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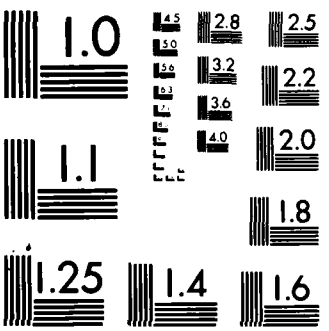

FINAL REPORT
GRANT N00014-77-G-0077

INNER EAR DAMAGE DURING DECOMPRESSION FROM DEEP DIVES 1975-1982

by

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SECTION ONE

LIST OF ACCOMPLISHMENTS UNDER THE GRANT, AND CONCLUSIONS

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List of Accomplishments under the Grant, and Conclusions

1. Techniques for the functional vestibular testing and behavioural hearing testing in monkeys were developed.

2. Techniques for anatomical study of the inner ears in monkeys were acquired and developed, including celloidin light microscopy, scanning election microscopy, and (through a colleague) transmission electron microscopy.

3. A simulated dive procedure was successfully developed to produce discrete inner ear damage in monkeys.

4. The histological picture of inner ear decompression damage was revealed, and in large numbers of monkeys. In both the cochlea and the vestibular apparatus, blood and blood-protein exudates appear in the otic fluid spaces. In the arms of the bony semicircular canals, small breaks in the bone occur.

5. It was demonstrated that in most cases of inner ear decompression damage, pathological new bone growth occurs in the arms of the semicircular canals some months after the decompression. This means that, weeks or months after suffering inner ear decompression damage, if a diver develops some degree of postural instability, or if visual blurring with head movement develops, or if tests show partial or even complete loss of function of semicircular canals, these developments can be understood as the result of new bone growth in semicircular canals, without recourse to central etiologies.

6. Understanding of the etiology of inner ear decompression damage was further advanced by the demonstration that it stems from disruption of the blood vascular supply to the inner ear, and specifically the venous supply.

7. It was shown that inner ear damage from decompression occurs discretely without any brain lesions. It must be considered rare indeed (we have never seen it) to have inner ear damage together with brain damage without the monkey's being obviously moribund. When vestibular and/or hearing symptoms occur during decompression, there is now some scientific basis for ascribing these symptoms to inner ear events alone, unless there is independent indication of central involvement.

8. It was shown that the occurrence of hearing deficits caused by inner ear decompression sickness can be followed by impressive recovery of hearing over a period of several weeks, although some degree of permanent deficit remains.

9. Sources of autopsy material from deceased divers were developed, and inner ears of two divers with a history of inner ear decompression damage were obtained (one from the Persian Gulf, and one from the North Sea). The pathological new bone growth first found in the monkeys was found in one of the human temporal bones.

10. It was found that hyperbaric oxygen therapy is beneficial in inner ear decompression damage, although such therapy does not prevent the subsequent pathological new bone growth.

11. Conclusion: the objectives of the work were realized.

SECTION TWO

SUMMARY AND PERSPECTIVE PAPER

INNER EAR DECOMPRESSION SICKNESS IN THE SQUIRREL MONKEY: OBSERVATIONS, INTERPRETATIONS, AND MECHANISMS

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INTRODUCTION

The insidious nature of inner ear decompression sickness during deep simulated dives is now well documented in the squirrel monkey (Saimiri sciureus) (1-3). In provocative ascents from 274 metres sea water (msw) while breathing a heliox gas mixture, the signs of decompression sickness appear suddenly, mainly in the form of a vigorous head nystagmus, which occurs between 62 msw and the surface. If these monkeys are sacrificed a few days after their dive, the inner ear spaces are usually congested with blood and blood proteins. In those monkeys sacrificed 20 or more days after sustaining an inner ear "hit", the most common pattern is that of ectopic new bone growth and/or the presence of fibrous material in the fluid spaces of the vestibular apparatus, particularly, in the semicircular canal spaces. Central-nervous-system damage in the centres subserving vestibular and auditory functions was only found when there was brain damage elsewhere as a result of a severe, generalized decompression sickness.

The purpose of the present report is to summarize the main findings of our research in the area of inner ear decompression sickness in deep diving environments. From these observations, a plausible theory has been developed which explains many of the results that were obtained. Furthermore, interpretation of the results suggests what procedures constitute acceptable therapeutic treatment for the successful manangement of inner ear decompression sickness.

MATERIALS AND METHODS

Animal Model

Male squirrel monkeys, free from ear infections and other disorders, were used in the study. On the day before the dive, both ear drums were surgically perforated under anesthesia so that the animal could equilibrate the middle ear with ambient pressure. Functional testing of the vestibular system was performed before and at selected intervals after the dives until the animal was sacrificed for histological study (see (3) for further details). The temporal bones were processed (horizontal sections, 20 μm thick) using the method of Igarashi (4); brain sections were similarly prepared (coronal plane, 20 μm thick).

Decompression Procedures

All experiments were performed in a small animal chamber (Bethlehem Corp., 0.173 m^3 capacity). The dive commenced with air to 13.9 msw; then with helium at a rate of 32.6 msw/minute to a depth of 274 msw. After one minute of bottom time, decompression began at a rate of 18.3 msw per minute to 61 msw; and then in steps of 6 msw every 4 minutes to the surface. Throughout the dive, the oxygen partial pressure was maintained at 50.7 kPa (= 0.5 atmospheres).

Hearing Tests and Training

In order to assess for possible cochlear damage, hearing was tested in a number of animals, both before and after the dive. The animals were trained, using a shock-avoidance procedure, to respond to tones. Pre-dive hearing thresholds were determined for a range of frequencies in each animal. The presentation of tones was controlled and delivered by a computer-based system (PDP 11/04 computer; Digital Equipment Corp.). During training and testing, the animals were isolated in a sound-proof booth (Industrial Acoustics Corp.).

After the dive, when the animals had recovered sufficiently, their hearing was again tested in order to obtain post-dive hearing thresholds. These tests were conducted on a regular basis until the animal's audiogram showed no further change, whereupon it was sacrificed for histology.

Hyperbaric Oxygen Therapy

With some animals, a regimen of hyperbaric oxygen therapy was instituted immediately after the dive. These animals were treated for 3 successive days with the U.S. Air Force modification of the U.S. Navy Table 6 Treatment (5) for decompression sickness. Initial trials with control animals had shown that the monkeys could withstand the Table 6 treatment without showing visible signs of oxygen toxicity.

RESULTS AND DISCUSSION

To date, some 250 monkeys have received inner ear hits in experiments at DCIEM, using this diving profile. Table 1 lists the distribution of new bone growth and fibrous material that has been observed in the semicircular canal spaces in 29 of these monkeys, which were sacrificed between 20 days and 661 days after inner ear decompression sickness. (The complete absence of ectopic new bone growth and/or fibrosis in the vestibular spaces has been observed in only seven hit monkeys with these long-term survival times.) Monkeys 119 and 145 were not recompressed after receiving a vestibular hit, but were brought to the surface slowly. Monkeys 135, 160, 166, and 169 were recompressed to depths which eliminated all behavioural signs of vestibular dysfunction before being brought to the surface slowly, at the rate of 0.3 msw/minute. The remaining 23 monkeys in Table 1 were compressed to twice the depth at which the hit occurred and then brought to the surface at the slow rate indicated above. Starting immediately after receiving their vestibular hit, monkeys 201 and 207 were subjected to a Table 6 treatment for three successive days.

Monkeys Pancho, Manuel, Juan and Enrico, who had been conditioned to discriminate between tones and no-tones, were tested for hearing thresholds before, and at selected intervals after, their dives.

Of the 34 monkeys with long-term survival times that received some form of the recompression treatment, 29 had no apparent behavioural signs of inner ear decompression sickness (such as nystagmus and/or unsteadiness) upon completion of the recompression schedule (including 6 of those in which there was no evidence of bone growth in the canal spaces). However, about one-third of these monkeys developed a positional nystagmus the following day, a clear indication of continuing vestibular dysfunction.

Regardless of whether or not the animal received the recompression and/or hyperbaric oxygen treatment(s), the entries in Table 1 indicate the same outcome, i.e., the gross infiltration of fibrotic tissue and/or new bone in the canal spaces, as a consequence of inner ear decompression sickness (cf. Figs. 1 and 2). (In animals sacrificed shortly after their dive, the manifestations of vestibular-apparatus damage appears in the form of severe hemorrhage in the semicircular-canal perilymphatic spaces, and as blood-protein exudates, mainly, in the endolymphatic spaces; in particular, as an agglutinate to the cupula of the crista ampullaris (3).) This new bone continues to grow slowly until it also occludes the endolymphatic space of the semicircular canal in some cases (see (e,p) entries in Table 1); and in others, it continues until it encroaches on the perilymphatic space of the ampulla (see * entries in Table 1). In either case, the likely result is the same: a progressive lessening of vestibular function, until total malfunction of the involved

duct(s) can occur. The table entries also illustrate that this bone growth occurs 1.5 times more often in the left labyrinth than in the right. Furthermore, it may occur in one or more canals on the same side of the head, or it may occur in one or more canals on both sides of the head. Any and all combinations are possible.

It appears that this new bone growth is caused, partly, by a ripping or irritation of the endosteum, which lines the inside of the bony semicircular canals (3). There is also convincing evidence that rapid decompression can generate forces of a magnitude sufficient to fracture the hard temporal bone that is contiguous to the bone comprising the semicircular canal, as well as the canal wall itself (6).

In an investigation of the biophysical mechanisms responsible for causing this type of damage in monkeys during rapid decompression, Ward and his colleagues (7) have produced a model which theorizes that, for certain conditions during decompression, large stresses can be produced by bubble nucleation and growth within osteoclast cell cavities in the bone, such as are found in temporal and canal bone. Bubble nucleation is thought to occur within the conical processes of the "ruffled" border -- a specialized membrane involved in bone resorption -- of an osteoclast cell. This theory predicts that the essentially-incompressible cytoplasm in the bone cells (which form a constant-volume, basically-closed cavity of

liquid-gas solution) experiences a pressure rise with bubble growth sufficent to produce a force capable of fracturing the bone in which it is contained. Near the surface during ascent, the fluid spaces of the semicircular canals would be at ambient pressure compared to the very high pressure that would be experienced by the cytoplasm within the osteoclast cavity during bubble growth, following exposure to 274 msw. Therefore, once a critical state is achieved, the bubble would grow, causing a large transient increase in pressure relative to that of ambient pressure. Accordingly, this would result in a sudden implosive fracture of the (presumably) rigid temporal and canal bone into the semicircular canal spaces during the final phases of decompression. This theory is consistent with some of the types of semicircular canal damage that have been observed histologically following rapid decompression (6). Venter and his colleagues (8) have found that a mean pressure of 1.6 ± 0.4 MPa is required to fracture the full thickness of the semicircular canals in squirrel monkeys; this pressure is consistent with that predicted by Ward's theory.

The implosive force also causes a bolus of pressure wave energy to move rapidly along the canal which could tear the endosteum and loosen the membranous semicircular ducts from their anchorage to the canal wall as well as to provide a substantial transient stimulus to the vestibular end organs. This transient stimulus likely explains why a vestibular hit appears suddenly during decompression in most of these cases. (Vestibular hits resulting from blood supply

changes to the vestibular end organs and from central vestibular dysfunction can also appear suddenly.)

The theory of Ward and his colleagues (7) also predicts that, under similar conditions, for less severe pressure, bubble nucleation and growth in bone cells will not produce the pressure required to break the bone. However, this pressure difference, which could be of the order of 0.5 MPa, would still damage or kill the bone cells by mechanical distortion. This mechanism could explain the empty bone cell cavities found in the long bones of divers suffering from aseptic bone necrosis, a debilitating condition which becomes progressively more evident several years after undergoing decompression procedures.

It is instructive to compare the manifestations and symptoms of inner ear decompression sickness in the squirrel monkey with those observed in the commercial diver. In particular, the clinical observations on 8 divers by Komordin (9) are pertinent and characteristic. The sickness appeared suddenly during decompression, at depths of 40-45 msw, 1-1/2 to 3 hours from the time the dive began. In all cases, the bottom depths exceeded 150 msw, and the breathing gas mixture was oxy-helium. Severe vertigo, nystagmus, nausea, emesis, tinnitus, a loss of spatial orientation, and a decrease in hearing were evident in most cases. Symptoms associated with the decompression syndrome, such as joint pain, itching of the skin, etc., were

usually absent. Immediate recompression was successful in only about a third of these instances, and then only under the conditions of high pressure for long periods.

We have recently completed the histological study of the temporal bones of a professional diver who died (of unrelated causes) 56 days after sustaining a severe inner ear hit to his left labyrinth following a dive to about 100 msw for 19 minutes on Trimix (10% nitrogen). Some six hours after the dive, during sleep, this diver experienced dizziness and knee pain and was, therefore, promptly recompressed at least twice (once for 52 hours) in an attempt to provide relief (unsuccessfully). Clinical tests indicated a total loss of vestibular function and a partial hearing loss in the left ear. The histological study revealed ectopic new bone growth and fibrosis in one of the semicircular canals of the left ear (10). Clearly, many of the manifestations and symptoms of inner ear decompression sickness in man and monkey are of a similar nature.

The insidious nature of the vestibular lesions makes it imperative that any diver who has experienced inner ear decompression s[illegible] should obtain follow-up clinical evaluation over a period of s[illegible]s before a clean bill of health is given. The fact that many [illegible]s experience vertigo during decompression suggests that pathological bone growth may, perhaps, be quite common in older divers. (It would be interesting to know whether or not divers hear

a click, snap, or bang preceding a vestibular hit, as might be expected on the basis of Ward's model (7).) The slow growth rate of this ectopic bone would be expected to give central-nervous-system compensatory mechanisms time to develop and restore normal balance during ambulatory situations when there is good visibility. However, exposure to conditions of neutral buoyancy and poor visibility, such as can occur while diving, could lead to disorientation and may threaten the life of a diver who had previously received such a hit.

Accordingly, it may be prudent for every diver who has sustained such a hit, to obtain either a temporal-bone computerized tomographic scan (11) or, better yet, one that utilizes nuclear-magnetic-resonance imaging techniques (since this does not require exposure to radiation but provides similar information) in order to assess the true nature of the damage before returning to diving. Moreover, older divers, even if they have not experienced a direct vestibular hit, should routinely obtain a thorough otoneurological evaluation (complete with scan) as there is some evidence that a series of sub-threshold vestibular assaults could produce a similar pathology.

The clinical practices of the French specialists in the treatment of decompression-caused ear injuries in divers are of interest to this study (12-15). Regardless of the dive, whether on compressed air or helium, shallow or deep saturation, the recommended practices

appear to be the same: the treatment consists of hyperbaric oxygen therapy in combination with vasodilators, corticosteroids, and heparin in small doses. Experience has shown that hyperbaric oxygenation with this type of adjuvant drug therapy is very effective in reversing peripheral cochlear dysfunction. However, the French have noted that, in spite of immediate treatment, peripheral vestibular lesions from dives to great depths can be severe from the onset; and, furthermore, may be permanent (16) and worsen with time (17). McCormick et al. (18) have recommended the prophylactic use of heparin and Novotny (19) the use of nicotinic acid, a reputed vasodilator, for ameliorating decompression-induced hearing loss.

Given the nature of the damage sustained by the peripheral vestibular apparatus during inner ear decompression sickness, it is not surprising that neither hyperbaric oxygen therapy (monkeys 201 and 207 in Table 1) or prompt recompression were found to be beneficial forms of therapy. Similarly, the beneficial effects of adjuvant drug therapy with vasodilators, anti-inflammatory agents, and anticoagulants would likely be extremely limited. Diazepam®, a tranquilizer and anti-convulsant agent, which has been recommended as adjuvant therapy for labyrinthine decompression sickness (20), would also seem to be powerless to either reverse or prevent this kind of damage, though it may provide some much-needed relief from vertigo.

Since the nature of the damage to the peripheral hearing organ

in squirrel monkeys is quite different from that to the vestibular apparatus, it may be that hyperbaric oxygen therapy as used by the French (12-15) and others (9,19) could be a beneficial form of treatment. Indeed, studies to date in our laboratory have shown that the cochlear damage following a successful hit first appears histologically only in the form of a blood-protein exudate and an occasional hemorrhage of the cochlea. Therefore, it is important that the highly-specialized receptor hair cells, which consume large amounts of oxygen, do not become anoxic as a result of blood interruption to the organ of Corti. (It bears mention that the organ of Corti always appears intact and functional in celloidin histological sections.) In animals sacrificed several months after receiving an inner ear hit, the cochlear fluid spaces have become quite clear, containing much less exudate than is observed shortly after the dive (Fig. 3). In this regard, the amount of exudate is similar to that observed in control animals. Moreover, bone and/or fibrotic growth have never appeared in the cochlea. Notwithstanding the cochlear deficits, gradual recovery of some hearing function was evident in monkeys which were tested behaviourally for hearing loss (Fig. 4), even though there was strong evidence that vestibular function remained severely interrupted (e.g., Pancho, Manuel, Juan, and Enrico in Table 1). Of course, the fluid spaces in the inner ear are shared by both the vestibular apparatus and the cochlea. Thus, injury to one organ often results in a decrement of sensory function in the other organ. In this regard, the hemorrhage or blood vessel blockage

that may result from bubble nucleation and growth in the ear vessels during decompression can be significant. The effect on the fluids in one organ may cause changes in the electrolytic and protein compositions in the other organ, resulting in an interference with the physiological mechanisms involved in normal sensory function. Presumably, as the initial disorder subsides and clears, the mechanisms which control sensory function are re-established. This might explain why, sometimes, there is sensori-neural hearing loss without any apparent concomitant pathological basis.

All of the hits that are recorded in Table 1 occurred at various depths during ascent, and never at the surface. There are, however, many instances of human divers whose symptoms appear suddenly, only after decompression has been completed. This could signify that there are gas bubbles which are trapped in the conical processes, but whose further growth has been inhibited by the rigid surrounding bone. It is possible that such bubbles could remain quiescent and asymptomatic, until some unknown, cataclysmic "jolt" causes them to continue to grow and, thereby, fracture the bone containing them. Accordingly, a prudent method for preventing this type of subsequent bubble growth might be the mandatory use of the U.S. Air Force modification of the U.S. Navy Table 6A Treatment (5) for gas embolism whenever decompression sickness is suspected*. Furthermore, as indicated above, it would appear that such treatment would benefit the cochlea.

Farmer (20) has indicated that a drug treatment which relies upon increasing inner ear blood flow, as has been recommended by the French and others (12-15,18,19), may result in additional bleeding; or, it might cause blood flow to be shunted to more peripheral regions, thereby counteracting its intended purposes. Such agents are considered by him to be potentially harmful and are not recommended. The results from this study would tend to support Farmer's reasoning.

SUMMARY AND CONCLUSIONS

As a result of our research on the nature of the inner ear decompression sickness resulting from deep dives, the following statements can be made:

1) The sickness occurs very suddenly during the ascent phase of the dive (between 62 msw and the surface, for squirrel monkeys on a 274-msw dive).

2) Prompt recompression appears to lessen (or even eliminate) the acute behavioural signs and symptoms of inner ear decompression sickness; it does not, however, reverse the pathological damage to the vestibular apparatus that is provoked by the dive.

3) Serial histological sections of the brains and temporal bones of monkeys which had received inner ear hits show that, if the only

symptoms are of a vestibular nature, then the problem is likely to be only in the ear, and not in the brain.

4) Cochlear damage first appears in the form of blood and blood-protein exudates in the otic fluid spaces; much later, these spaces appear clear, similar to those observed in control animals. Hearing tests show that some of the monkeys gradually recover some of their hearing deficit (without benefit of adjuvant drug therapy); however, a residual loss remains permanently.

5) Vestibular-apparatus damage first appears in the form of severe hemorrhage and blood-protein exudates in the otic fluid spaces; later, these spaces become invaded by ectopic new bone growth and fibrous material.

6) Histologically, temporal- and canal-bone breaks are prevalent in some of the hit monkey ears. A theory was developed (7) which predicts that such breaks could occur from "imploding" forces that are caused by the large pressures (1.6 MPa or more (8)) that are generated as a result of bubble nucleation and growth within osteoclast (bone) cells during decompression.

7) Cochlear lesions likely occur as a result of bubble formation and growth within microvessels, and their consequent blockage and/or rupture causing hemorrhage and/or blood-protein exudatation. Because

of the damage of anoxia to the highly-specialized receptor hair cells of the cochlea under such conditions, immediate hyperbaric oxygen treatment would appear to be a necessary requirement.

8) Vestibular lesions of the type described in this report would not benefit from (nor be aggravated by) hyperbaric oxygen therapy, whether the treatment used is for decompression sickness (Table 6 treatment) or for gas embolism (Table 6A treatment). However, since cochlear lesions often appear with vestibular lesions, such treatment should be a recommended practice whenever a cochleovestibular insult is indicated.

9) Adjuvant drug therapy that increases inner ear blood flow is contraindicated in the treatment of inner ear decompression sickness.

10) Any diver who has experienced inner ear decompression sickness should obtain extensive follow-up clinical evaluations before returning to diving. Older divers, whether or not they have received a direct inner ear insult during decompression, should routinely obtain a thorough otoneurological examination.

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Table 1. Distribution of new bone growth and fibrous material in squirrel-monkey semicircular canals[+]

Monkey	Depth of Initial Hit on Ascent, msw	Post-Dive Survival Time, days	New Bone Growth and Fibrous Material	
			Left Labyrinth	Right Labyrinth
8	12.2	38		ASC(p)
24	61.9	379		ASC(p), LSC(p), PSC(p)
32	18.3	211		ASC(p), LSC(p), PSC(p)
33	6.1	93		ASC(p)
36	6.1	184	ASC(e,p)*, LSC(p), PSC(e,p)*	
51	12.2	56	LSC(p), PSC(p)	ASC(p), LSC(p), PSC(e,p)
52	36.6	126	LSC(p)	LSC(p)
62	24.4	383		PSC(p)
68	30.5	290	ASC(p)*, LSC(p)*	
103	6.1	637	LSC(p)	
117	18.3	147	ASC(e,p)*, LSC(e,p)*, PSC(e,p)*	ASC(e,p)* PSC(e,p)*
119	18.3	133	ASC(e,p), LSC(p), PSC(e,p)*	
135	18.3	388	ASC(p), LSC(p)*, PSC(p)	ASC(e,p),LSC(e,p)*, PSC(e,p)
136	30.5	213	ASC(p) PSC(p)	ASC(p), LSC(p), PSC(p)
145	7.6	161	ASC(e,p)*, LSC(e,p)*, PSC(p)	
147	30.5	188	PSC(p)	
160	30.5	65	ASC(p), LSC(p), PSC(p)	
166	18.3	70		ASC(p)
169	12.2	20	ASC(p)*, LSC(p)*, PSC(p)	
183	11.3	490	ASC(p), LSC(p), PSC(p),	ASC(p)
185	6.1	353	ASC(e,p)* LSC(p), PSC(p)	
187	12.2	21		ASC(p), LSC(p), PSC(p)
201	18.3	197	ASC(p), LSC(p), PSC(p)	ASC(p), LSC(p)
207	30.5	185	ASC(e,p), LSC(p), PSC(e,p)	ASC(p), LSC(p), PSC(p)
211	18.3	661	ASC(p), LSC(p)*, PSC(p)	
Pancho	6.1	239	ASC(e,p)*, LSC(p)*, PSC(e,p)*	ASC(p), LSC(p), PSC(e,p)
Manuel	30.5	333	LSC(p)*, PSC(p)	ASC(p), LSC(p)*, PSC(p)
Juan	30.5	178	ASC(p), LSC(p)*, PSC(p)	
Enrico	6.1	322	ASC(p)	

ASC, LSC, and PSC signify the anterior, lateral, and posterior semicircular canals; e and p, the endolymphatic and perilymphatic spaces, respectively.

[+] Information for monkeys 8–68 inclusive has appeared previously in Tables 4 and 5 of Ref. 3.

* New bone growth and fibrous material were also found near ampulla and utricle in perilymphatic space.

Footnote

* DCIEM's only effort with the modified Table 6A Treatment was equivocal. Monkey W13 (not mentioned previously), upon receiving a central-nervous-system (CNS) hit at 30.5 msw on ascent from a 274-msw dive, was recompressed to 50 msw. This removed the CNS hit but left the monkey with an apparent vestibular hit (possibly a hit in the vestibular central pathways) that persisted until the animal was (slowly) decompressed to 19.5 msw, whereupon all signs of the hit disappeared. On reaching surface, monkey W13 was given a single modified Table 6A Treatment. This monkey was sacrificed 86 days after the dive; subsequent temporal bone histology indicated that the vestibular organs were intact and in good condition.

Figure Captions

Fig. 1. Normal posterior (PSC) and lateral (LSC) semicircular canals and associated structures in a horizontal section from the right labryinth of monkey 166. The membranous posterior (PSD) and lateral (LSD) semicircular ducts lie within the respective canals. Abbreviations: MU = macular utriculi -- the sensory end organ for the detection of linear accelerations and gravity which is located in the utricle (U); CR = crista ampullaris -- the sensory end organ for detecting angular accelerations; CU -- cupula (which interfaces with CR and normally fills the ampullary space in its plane of projection, but has shrunk as a result of tissue fixation); LA = ampulla of LSC; e and p = endolymphatic and perilymphatic fluid spaces, respectively. The dark half-circular band on the distal surfdace of the crista is its sensory epithelium. Bar = 50 μm.

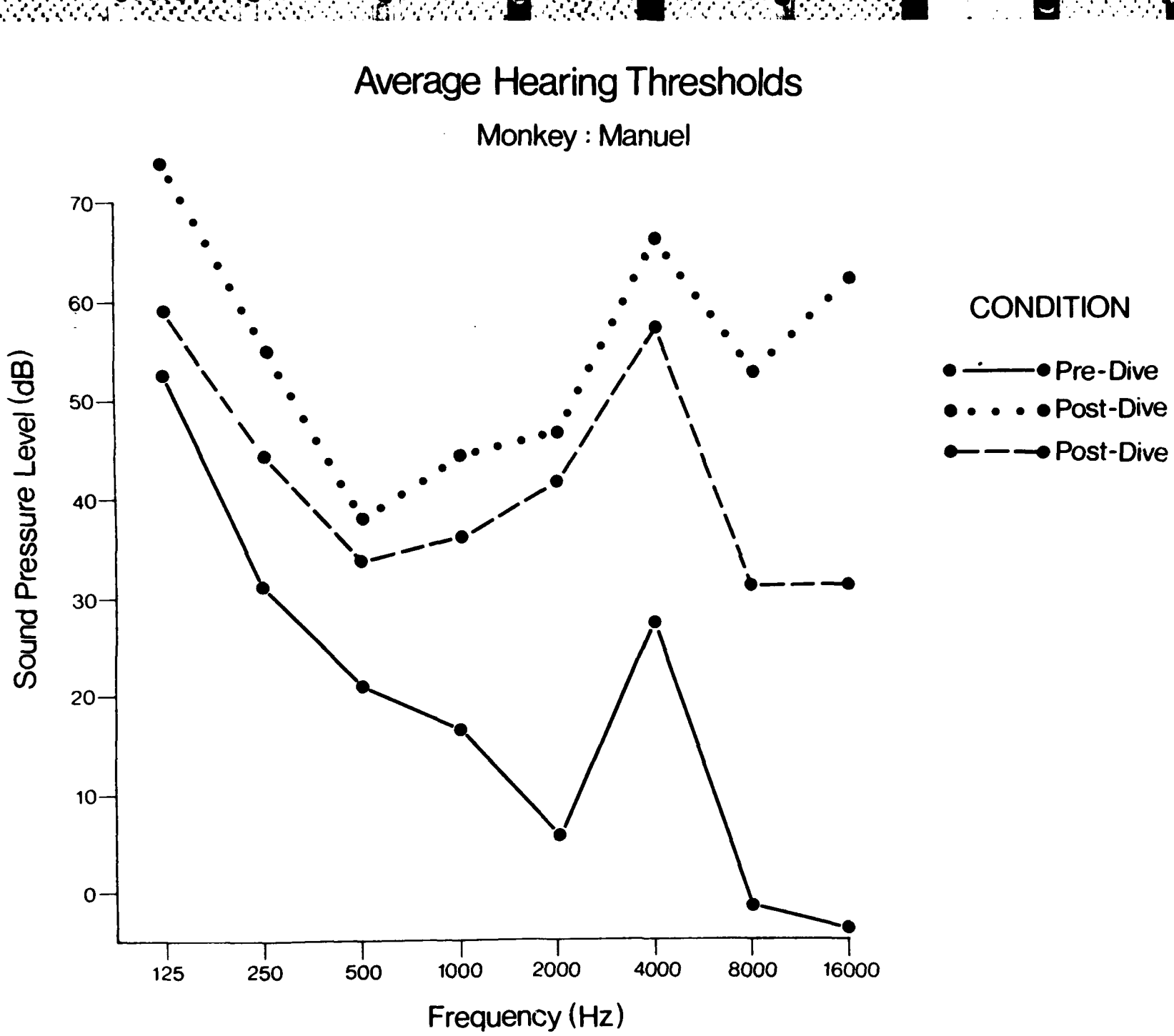
Fig. 2. Horizontal histological section from the left labyrinth of monkey Pancho, illustrating extensive bone and fibrotic growth (FOM) in the otic fluid spaces in the posterior (PSC) and lateral semicircular canal (LSD, LA) systems. Note that the cupula is missing and that the "bald" crista ampullaris (CR) has only a very thin band of sensory epithelium. This clearly indicates a non-functioning sensory end organ. Both

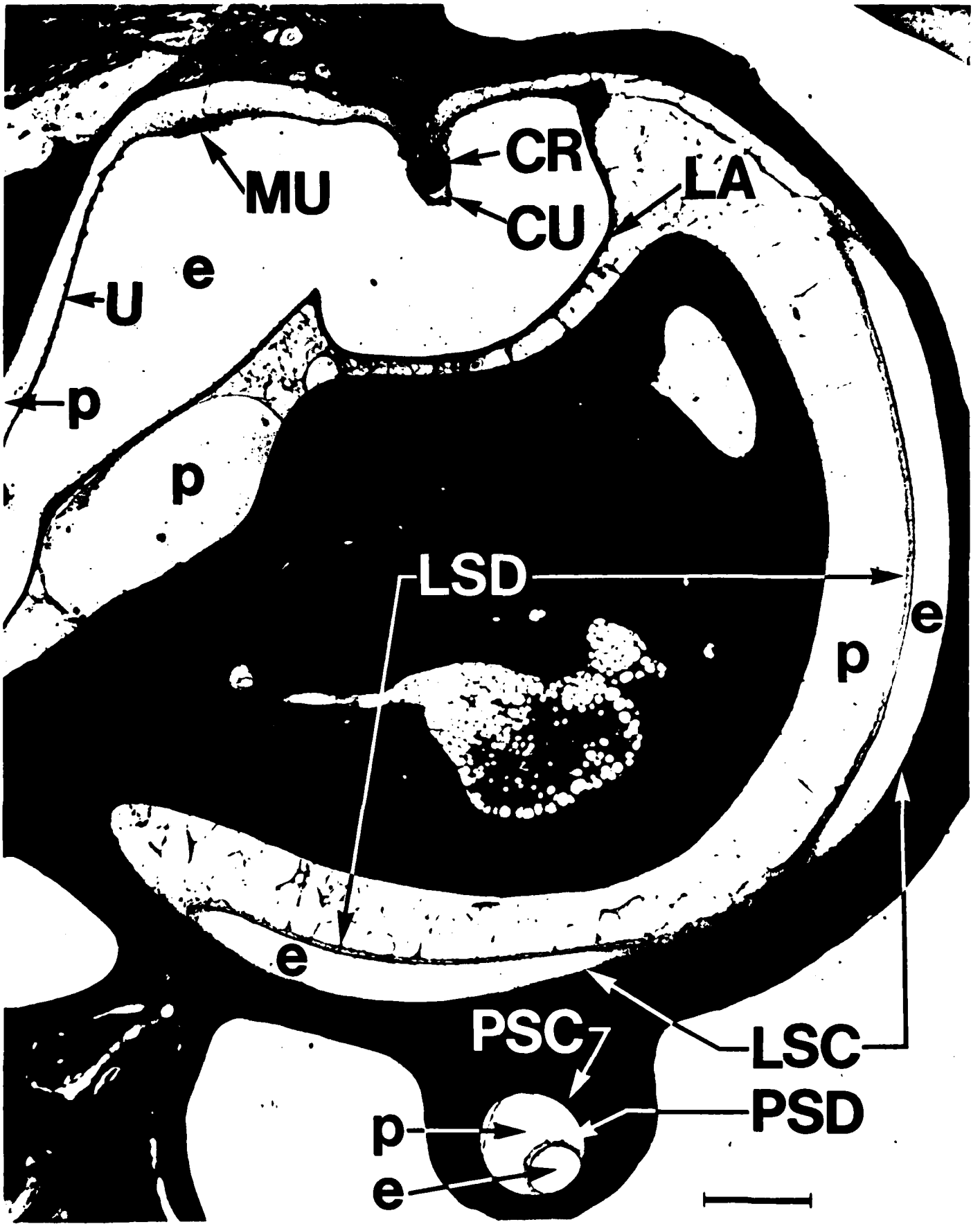
otic fluid spaces in the PSC have been blocked by ROM, rendering that canal non-functional also. Symbols: U = utricle; * designates otoconial (ear-stone) mass that has been displaced from the macula utriculi (which is not clearly defined in this photo). Bar = 50 μm.

Fig. 3. Horizontal histological section illustrating the presence of a normal-appearing cochlea next to a pathological lateral ampulla (LA) from the left labyrinth of monkey 169. Both the endolymphatic (e) and perilymphatic (p) spaces in the cochlea are clear, and the organ of Corti (OC) — the sensory end organ of hearing -- appears normal. The presence of fibro-osseous material (FOM) in the perlymphatic space of the LA is evident. Abbreviations: SV, ST, SM = scalae vestibuli, tympani, and media, respectively (otic fluid spaces in the cochlea); others as in Fig. 1. The * identifies cupular remnants which have become detached from the CR. Bar = 50 μm.

Fig. 4. Curves of average hearing thresholds, in decibels (dB) as a function of the stimulus frequency for monkey Manuel. The conditions are: (1) pre-dive (averaged over 3 tests), (2) post-dive 1 (92-125 days after the dive; averaged over 4 tests), and (3) post-dive 2 (287-301 days after the dive; averaged over 4 tests). As indicated, there was some

recovery of hearing between the post-dive-1 tests (when the hearing threshold was at its worst) and the post-dive-2 tests (the final tests before the animal was sacrificed 32 days later). The curves indicate that a (possibly-permanent) residual hearing deficit of 10 dB (at the lower frequencies) to 30 dB (at the higher frequencies) remains at the time of sacrifice.


Average Hearing Thresholds
Monkey : Manuel
Sound Pressure Level (dB)
70
60
50
40
30
20
10
0
125
250
500
1000
2000
4000
8000
16000
Frequency (Hz)
CONDITION
Pre-Dive
Post-Dive
Post-Dive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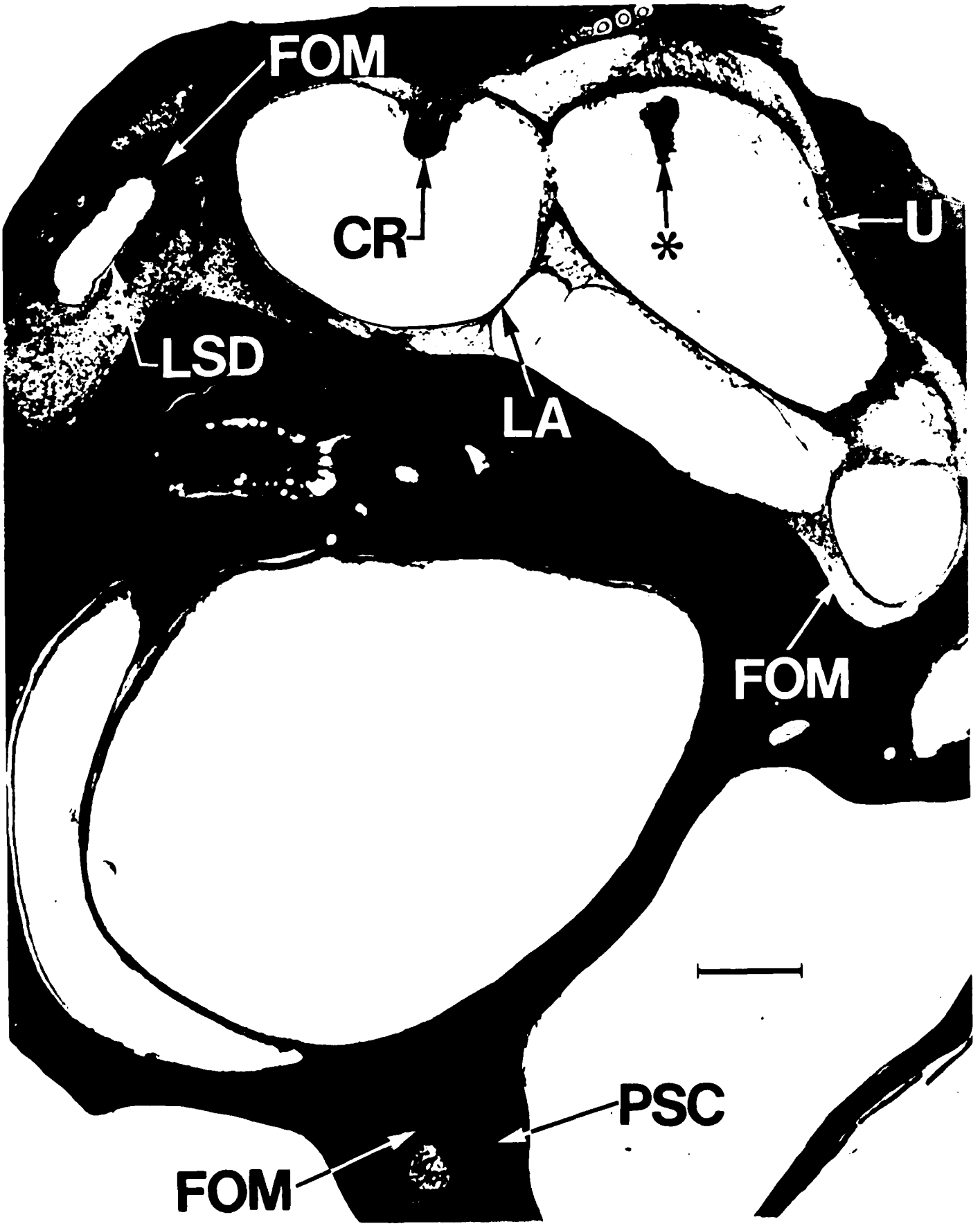
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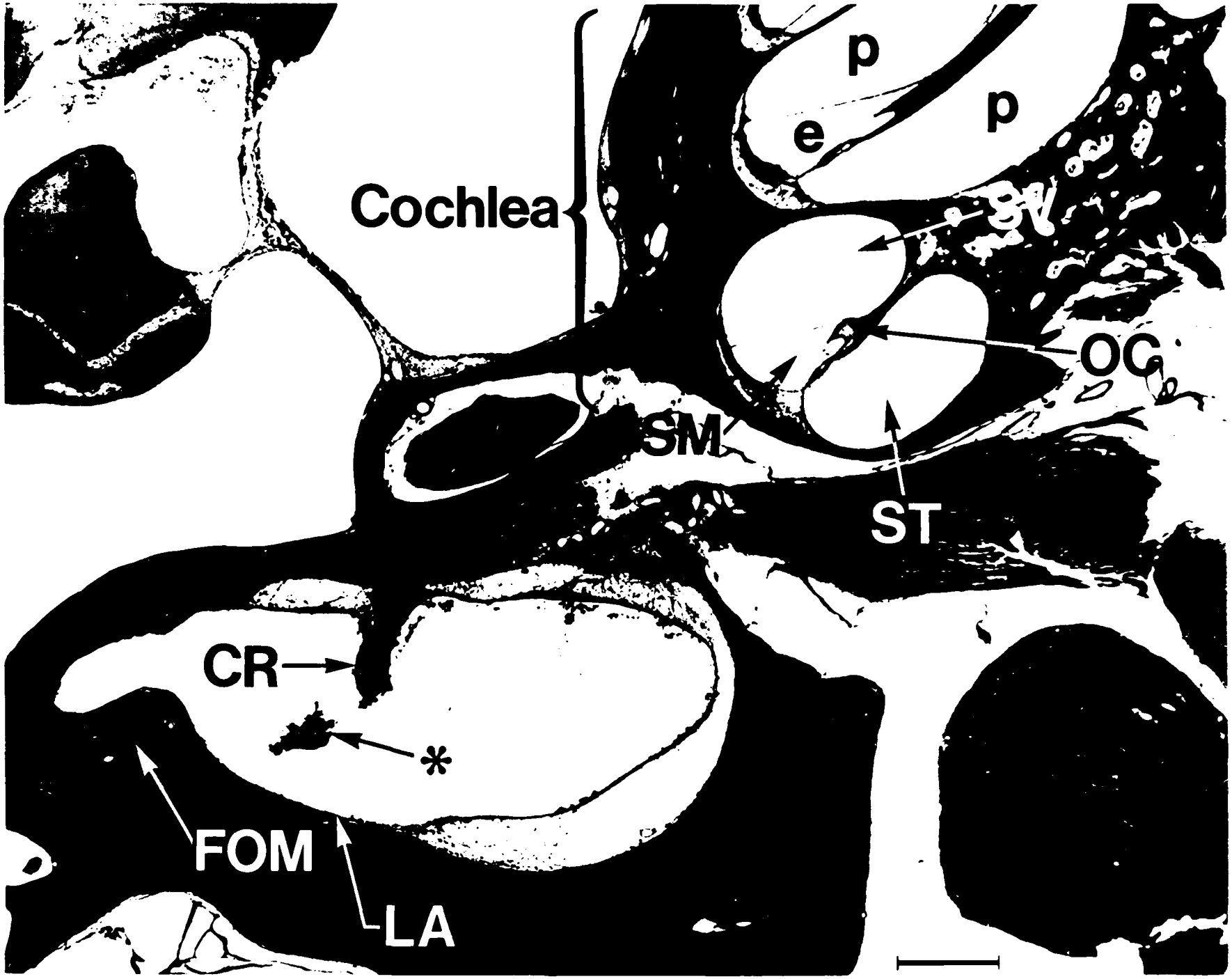
Cochlea
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FOM
LA

SECTION THREE

FINDINGS IN HUMAN TEMPORAL BONES

DAMAGE TO THE MIDDLE EAR AND THE INNER EAR IN UNDERWATER DIVERS

K.E. Money, I.P. Buckingham, I.M. Calder, W.H. Johnson, J.D. King, J.P. Landolt, J. Laufer and H. Ludman

Introduction

The direct action of the pressure changes that are encountered in diving can cause damage to the ear in at least three distinctly different ways:

(1) A relatively low pressure in the middle ear during descent[1], if large enough, can cause bleeding into the middle ear and even rupturing of the ear drum.

(2) In attempting to raise the pressure in the middle ear during descent, "clearing the ears" with a Valsalva manoeuvre, if attempted too vigorously, can result in breaking of the round window membrane and leaking of perilymph into the middle ear. One postulated mechanism[2] to explain this damage includes a raising of the intracranial pressure by the Valsalva manoeuvre, the transmission of this pressure to the perilymph space, and a progressive bulging of the round window membrane into the middle ear until a rip in the membrane allows perilymph

to escape. Another postulated mechanism[3-4] includes the sudden success of a forceful Valsalva maneouvre: the inwardly-bulging ear drum suddenly bulges outwardly, pulling the stapes outward and "blowing in" the round window membrane with resulting rupture of the round window membrane. Regardless of the mechanism of damage, the "inner ear barotrauma"[3] results in a perilymph leak into the middle ear[5], and the diver experiences some or all of: dizziness, tinnitus, and hearing loss. In some divers, bed rest with the head elevated for several days allows healing to take place, but in some cases surgical repair of the round (or oval) window is required to prevent permanent loss of hearing.[4-7]

(3) During ascent from a dive, the Eustachian tube normally opens without difficulty and large pressure differences across the ear drum do not occur. However, if the breath is held during ascent (a mistake that is frequently fatal because of "burst lung"), then the reduction of pressure outside the ear drum could, in theory, result in the bulging of the ear drum outward, causing rupture of the ear drum with concomitant bleeding into the middle ear.

Unrelated to the damage that is caused directly by these pressure changes, is the damage to the ear that is caused indirectly

by the pressure changes, i.e., by means of decompression sickness (bends or hits) of the inner ear. If a dive is deep and prolonged, then excessive quantities of nitrogen or other inert gases are dissolved in the body tissues. Then, if the ascent is too rapid, these gases will come out of solution and form bubbles that can cause extensive damage. Such decompression damage to the inner ear is one of the most common kinds of bends associated with deeper diving[8], and it presents itself as hearing losses and/or dizziness in divers[9-10]. The damage is usually unilateral and can be reversed with prompt standard recompression procedures[9-10] (including hyperbaric oxygen therapy[11]) but the hit sometimes results in permanent damage (even a "dead ear"). In experimental animals,decompression damage to the inner ear includes bleeding into the perilymph and endolymph fluid spaces of both the cochlea and the vestibular apparatus[12-15], small breaks in the temporal bone[16], and invasion of the semicircular canals by new bone growth[16-17].

The present study utilized post-mortem human material to determine whether or not the clinical manifestations of damage to the ear in diving are consistent with the observed pathology, and, moreover, to see whether or not the pathological findings in experimental animals have their counterparts in the human material.

Methods

The study material includes the temporal bones of: (1) divers who had died from "burst lung", because of breath holding during ascent (to see whether or not there was bleeding in the middle ear space), (2) divers who had a history of inner ear bends and who died subsequently (because of accident or other causes), and (3) divers who died underwater for reasons unknown. The temporal bones were sectioned serially for histological study using the celloidin method of Igarashi[18].

Results and Discussion

Burst Lung Findings

It was found that bleeding into the middle ear, and rupture of the ear drum, does indeed occur in association with the "burst lung" condition that results from ascent while holding the breath. Figure 1 shows this quite explicitly, with bleeding from the ears occurring in a diver who died underwater from burst lung. On both sides, the ear drums were ruptured, and bleeding into the middle ear resulted in the blood appearing in the external ears. The amount of blood is much greater than the amount that could come from the tiny vessels supplying the tympanic membrane itself, and it seems likely that a direct pressure difference caused rupturing of vessels in the

lining of the middle ear cavity.

Ascending with closed glottis is one of the most elementary and basic errors a diver can make and it is, therefore, reasonable to think that such an error would usually occur only after a considerable provocation such as: (1) gross contamination of the breathing gas (for example, if the diver inhaled oil or water or vomitus, he might panic and hold his breath while swimming for the surface); (2) decrease in cerebral competence (for example, a toxicosis); or (3) a disorientation (for example, in conditions of restricted visibility, a vestibular malfunction could make the diver think he is swimming down when he is actually swimming up). Disorientation with erroneous perception of the down direction occurs readily underwater[10] and, at least in aircraft pilots, disorientation can be strongly compelling even when correct symbolic information about orientation is available. It seems obvious then, that disorientation from whatever cause could give rise to middle ear and/or inner ear damage (and/or burst lung) and that, conversely, inner ear damage from whatever cause could give rise to disorientation.

Inner Ear Decompression Sickness

As in experimental animals[14], it was found also in the human material that decompression sickness (resulting from bubbles of inert gases coming out of solution with decreasing pressures, i.e.

bends), can cause bleeding into the perilymph spaces of the cochlea. Figure 2 shows this phenomemon in a squirrel monkey that had been exposed to a simulated dive[17] and in a human diver. It is possible that this bleeding is associated with the hearing losses that sometimes occur with decompression sickness in human divers[9-10] and in animals exposed to similar pressure schedules[19].

One human diver suffered severe inner ear symptoms, hearing loss with tinnitus, and dizziness, some 5 hours after ascent from a dive to 100 metres of sea water for 19 minutes. The episode was clearly one of decompression sickness and it included also some joint pain (another common result of inert gases forming bubbles in tissues). It was, therefore, entirely unrelated to the middle ear problems of diving; it was caused by bubbles of inert gas. Subsequently, his hearing loss, in the left ear, was documented clinically (Fig. 3) as was his complete loss of vestibular function in the left ear. It bears mention also that a variety of recompression treatments were employed at the time of the hit; none of which alleviated the condition. This diver died suddenly, of unrelated causes, 56 days after the dive that caused his recompression damage. Figure 4 shows the new bone growth that had started to invade one of the semicircular canals of his left ear. This peculiar, and manifestly maladaptive, bone growth has been shown to occur in squirrel monkeys as a result of decompression damage to the inner ear[17], and it is significant that decompression can also

provoke this bone growth in man.

In monkeys, the new bone growth following decompression appears to be initiated by rips in the endosteum or by tiny fractures (with displacement of small chips of bone) in the bony semicircular canals[16-17]. The inner ear will produce maladaptive new bone growth in response to a variety of stimuli, and in man such growth has destroyed the inner ear following trauma to the endosteum, inner ear hemorrhage, infection, or interference with the blood supply[20-21].

Other Findings

Figure 5 shows the middle ears and inner ears of a diver who died underwater from causes undetermined. In this diver, the round window membrane was ruptured on the left side and the middle ear is almost filled with blood. On the right side, the round window membrane was intact and the middle ear contained only a little blood. The large amount of blood in the left middle ear suggests that the bleeding occurred under pressure (that is, while the heart was beating). The pressure required to rupture the round window membrane could have been produced either by a violent Valsalva manoeuvre or by an ascent with a closed glottis. It is possible that the rupture of the round window membrane was followed by a vestibular malfunction resulting in a disorientation that contributed to the death of the diver.

Conclusions

Evidence was found that, in addition to burst lung, ascent while breath holding can result in bleeding into the middle ear and rupture of the ear drum. Following decompression sickness of the inner ear, similarities were found in the histological pictures of the ear damage in man and in monkeys. The new bone growth in the semicircular canals, found here in man following inner ear decompression sickness, can be expected to attenuate the functions of the semicircular canals over the following several months, and such attenuation, when observed clinically, can be understood on the basis of events in the inner ear, without recourse to etiologies involving the central nervous system[22]. Such maladaptive bone growth might also be responsible for the "late deficit" that can follow the more common kinds of fracture or other injuries of the temporal bone[23].

Acknowledgement

This study was supported in part by the Office of Naval Research, USN, Grant N00014-77-G-0077.

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Fig. 1 Diver who died from "burst lung".

The blood on the face came from the middle ear.

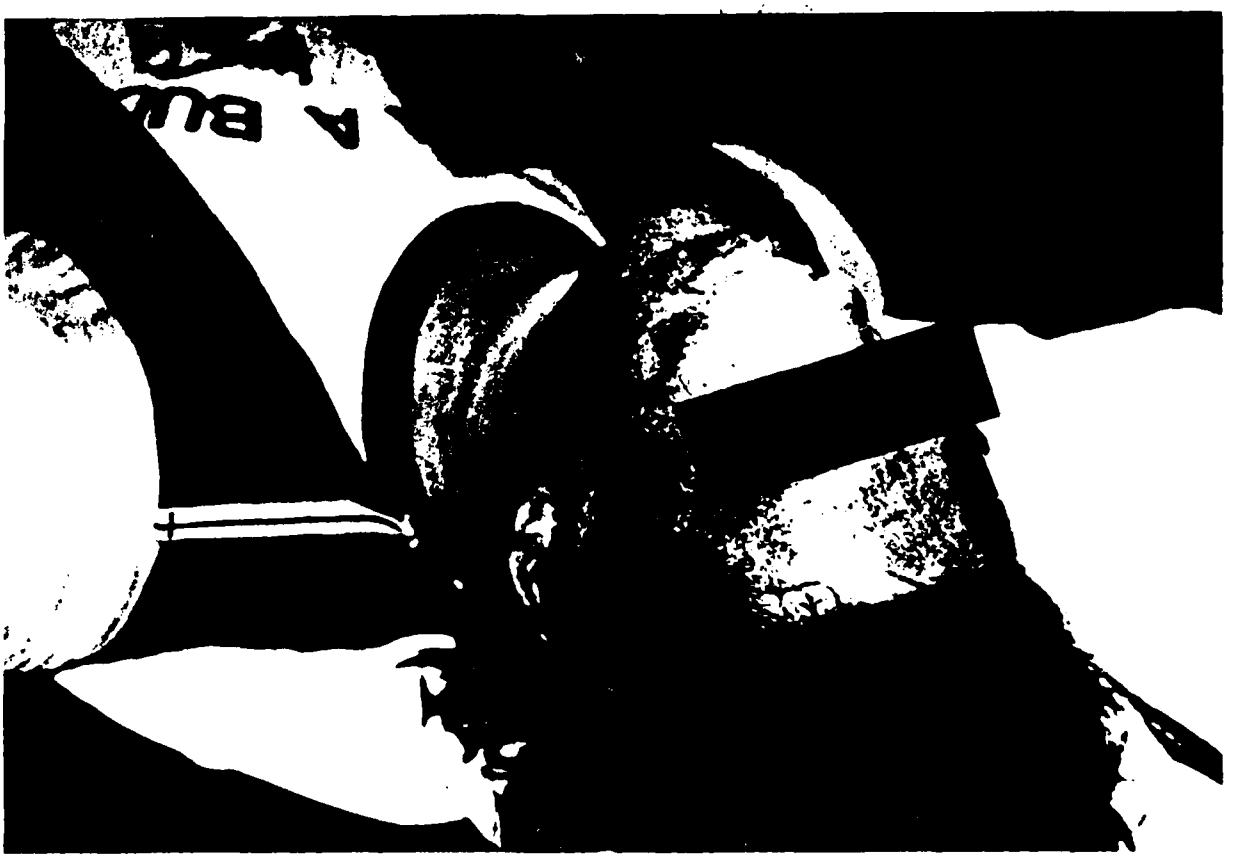
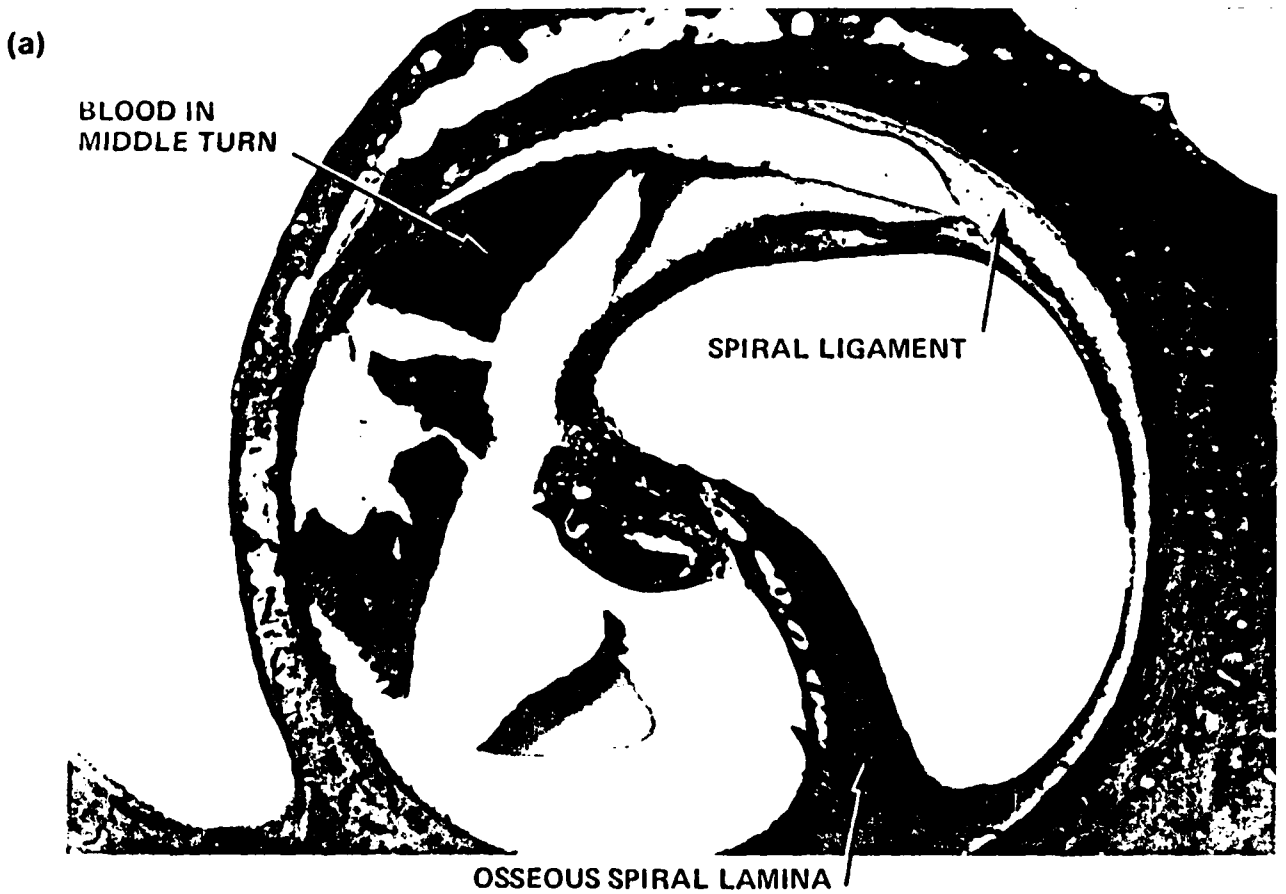


Fig. 2 Blood in the cochlea of a squirrel monkey and the cochlea of a human diver, following decompression sickness.
(a) Blood in the middle turn of the cochlea in the diver.
(b) Blood in the basal turn of the cochlea in the monkey.



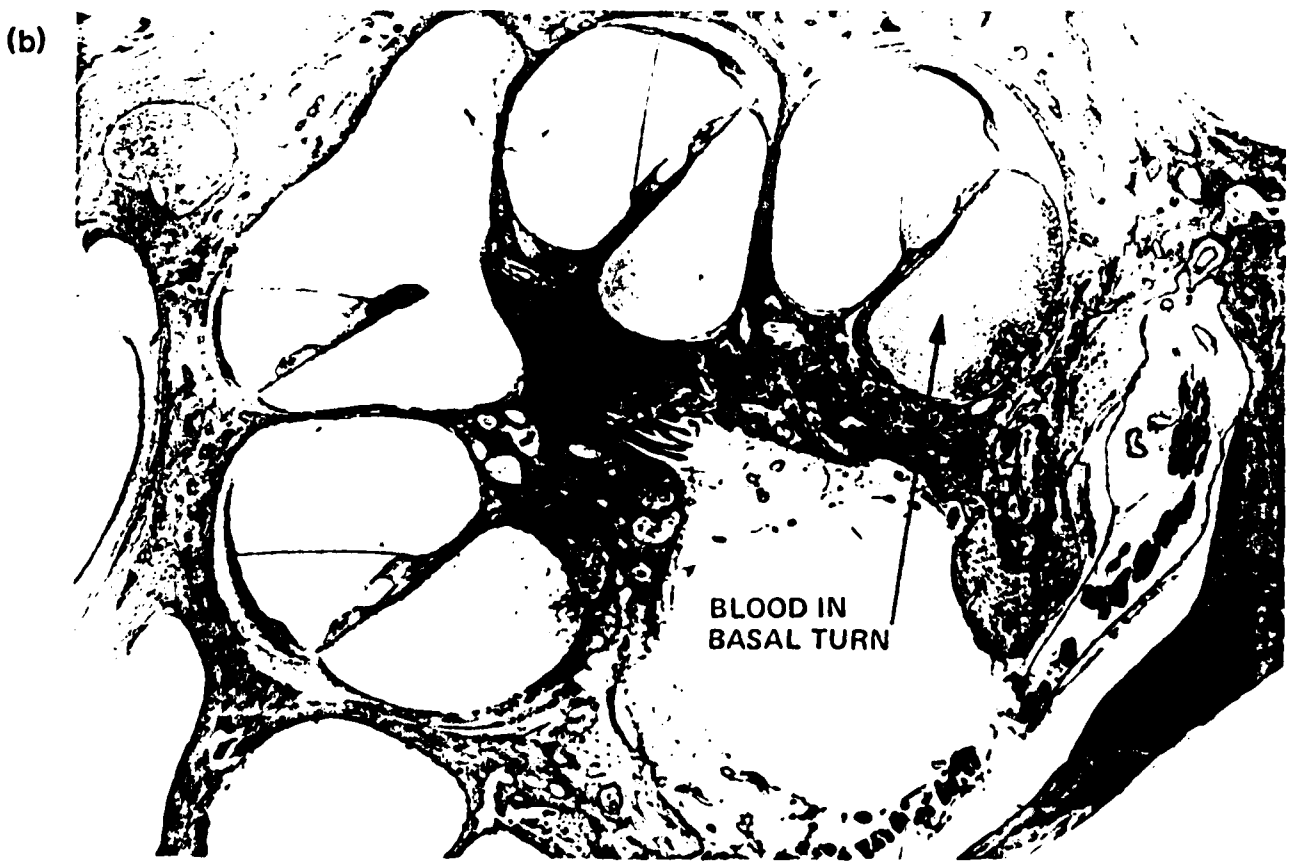


Fig. 3 Audiograms of diver following decompression sickness.

Open circles indicate near-normal levels in the right ear; the X's indicate the very large hearing losses in the left ear. This diver also had complete loss of vestibular function in the left ear.

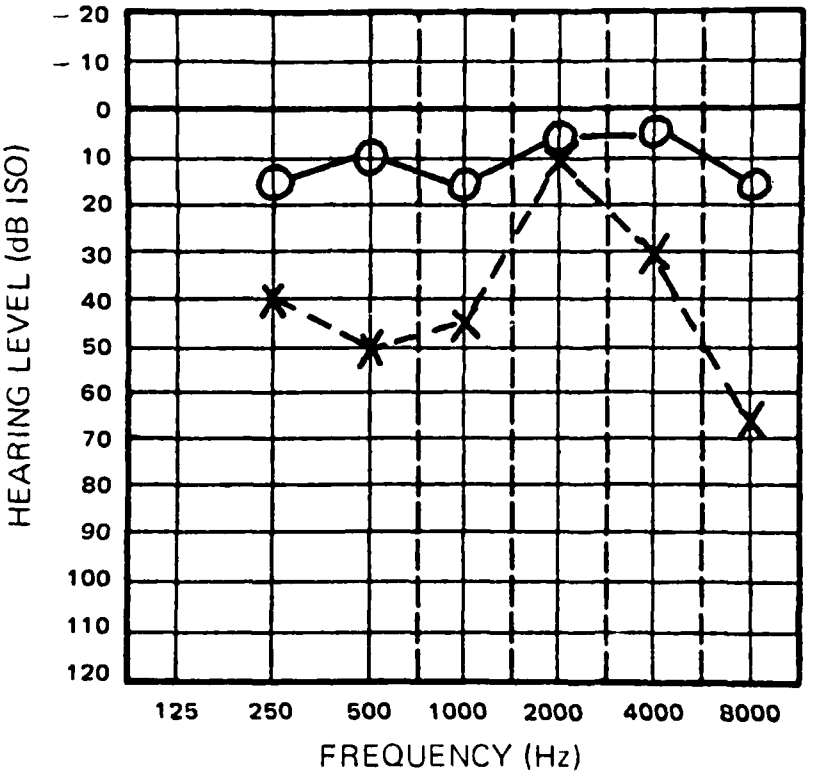


Fig. 4 Sections across a normal (top) and an abnormal (bottom) semicircular canal from a human diver.
This diver suffered decompression damage to his left ear and died of unrelated causes 56 days later. The pathological new bone growth in the (bottom) semicircular canal is typical of that produced in monkeys by decompression damage.

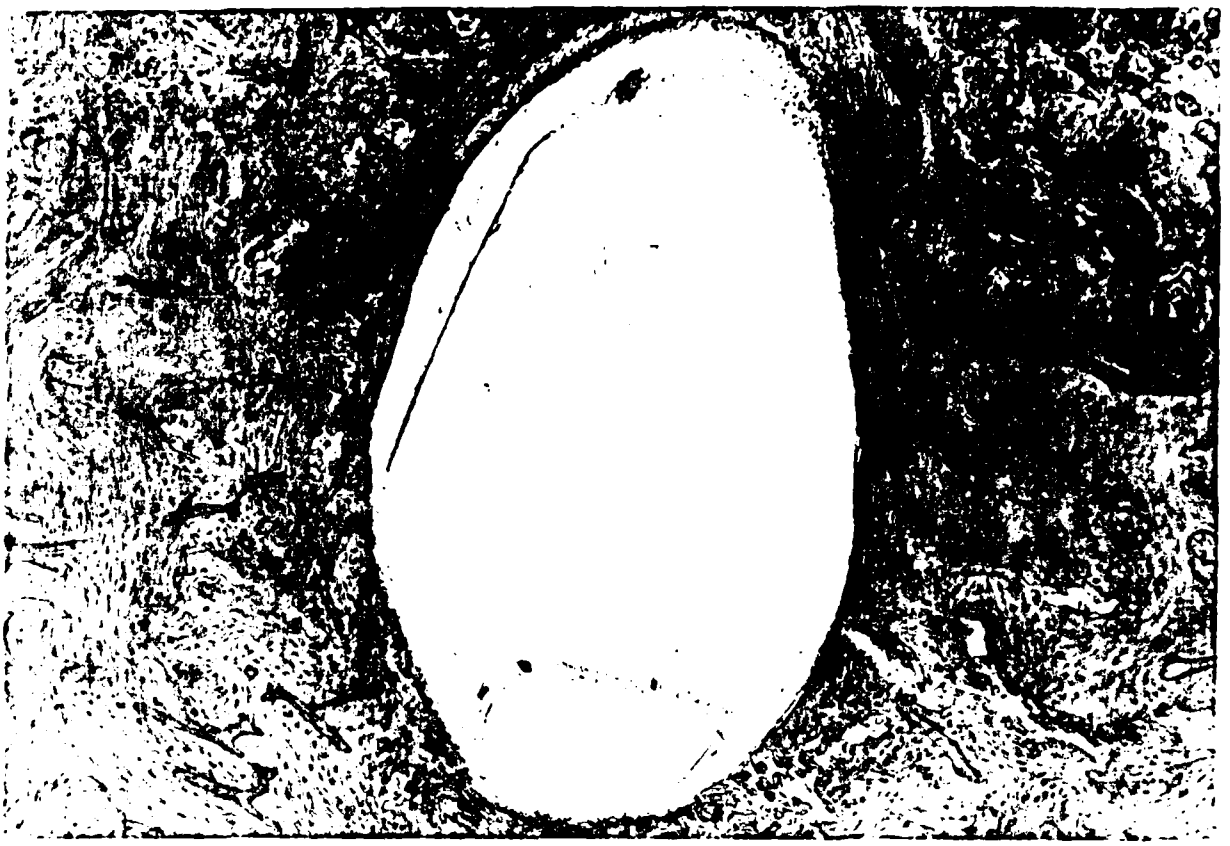
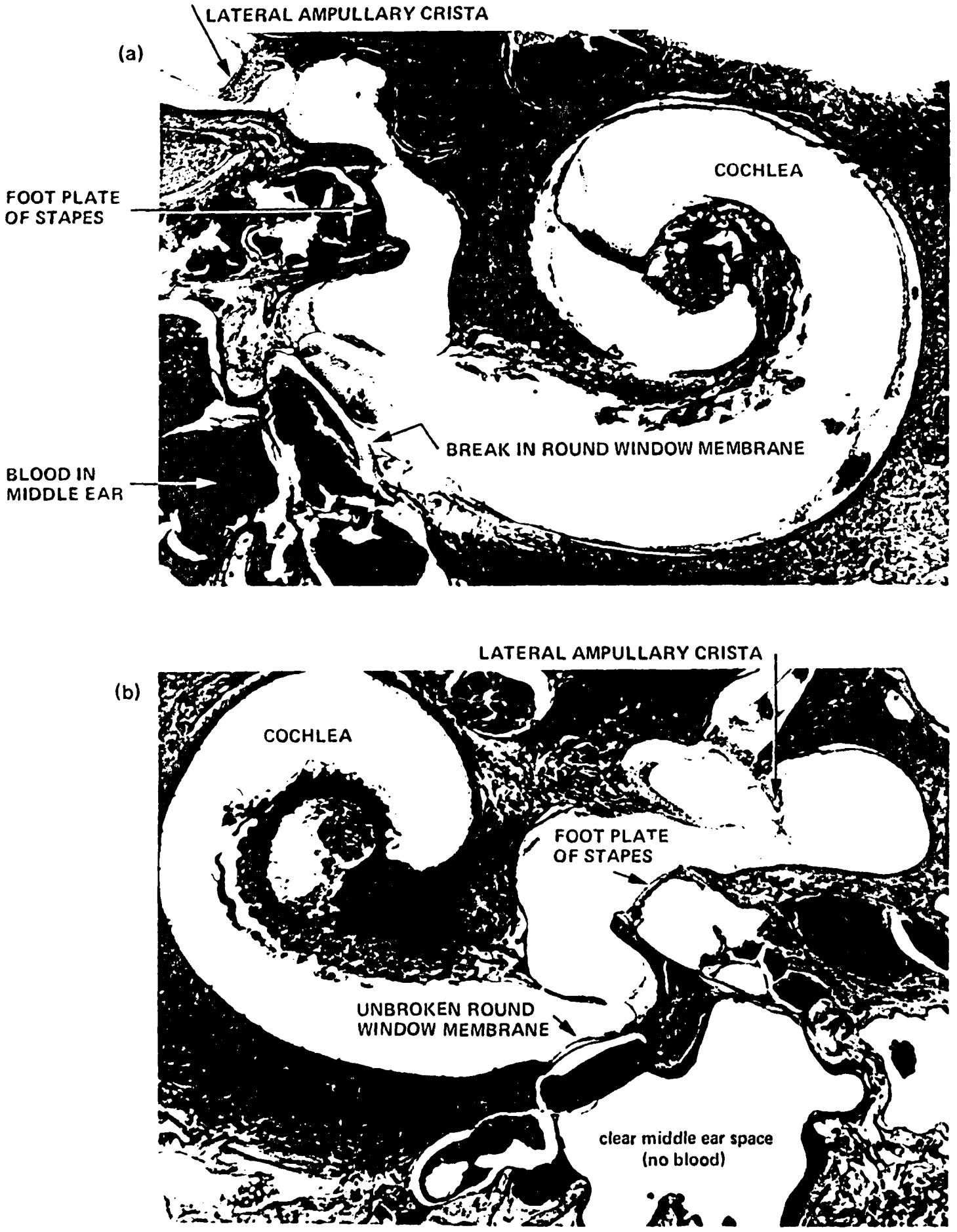


Fig. 5 Middle and inner ears, left (a) and right (b), from a diver who died underwater from causes unknown. The left ear has the round window membrane ripped and the middle ear filled with blood; the right ear is essentially normal.



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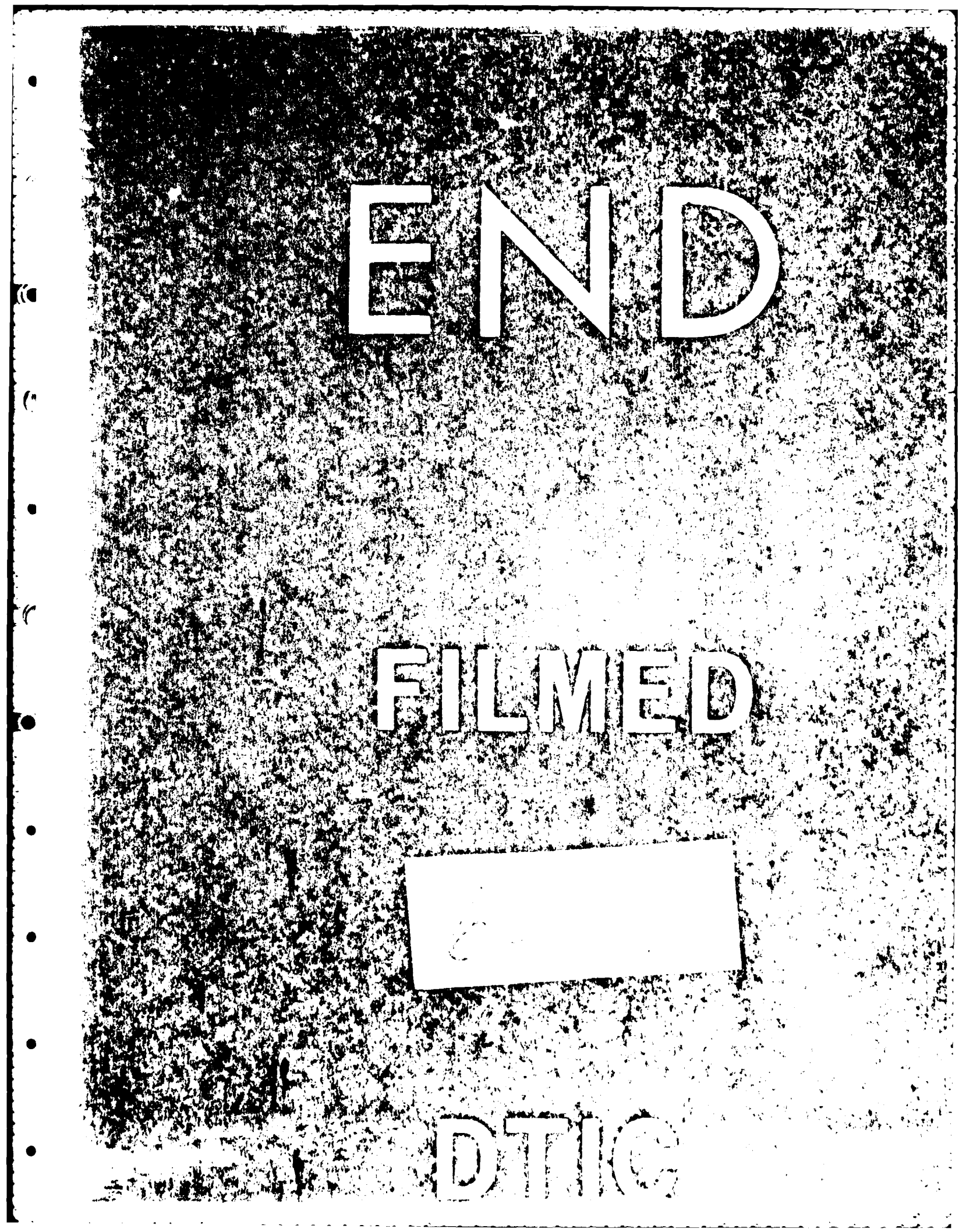
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