614 (Fig. 9A). Cells of unknown origin showing proliferation of microvilli, possibly involved in endocytosis are also seen (Fig. 9A, asterisk). Fig. 9B is a 1 $\mu$m radial section through a tissue with pathology similar to that seen in Fig. 9A. This section confirms that the IHC is present (arrow). However, note the diminished density of nerve fibers (double arrows). The pillar cells are present, but the OHCs and Deiters' cells 1 and 2 are completely gone. Remnants of Deiters' cells in row 3 appear to connect the reticular lamina with Hensen cell layer (arrowhead). Fig. 9C shows that the space originally occupied by the OHCs and Deiters' cells, is covered by an epithelial cell layer which spans the distance from the stria vascularis to the inner sulcus. Fig. 9D shows the margin of the most severe lesion (1.8 kHz). While many of the IHCs are present, their stereocilia are distorted and fused. These photomicrographs are from subject No. 3614. To put these pictures into perspective, the cochleogram from No. 3614 is plotted with the average cochleogram of subjects from the previous experiment, exposed to the same noise (Fig. 2). It should also be

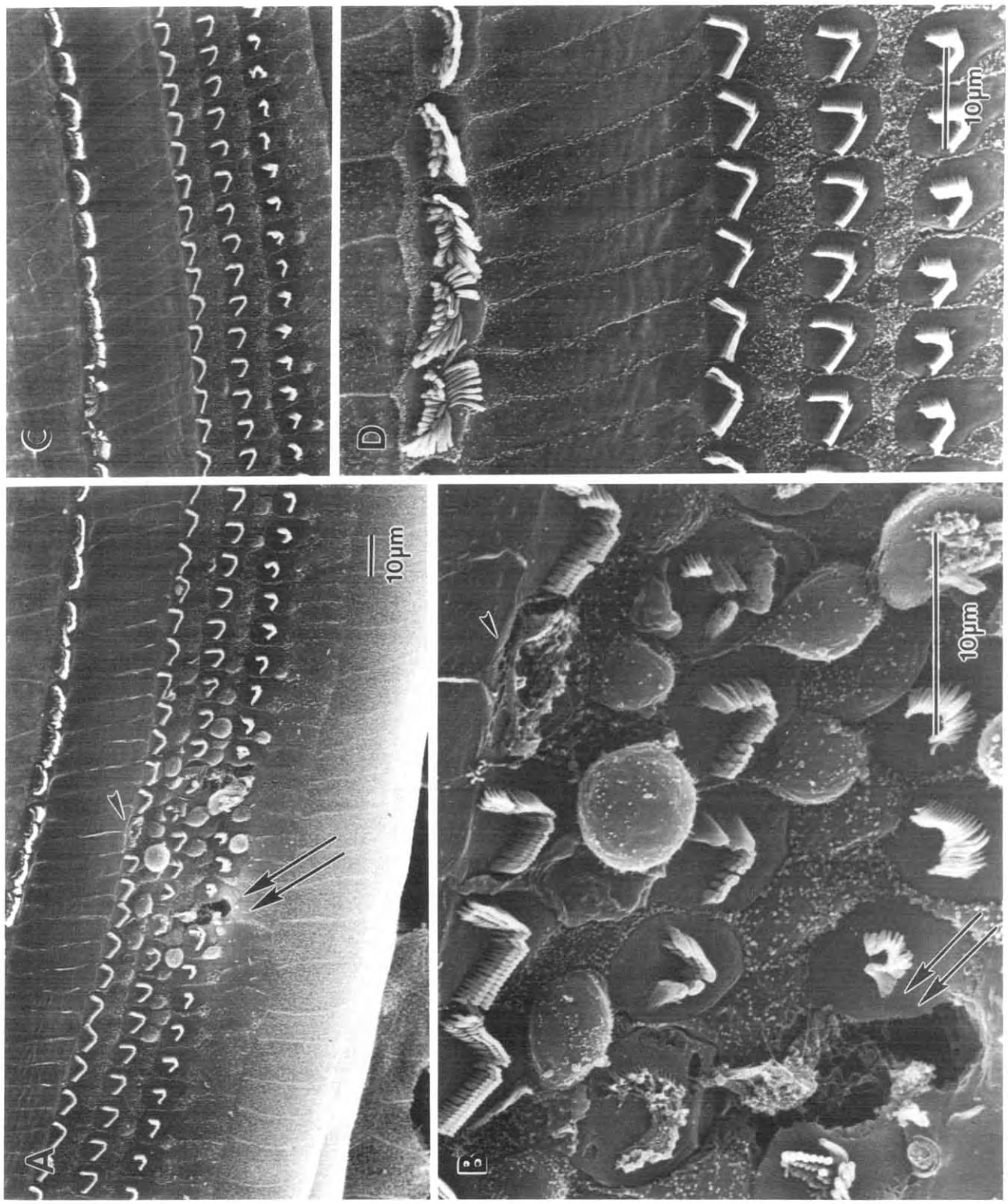
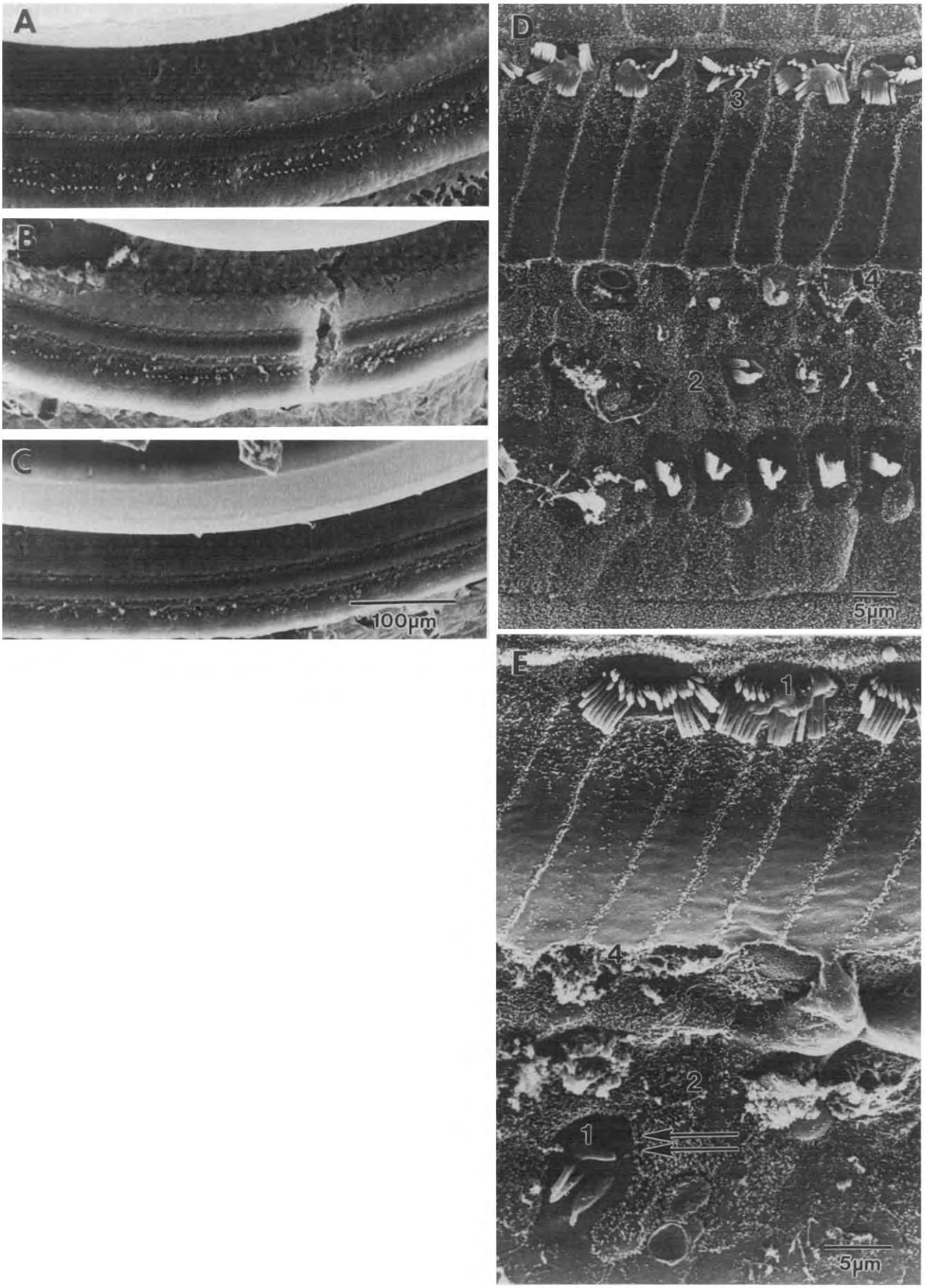



Fig. 7. (A) Low and (B) high power view of small 0.04 mm lesion in the 1 kHz region immediately after 131 dB exposure. OHC1 damage (arrowhead) and advanced OHC3 damage with holes in the reticular lamina are present (two arrows) (No. 3574). (C) and (D) are from the more apical region (0.8 kHz) and show relatively normal OHC and splaying of IHC stereocilia.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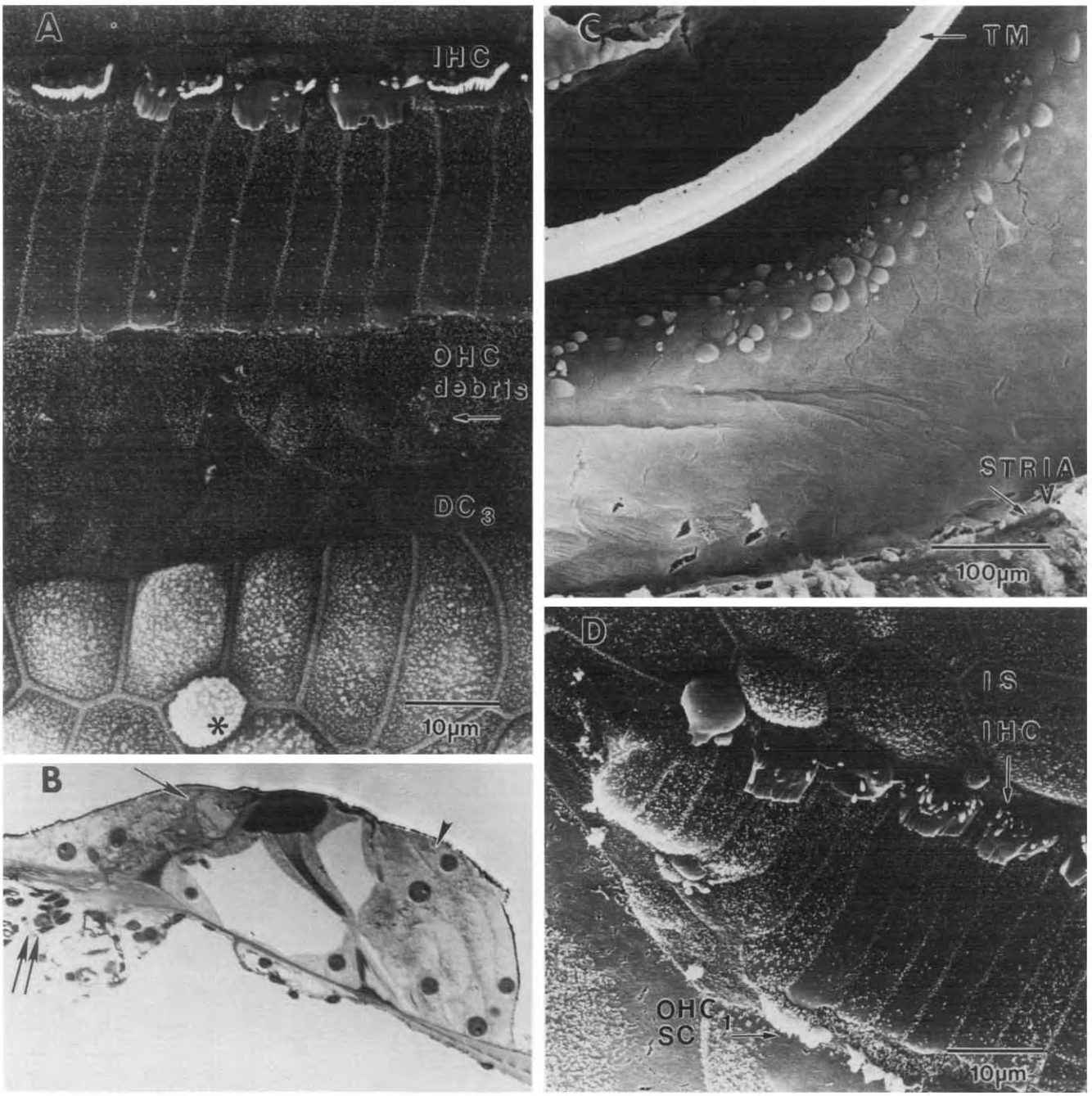



Fig. 9. Surface of basilar membrane 30 days after exposure to 131 dB (A) shows intact IHC and pillars and missing OHC in the 1 kHz region. (B) 1 $\mu$m cut through the cochlea in the region similar to that shown in (A). OHCs are absent but IHC is still present (arrow). Note the reduced density of nerve fibers (two arrows) and proliferation of Hensen cells (arrow head). (C) organ of Corti exposed for SEM has almost complete degeneration of OHC's and replacement with epithelial cells in the 4 kHz region. (D) 1.8 kHz region shows a margin of degeneration of OHC with some OHC1 stereocilia present (3614L).

Fig. 8. (A,B,C) 24 h after exposure to the 131 dB impact noise extensive pathologies from 0.9 to 2 kHz appear. (D) and (E) are higher power views. (1) fused stereocilia, (2) missing OHC, (3) splayed IHC stereocilia (4) rupture of tight cell junction of pillar cells (No. 3594). Double arrows point to an OHC with fused stereocilia, in row 3, amidst missing OHCs.

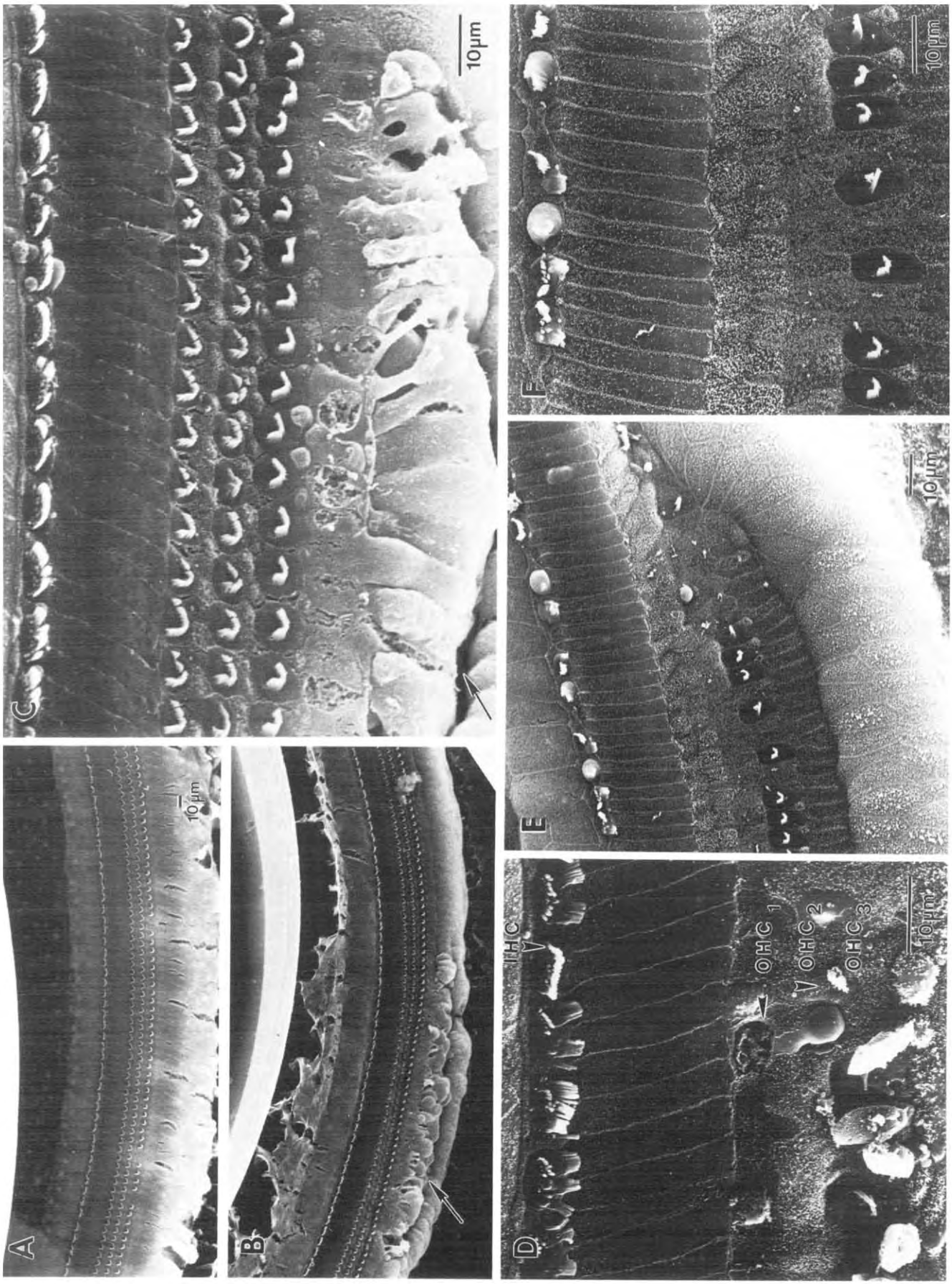



Fig. 10. (A) A control cochlea (No. 3631); (B) and (C) show small outer hair defects and mechanical lesions at the margin of the Deiters'/Hensen's cell region immediately after exposure to 131 dB at rate of 4/s (No. 3632). (D) 24 h later the pathology is greater and involves larger region of cochlea (No. 3610). (E) and (F) 30 days after the exposure, the pattern of pathology had stabilized with extensive regions of missing hair cells (No. 3612).

noted that in the previous experiment this exposure produced an average PTS of 50 dB at 2 kHz (Fig. 1).

*131 dB 4/s exposures*

In an earlier study of impact noise, Henderson et al. (1991) had shown that the faster rate of presentation (4/s) produces more hair cell loss and hearing loss than exposures at the slower, 1/s rate. The 131 dB exposure was repeated with a higher rate of presentation (4/s). Fig. 10A is from a control subject (No. 3631). Figs. 10B and C show a low power view of the cochlea immediately after the exposure (No. 3632). These figures show clefts at the Deiters'/Hensen's margin in the 1.5 and 3 kHz regions. It is noteworthy that the 131 dB exposure produces more severe lesions when presented at a rate of 4/s than at 1/s. However, the data from the 4/s exposures are similar to those from the 1/s exposures (Fig. 7) in that the pathological changes are greater at 24 h post-exposure (Fig. 10 D, subject No. 3610) than immediately after exposure (Fig. 10B). The differences between the extent of damage at 0 and 24 h is probably not as great as the surface perspective indicates. It is likely that many of the cells are damaged even in the animals sacrificed immediately, but the damage is not reflected in the surface view. The lesion in the 1 kHz region (Fig. 10D) shows missing OHCs and fused and splayed IHC stereocilia with one IHC with relatively normal stereocilia (arrowhead). By thirty days (No. 3612), the cochlea shows extensive lesions with missing OHCs in rows 1 and 2 and missing IHCs or deformations on the IHC stereocilia (Fig. 10E and F).

*137 dB exposures*

The 137 dB exposure caused severe mechanical damage ((No. 3581), including an actual ripping of the organ of Corti off the basilar membrane (Fig. 11A, arrow) as well creating large clefts at the margin of the Deiters' and Hensen's cells exposing the Deiters' cell and exposing the Deiters' cell bodies in row 3 (DC) (Fig. 11B). Figs. 12 and 13 show the status of the left and right cochleas of the another animal sacrificed 24 h after the 137 dB exposure. Both cochleas (No. 3617) show a large piece of the organ of Corti ripped from

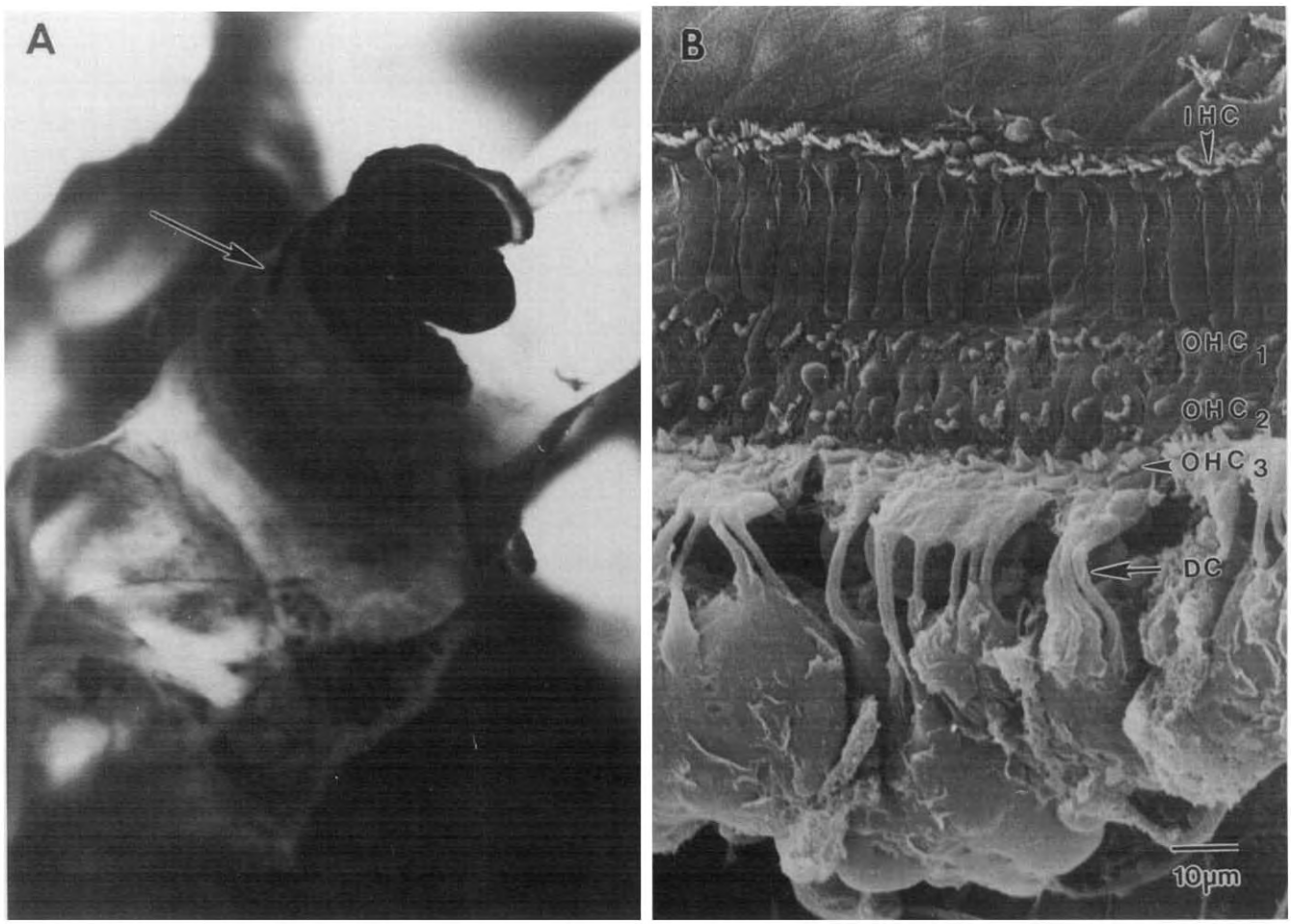



Fig. 11. (A) A low power view showing ripped organ of Corti (arrow) immediately after 137 dB exposure. (B) SEM view of 0.9 kHz region showing extensive stereocilia defects and a large rip at the third row of OHC (No. 3581).

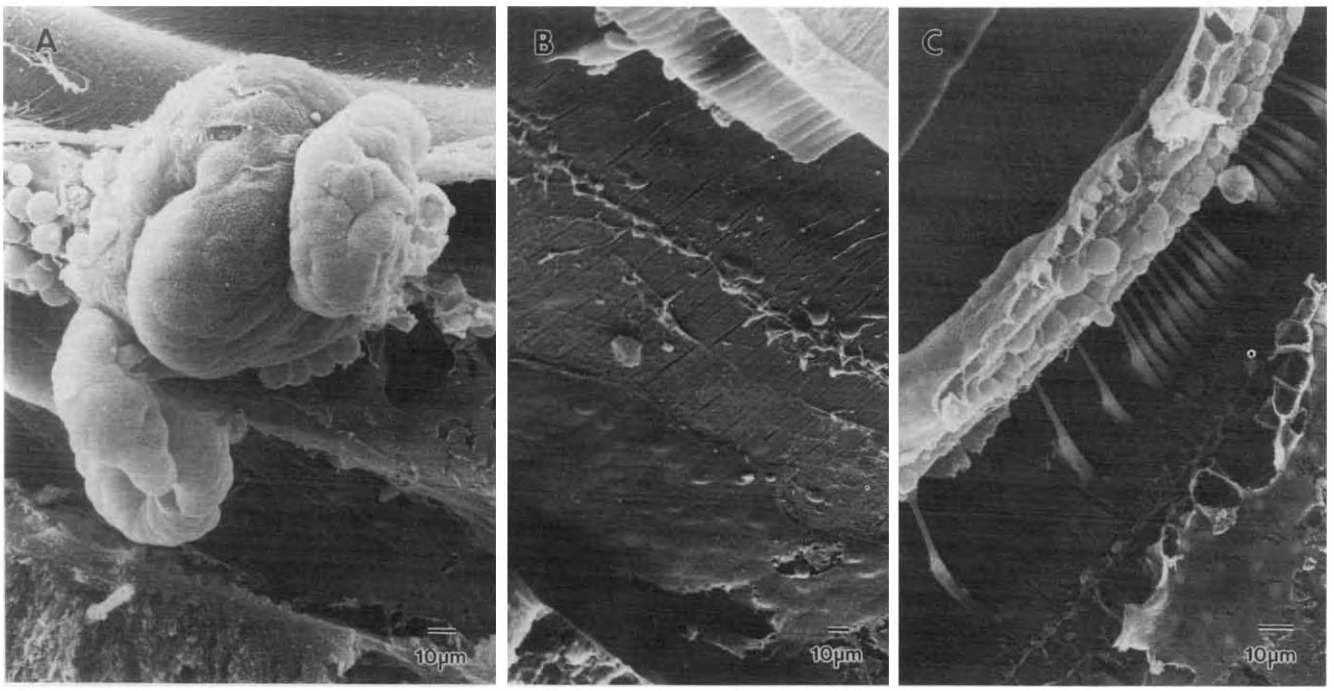



Fig. 12. (A) The ripped and degenerating organ of Corti from the left cochlea 24 h after exposure to 137 dB (B and C) show details of missing pillar cells and basilar membrane without OHC, Deiters' cells and Hensen's cells (No. 3617L).

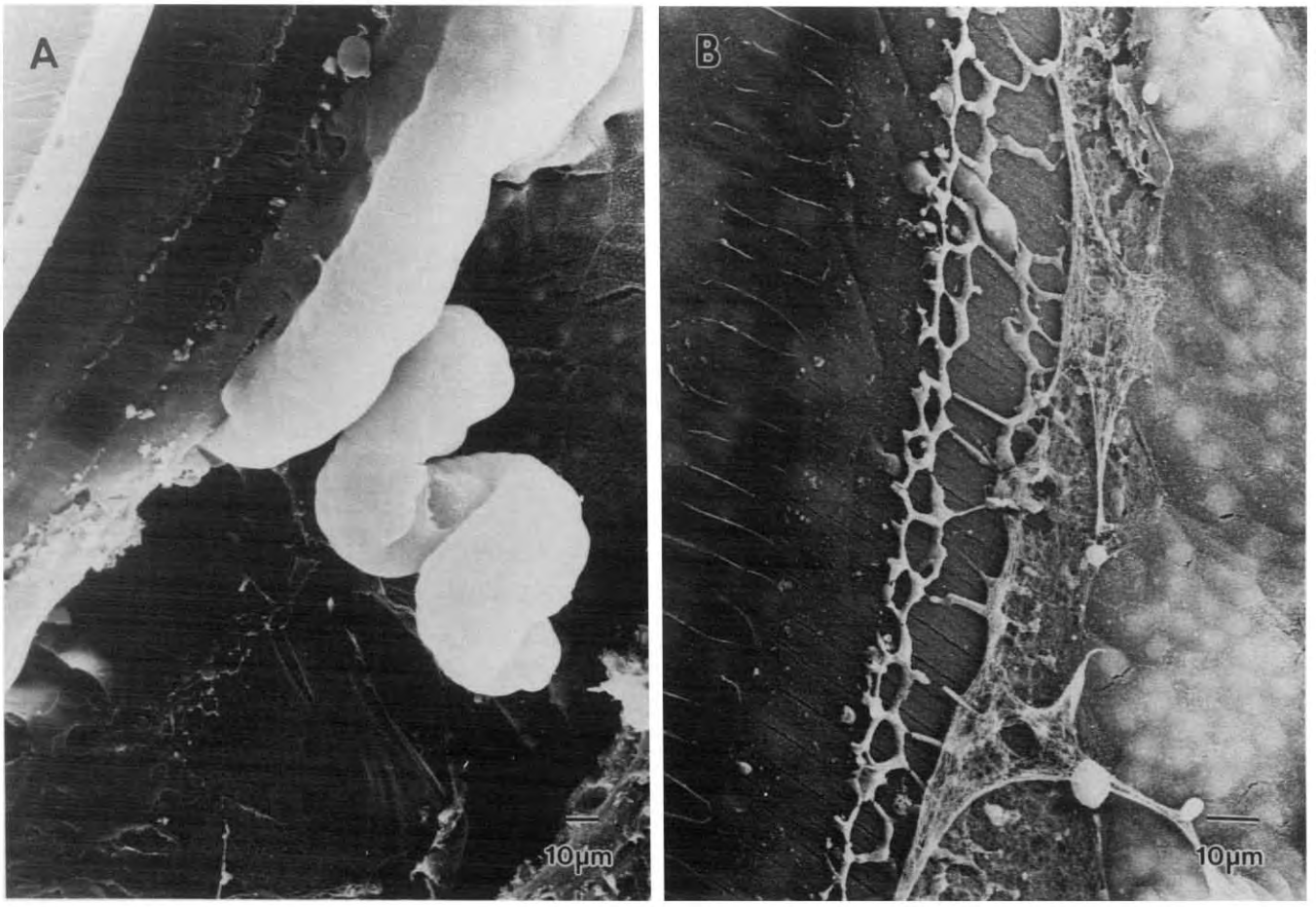
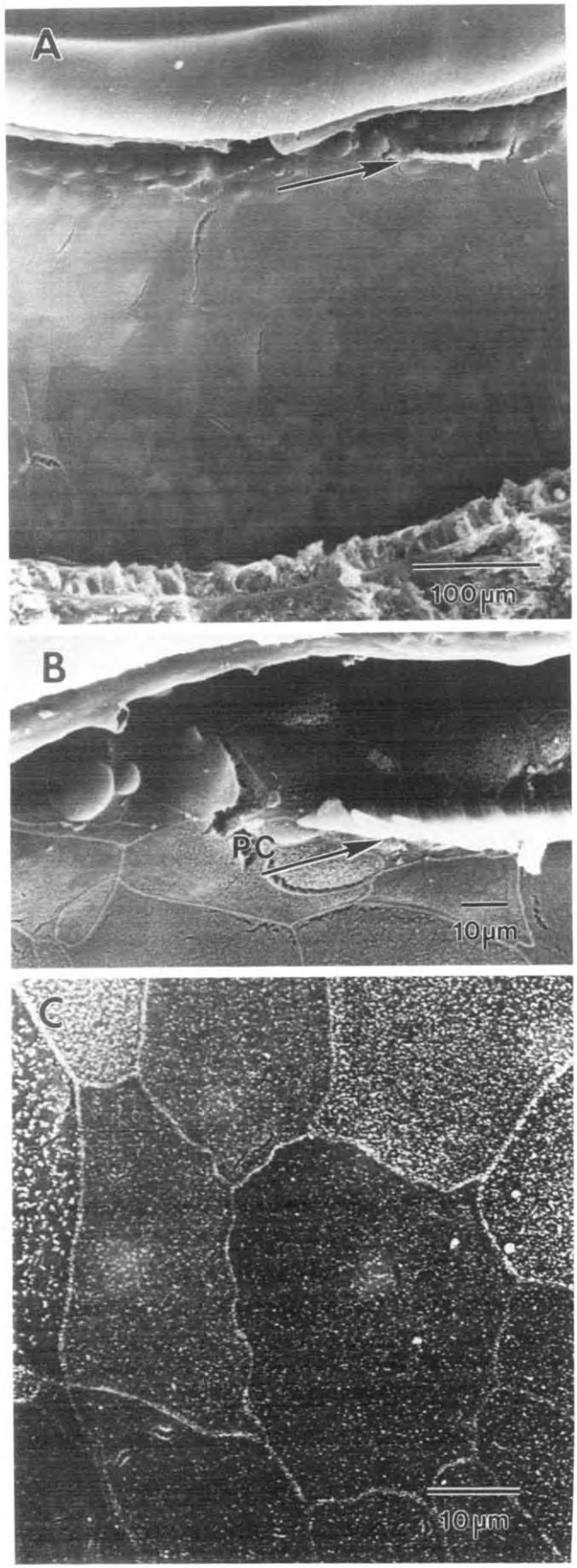



Fig. 13. (A) Note the symmetry of the pathology in the right ear (No. 3617R) with the left chinchilla cochlea shown in Fig. 12. (B) Network of degenerating nerve fibers.




the basilar membrane (Figs. 12A and 13A). The 'ripped' tissue is degenerating and is expected to be absorbed by approximately ten days (Hamernik et al., 1984b). Figs. 12B, 12C and 13B show that the basilar membrane is devoid of OHCs and supporting cells and only the remains of the degenerating outer pillar cells are seen. The remaining pillar cells look reasonably normal, however, the orderly line of pillars is disrupted with patches of missing cells (Fig. 12C).

By thirty days, the organ of Corti is missing to a large extent and is covered with a simple layer of epithelial cells (No. 3467). These cells are likely to be an outgrowth of Claudius or Hensen's cells that now span the whole extent from the stria vascularis to the inner sulcus (Fig. 14A). The surface of the cells show a proliferation of microvilli which is characteristic of cells in the organ of Corti 1 to 30 days following acoustic trauma. Fig. 14B and C show a magnified view of a section shown in Fig. 14A. A few remaining pillar cells may be seen in Fig. 14B and the epithelial covering replacing the organ of Corti may be seen in Fig. 14C.

## 4. Discussion

### *4.1. Recovery from TTS*

The recovery of evoked potential thresholds following exposure to continuous noise depends on the acoustic parameters of the noise exposure. If the exposure is of a relatively short duration ($< 8$ h), and the effects are temporary, then the recovery of hearing is linear-in-log time, i.e., the rate of recovery is maximal immediately after the exposure and as time after the exposure increases the rate of the recovery decreases (Ward, 1963). If the exposure is of a longer duration ($> 8$ h), and if part of the hearing loss is permanent, then the recovery process is more complicated. At the cessation of the noise, hearing loss may be stable for several hours, and then begin to recover in a linear-in-log time pattern. This pattern of 'delayed' recovery is associated with permanent damage to the cochlea and permanent hearing loss (Bohne, 1976; Bohne and Rabbitt, 1983).

There is substantial variability in the reaction to exposure to impact or impulse noise. A non-linear recovery pattern is quite common and has been carefully described by Hamernik et al. (1988). Initially, here is a period of recovery, then by 2 to 8 h after the

Fig. 14. Thirty days after the 137 dB exposure there are extensive regions of missing organ of Corti with some pillar cells present (arrow) (A and B) which is replaced with epithelial cells covered with microvilli (C) (3567L).

exposure hearing loss begins to grow again; finally, by 8 to 24 h hearing loss again begins to recover following the typical linear-in-log time pattern. One of the goals of this research was to better understand the histological changes following exposure to impact noise. When one contrasts Fig. 4A and Fig. 5D, for the 119 dB exposures and Fig. 7A and Fig. 8 A,B and C for the 131 dB exposures, it may be seen that the cochleas appear much more damaged 24 h after the exposure than immediately after. The status of the cochleas immediately after the exposure (119, 131, 131 at 4/s) all reveal restricted lesions (Fig. 4A, 7A, 10 B and C) or small clefts at the Deiters'/Hensen's cell junction. In spite of minor lesions shortly after the exposures, the auditory system manifests substantial hearing loss. By 24 h, large portions of the cochlea do not recover from the mechanical trauma and continue to degenerate (Figs. 5D, 8A, B and C, 10D). Often, there is evidence of growth of threshold shift several hours after the cessation of exposure. The more extensive pathology seen 24 h after the exposure may explain the growth of hearing loss.

### *4.2. Critical level*

The concept of a 'critical level' has had different meanings during the last twenty-five years. McRobert and Ward (1973) discussed the critical level in terms of the level of an impulse (impact) that reliably caused a temporary hearing loss. Spoendlin (1976) introduced another form of 'critical level'. He suggested that when the level of an impulse exceeded some level, the mechanism of damage shifts from being metabolic to mechanical. In his experiments, the critical level was determined to be 120 dB, however, the stimulus he used was actually a noise burst (i.e., rise/fall time and plateau), rather than a true impulse or impact noise. Based on the current study and the experiments by Henderson et al. (1991), the 'critical level' is likely to be around 125 dB (Figs. 1 and 2).

For the exposures used in this experiment, there are obvious mechanically induced lesions in the cochleas. At 137 dB the mechanical destruction is reflected as a ripping of the organ of Corti off the basilar membrane (Figs. 11, 12 and 13). The ripping of the organ of Corti was initially reported by Hamernik et al. (1984a) following exposures to 160 dB impulse noise exposures. At lower levels, 119 and 131 dB, the mechanical failures are reflected as longitudinal clefts at the lateral margin of the organ of Corti.

While there is no question that high level impact as well as impulse noise can damage the cochlea directly, the actual critical level may depend upon the acoustic parameters. For long duration exposures (eg.: 200 ms), the critical level is likely to be around 125 dB. However, as the duration of an impulse or impact is decreased the 'critical level' increases. Experiments by Danielson et al. (1991) with impulses of several milliseconds revealed a 'critical level' between 135 and 155 dB (most likely to be close to 155 dB). Unfortunately, there is not enough data to confidently state the relation between the duration of an impact/impulse noise and the critical level. However, there is enough data to confirm the general rule that the 'critical level' decreases with increased duration and amplitude. Furthermore, impacts in the range determined to be safe (between 115 to 140 dB) can cause direct mechanical damage to the chinchilla cochlea.

## 5. Conclusions

The foregoing experiments were designed to address three specific objectives.

The first, was to see if the extent and severity of the cochlear pathologies changed in a way that paralleled the non-linear recovery curves, following impact noise exposures. In spite of the limited samples of histology, it is clear that the cochlear pathologies were always greater 24 h after the exposure compared to immediately after the exposure. While the connection between specific pathologies and hearing loss is tenuous, it is clear that the hearing loss and the extent of cochlear pathologies parallel each other to the extent that immediately after the exposure both measures reflect less damage than 24 h later.

The second objective was to see if the impact noise could produce mechanical damage. On this question, there is not much doubt. Data from 125, 131 and 137 dB exposures show mechanical damage that ranges from broken tight cell junctions to actual separation of the organ of Corti from the basilar membrane. We have known for some time that impulse noise can damage the cochlea by direct mechanical failure, the results of this study show that impact noise with substantially lower peak levels can also cause mechanical damage. Therefore for the purposes of noise standards lower level impacts ($< 125$ dB peak levels) can safely be evaluated like continuous noise, while higher level impacts should be evaluated like impulse noise.

The third objective was to see if the critical level is reflected in the pathological data as well as the earlier audiometric data (Henderson et al., 1991). This question is clearly answered affirmatively in that there is substantially greater pathological changes seen for the 131 and 137 dB exposures compared to the 119 dB exposure. Exposures below the critical level did not produce dramatic pathologies; while exposures above the critical levels did, in spite of the fact that all exposures had the same total energy.

It should be recognized that the critical level is related to the 'signature' of the impulse noise and

varies depending upon the spectrum, duration and probably the repetition rate of exposures. Nevertheless, the concept of critical level is supported in the histological data, as well as, the previous audiological data. The existence of the critical level is a strong evidence against using a system for evaluating the hazards of impact noise, that is based on some form of the 'equal energy hypothesis'.

## Acknowledgement

Work supported by NIOSH (1 R010H011522100A2).

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