

THE EAR AND HEARING IN AQUATIC AND DYSBARIC ENVIRONMENTS

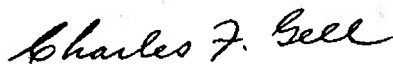
by

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NAVAL SUBMARINE MEDICAL RESEARCH LABORATORY  
REPORT NUMBER 746

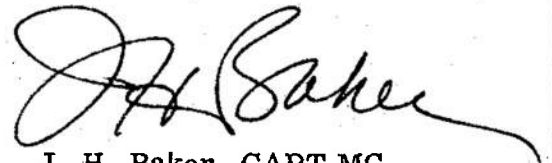
Bureau of Medicine and Surgery, Navy Department  
Research Work Unit M4306.03-2090DXC9.01

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## SUMMARY PAGE

### THE PROBLEM

To survey what is known of the ability of the human swimmer to use underwater acoustic cues, and to explore the auditory medical problems common to aquatic and dysbaric chamber environments.

### FINDINGS

The inner ear handles acoustic energy in much the same way as airborne or vibratory energy, but the conduction routes through the head are different in kind and/or degree. Airborne hearing exceeds underwater hearing acuity by 30-60 dB, being greater at the higher frequencies. Acoustic levels, potentially damaging to the ear, exist under water. Underwater speech discrimination can be quite good; but acoustic orientation, often said to be impossible, can yield minimum audible angles of the order of 10 and even of 5°. Acute and often serious medical problems of the ear and hearing in aquatic and dysbaric environments are fully discussed, with notes on prevention and general treatment.

### APPLICATION

For the use of diving technologists and others seeking to utilize acoustic cues in underwater operations, and for submarine and diving medical officers with responsibility for personal hearing conservation.

### ADMINISTRATIVE INFORMATION

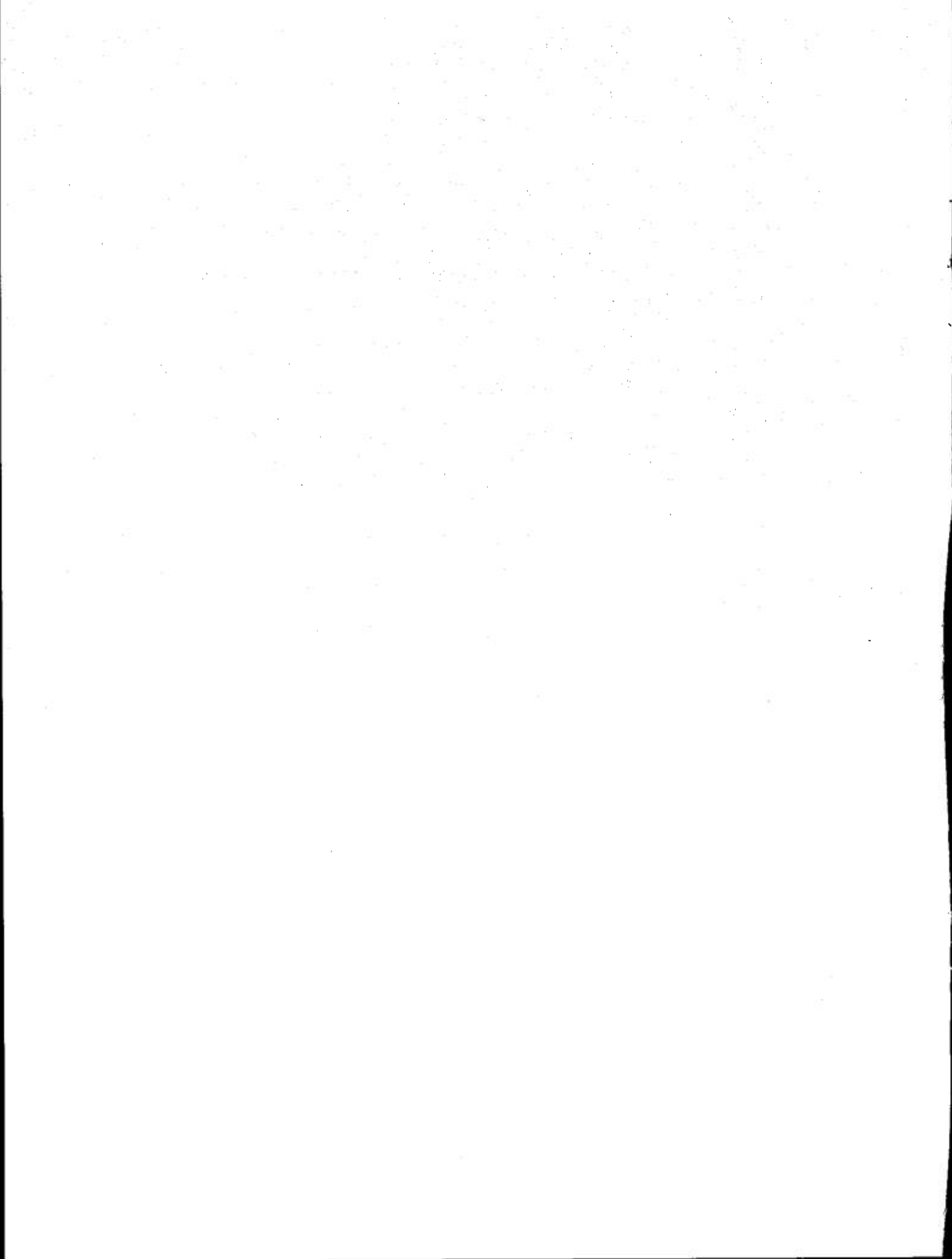
This study was partly supported by the Office of Naval Research, Contract No. RR042-09-02, NR197-018X.

This investigation was conducted as a part of the Bureau of Medicine and Surgery Research Work Unit M4306.03-2090DXC9 - Acoustic Orientation and Navigation by Navy Divers. This report is number one on this work unit. The manuscript was submitted for review on 2 May 1973, approved for publication on 29 May 1973 and designated as Naval Submarine Medical Research Laboratory Report Number 746.

PUBLISHED BY THE NAVAL SUBMARINE MEDICAL RESEARCH LABORATORY

## ABSTRACT

A non-quantitative theory of human hearing is sketched for the case of the head immersed in water, based upon what is known of hearing in air and hearing by bone conduction. A consideration is given of four routes by which acoustic waterborne energy is transmitted to the fluids of the inner ear. Quantitative data are presented from recent studies of the thresholds of underwater auditory acuity, and of the relations between airborne and waterborne thresholds. Sound levels possibly hazardous to free swimmers are suggested and the intelligibility of speech passed through water is discussed. Studies on the ability of the human to localize sounds under water are discussed. The acute otorhinological problems of the outer, middle, and inner ear are discussed in both aquatic and dysbaric environments. A bibliography of 250 items is presented which constitutes a valuable reference tool.



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INTRODUCTION

A. The Relation of Underwater Hearing to Air- and Bone-conducted Hearing.

With the invention and widespread use of SCUBA apparatus, underwater sleds, and the small submerged habitats which are the precursors of whole cities under the sea, man is thrown on his own sensory resources in an essentially hostile, though often very beautiful, environment. It cannot be assumed that his sense organs will be equally efficient in these new surroundings, evolving as they are to function in an altogether different milieu. In fact, we now know that in several ways man's sense organs are less efficient in water. Thus, it becomes important to document just what reliance, if any, we can place upon the evidence of our senses when immersed in the sea. A bibliography is available (Kennedy, 1972), and two summary papers (Edmonds et al., 1973, Adolfson and Berghage, In Press).

A theory of underwater (U/W) hearing has yet to be explicitly stated. However, one can make the reasonable assumption that U/W hearing is only a special case of bone-conducted hearing. Of course, waterborne acoustic energy would impinge on the whole submerged head at all points rather than impinge at a single point on the skull as in the case of the bone-conduction vibrator.

It has been demonstrated (see Bekesy, 1960, p. 128 ff; Lowy, 1942; Wever and Lawrence, 1952) that bone-conducted (BC) and air-conducted (AC) stimuli are analyzed in exactly the same way by the auditory system once the energy enters the cochlea by any route or routes. Thus an understanding of how the submerged human handles waterborne acoustic energy can be gained through an understanding of how the auditory system analyzes airborne acoustic energy.

The reader should first refresh his grasp of the gross anatomy and general plan of operation of the outer, middle, and inner ear (see, for example, Littler, 1965; Towe, 1966; Thompson, 1967; Davis and Silverman, 1970; and Gulick, 1971). The more advanced student should consult Zwislocki (1965) or the relevant chapters in Tobias (1970, 1972). Furthermore, since material on BC hearing is specifically applicable to problems in U/W hearing, the reader is referred to treatises on BC hearing in Barany (1938), Bekesy and Rosenblith (1960), and Tonndorf (1966).

B. The Routes by which U/W Acoustic Energy Enters the Cochlea.

The dominant route must be by way of compressional waves through the bones of the skull and the contents of the endocranium to the cochlear capsule. In addition, however, the inertia of the ossicular chain and perhaps also of the fluids of the inner ear

must play a role, and well as the spaces within the head filled with air.

(1) The Compressional or Direct Route. Acoustic energy in water finds no especial barrier at a water/head interface, since the acoustic impedances of water and of the human body are similar, so that most of the U/W acoustic energy is admitted, and transmitted through the head as waves of condensations-rarefactions. This energy may be transmitted directly through the bones and also directly through the soft tissues within the endocranium; it modifies the volume of the cochlear capsule and a displacement of the cochlear partition results. An early account was given by Herzog (1926).

In order for this energy to move the basilar membrane, and thus create an auditory sensation, it is arranged that the surface area of scala vestibuli be considerably larger than the surface area of scala tympani (5:3), and its pressure release window larger and much more compliant (20:1), so that as the acoustic energy acts upon both scalae equally, the basilar membrane is forced downward into scala tympani, and hearing results. This route is said to dominate other routes in human BC hearing above 1 kHz.

(2) The Inertial Route. The ossicles are suspended in the middle ear by attachments to the eardrum and oval window, and by four suspensory ligaments, a system which strongly prevents vibratory motion in any other mode than the normal rocking around a horizontal fore-and-aft axis when driven by the eardrum. But the ossicular chain is only very loosely coupled to the skull, and when U/W acoustic energy impinges on

the head the skull is vibrated but the ossicles by their inertia lag in motion, and when they do move they do so in the only way possible, namely, their mode of vibration to AC sound. Thus a differential movement is set up between the stapes footplate and the lip of the oval window, and the effect is exactly as if the stapes were driven in its usual AC mode. Proof of this route is due to Barany (1938):

Legoux and Tarab (1959) repeated Smith's (1943) experiment fixing the ossicular chain, and found a maximum BC hearing loss of up to 15 dB at 500 Hz, reducing to 0 dB at 2.5 kHz.

It is also possible that the basilar membrane can be displaced by a difference in the inertia of the fluids in scala vestibuli and scala tympani (see Ranke, 1953, pp. 57-59, especially his Fig. 45).

(3) The Usual Air-Conduction Route. Should there be a bubble of air in the ear canal of an U/W swimmer, acoustic energy would ensonify that bubble (create sonic energy in the medium), as it would all air-filled cavities in the head, and that airborne energy could enter the cochlea through the usual ossicular route. It is therefore on a first look surprising that U/W thresholds have been reported (Hollein and Brandt, 1969) not to vary appreciably whether there is or is not air in the ear canal. It would seem that this route is not of great importance in U/W hearing. However, loading the eardrum with water has other effects besides displacing the usual bubble of air; there would be a drastic change in the compliance of the eardrum. Tonndorf (1966) showed



that completely filling the meatal cavity in the cat created a loss of 20 dB to BC tones at low frequencies. Of course, loading the eardrum with water does not entirely eliminate ossicular inertia, as the other end (the stapes footplate) is still relatively free to move.

Bekesy (1941) felt that the air bubble in the meatus in response to a BC sound was ensonified by way of differential motion of the meatus and lower jaw. This was corroborated by Franke et al. (1952). On the other hand, evidence that it is the BC vibrations of the bony walls of the meatus which largely drive the meatal air, rather than the lower jaw, was obtained by Allen and Ferandez, 1960; Brinkman et al. 1965; and Tonndorf et al. 1966. The latter evidence would explain why U/W hearing is no better with the lower jaw loose than with it clenched.

(4) Air in the Middle Ear Cavity. No one has explored in the animal the effect of this route with U/W sound; but Tonndorf (1966) showed that hearing was not appreciably changed when the bulla (a part of the middle ear cavity in the cat) was opened, thus allowing the sonic energy in the middle ear to "leak" outside rather than enter the cochlear fluids through the round window. Tonndorf felt this route was negligible in BC hearing. However, in a strong U/W acoustic field the sound pressure levels in the tympanum may be quite high also, and a by no means negligible quantity of energy may enter the cochlea.

Groen (1962) has raised the possibility that the air in the tympanum must control the compliance of both the eardrum and the round window, each in pro-

portion to its size. It should be that U/W hearing would change, in the direction of better low-tone hearing due to an increased compliance, if the tympanum were filled with helium or one of the lighter gases.

Waterman and Smith (1970) took AC audiograms on Ss breathing air or an 80%-20% HeO<sub>2</sub> mixture. When the Ss first donned the breathing mask they ventilated the middle ear and immediately took interrupted-tone audiometry in a soundproof room. At 125 and 1000 cps there was no audiometric loss, but all three Ss showed losses (5-11 dB, mn=8.0) at 8 kcps. However, after 30 min of HeO<sub>2</sub>, this shift had disappeared. This experiment showed that He of itself did not have any systematic effect on audition, but that a change in compliance of the middle ear did immediately deteriorate high-tone hearing. There is no ready explanation as to why it did not improve low-tone hearing, nor why the effect disappeared over 30 min.

Brandt (1967) found about a 5 dB degradation of threshold at all frequencies in divers at 105 ft. when breathing HeO<sub>2</sub> than when breathing compressed air. Inasmuch as Waterman and Smith showed that the gas itself had no such effect, and any physical change such as the compliance of the eardrum should have a frequency-dependent effect, this finding is not understood at present.

## II. ABSOLUTE THRESHOLDS OF U/W HEARING

### A. General

A number of workers have immersed divers in water and taken some sort of

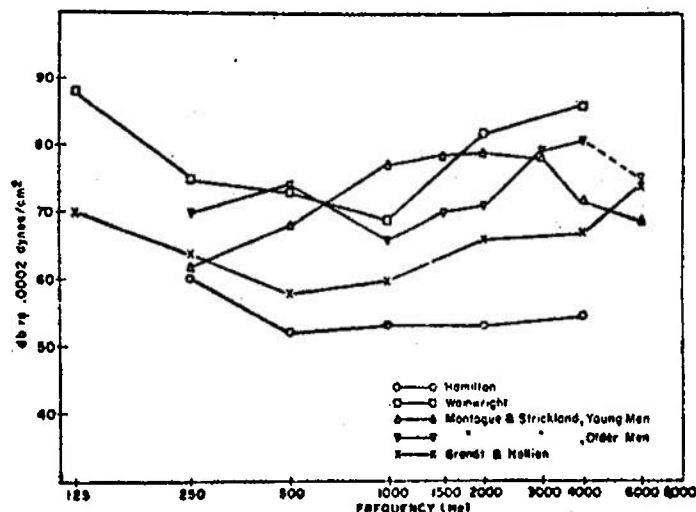


Fig. 3. Human underwater hearing. From Hamilton, 1957; Wainwright, 1958; Montague and Strickland, 1961; and Brandt and Hollein, 1967.

differences do likewise. This paper adds the important information of significant correlations between individual AC audiometric losses vs AC-U/W differences: the more the AC loss the less the AC-U/W differences. This is explainable only if U/W hearing is largely by BC hearing.

Brandt and Hollein (1967) submerged 5 M and 3 F experienced young divers and collected U/W thresholds under ideal environmental conditions and with sophisticated test and calibration techniques. Their data for two diver depths are in Fig. 3. The AC thresholds reported were shown to be contaminated by 10-15 dB at 500 Hz by unavoidable noise in the field, but when this is allowed for, the AC-U/W differences were 31 dB at low tones at 56 dB at 8 kHz, a bit less than often reported between 1-4 kHz. Brandt (1967) with the same equipment and much the same divers had found a 60-dB AC-U/W difference in 2 kHz.

Brandt and Hollein made the important observation that the peak of U/W sensi-

tivity is at a lower frequency (500-1000 Hz) than that of AC (1-4 kHz).

Smith (1969) immersed 13 experienced divers with known-normal AC and BC audiograms to a head depth of 15 ft and collected U/W thresholds. The data for the 8 normals (Fig. 4) interweave  $\pm 5$  dB with those of Brandt and Hollein (1967). Smith corroborated Brandt and Hollein on the frequency region of maximum sensitivity, and added that in his data (see Fig. 5) this frequency region was in U/W hearing not only displaced downward an octave, but also was a much narrower figure.

For 5 Ss with hypacusis, Smith found that a depressed AC alone does not affect U/W threshold, but a depressed BC threshold leads to some relative loss of U/W sensitivity.

#### D. Effect of Air in External Auditory Meatus.

The 3 divers of Reysenbach de Haan (1956) found that a bubble of air in the

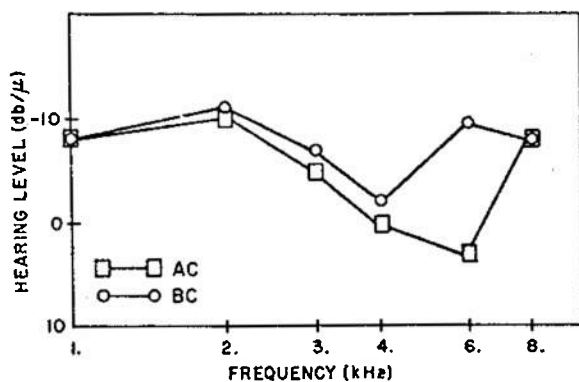


Fig. 4. Underwater hearing acuity of 8 men from 1-8 kHz with known normal BC hearing. From Smith, 1969.

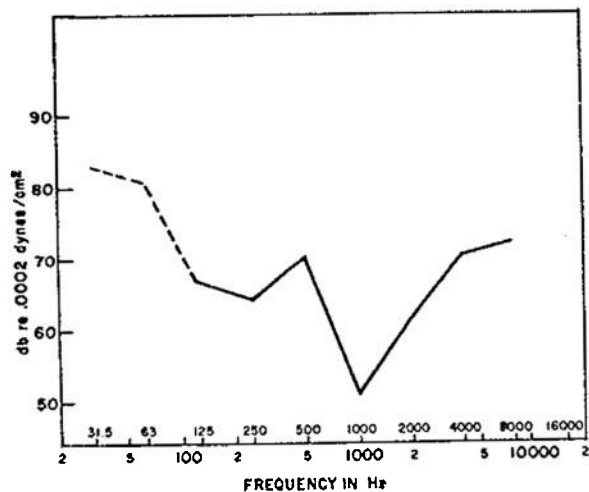


Fig. 5. Underwater acuity of 5 men from .125-8 kHz with no BC hearing loss. From Smith, 1969.

meatus degraded threshold at 1 kHz by about 21 dB, but improved threshold at all higher frequencies existed up to 15 kHz.

The divers of Hamilton (1957) and of Wainwright (1958) experienced no change in loudness of U/W sounds when they put their fingers in their ear canals, but no especial attempt had been made to

eliminate air bubbles in the canals, and whether any were present is not known.

Hollein and Brandt (1969) immersed 4 M and 3 F experienced divers to 12 ft and collected U/W thresholds with especial care to retain or exclude a bubble of air in the canals. Fig. 6 shows the mean AC and U/W data. The effect of the bubble was negligible except at 250 Hz, where threshold was better by 6 dB without the bubble. This may eventually tell us something about the compliance of the eardrum and middle ear at 250 kHz or it may have been a chance reading. Hollein and Brandt suggest the data mean that U/W hearing is exclusively by (compressional) BC and that the middle ear does not contribute (see also Hollein and Feinstein, 1972; Feinstein, Hollein and Hollein, 1972; and Hollein, et al., 1973).

#### E. Effect of Depth.

Brandt and Hollein (1969) pursued the matter of U/W hearing in 4 M and 2 F experienced divers, adding the information that water depth from 35 to 105 ft was not significant. Their results (Fig. 7) corroborate earlier data as to absolute U/W thresholds and AC-U/W differences. They explain the somewhat elevated U/W data at 105 ft as first-session practice effects.

### III. DIFFERENCES BETWEEN AC vs U/W HEARING ACUITY

The best estimates available to date are that AC hearing exceeds U/W hearing by 30-60 dB, somewhat greater at the higher frequencies. This represents a

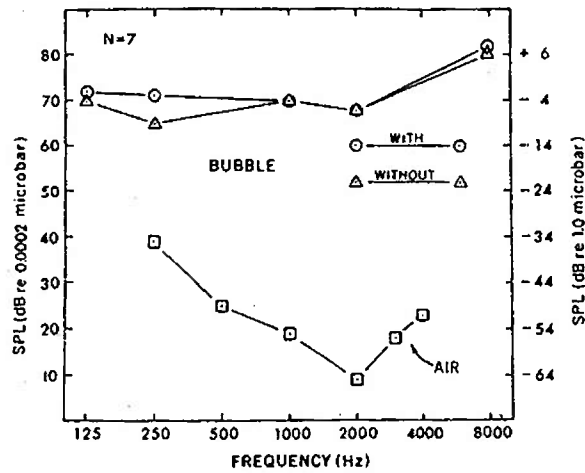


Fig. 6. AC and U/W audiograms, the latter with and without air in the meati. From Hollein and Brandt, 1969.

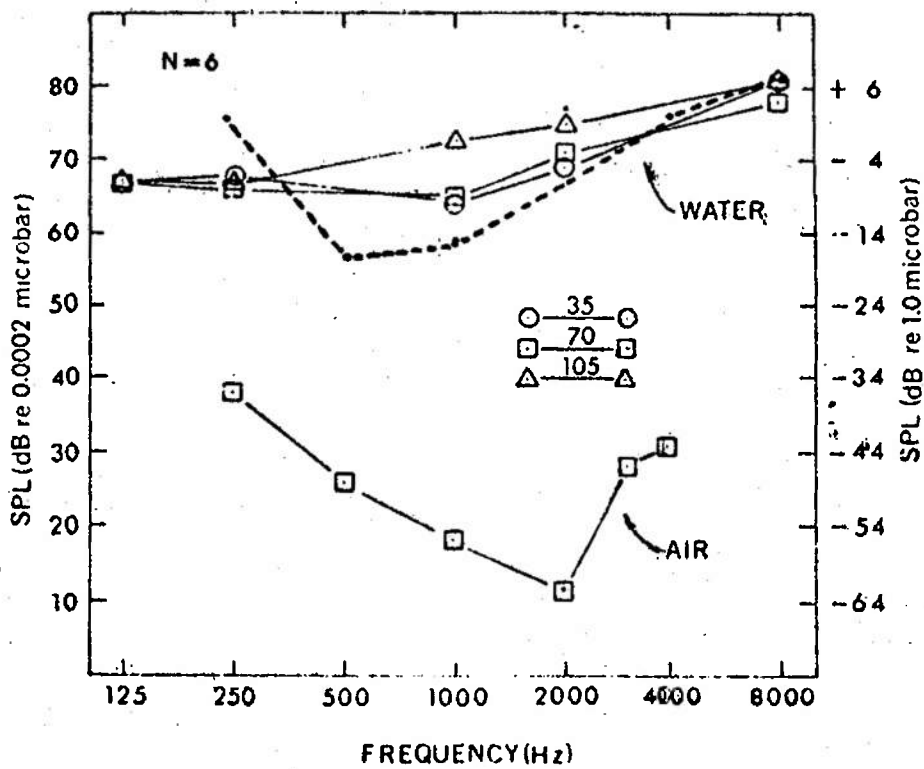


Fig. 7. AC and U/W audiograms, the latter at different depths. From Brandt and Hollein, 1969 -- Similar data on 7 divers at 30 ft depth, from Hollein and Feinstein, Comm. Sci. Lab. Report #40, 1972, Univ. of Florida, Gainesville.

seeming inefficiency of the AC-U/W mechanism, where the threshold SPL in air is much less than in water. However, the efficiency of U/W hearing is seen to be just as good as AC when one reflects that AC and U/W hearing are about equally sensitive to the same vibratory amplitudes, the one of the eardrum and stapes, the other of the skull.

Zwislocki (1957) took special pains to insulate human ears from AC sound, by using special ear plugs, and took audiograms in air by free-field loud-speaker. The difference with and without the plugs was taken to be threshold by BC and lies (Fig. 8) 45-68 dB below AC threshold. This AC-BC difference is similar to the AC-U/W difference, and lends some credence to a correspondence between BC and U/W hearing.

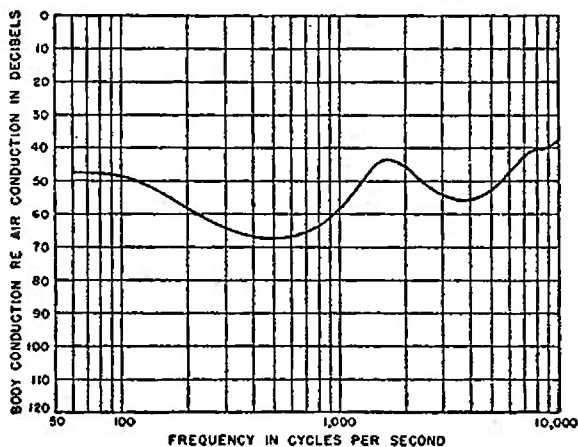


Fig. 8. Relation between AC and BC hearing in a free field. From Zwislocki, 1957.

In the seal, Møhl (1968) found that the difference between threshold AC vs U/W hearing was only 15 dB. Whether as compared with man's the seal's AC hearing is somewhat poorer, or his U/W hearing relatively better, cannot now be confidently stated. If its U/W hearing is relatively better than man's, as seems probable, it would be instructive to learn in what respects its hearing mechanism has evolved to match its U/W habitat.

#### IV. MAXIMUM INTENSITY FOR SAFE U/W HEARING

It is within the capability of present amplifier and transducer technology to create pure U/W tones loud enough to damage U/W hearing permanently. This sonic hazard is in addition to the effect of U/W explosions which create steep-front pressure waves and can easily damage body tissues. Montague and Strickland (1961) found that 50% of 17 young experienced divers signalled that, when they faced the transducer, a tone of 1500 Hz at about 172 dB re .0002  $\mu$ bar was so annoying that they did not want it increased further. Fig. 3 shows this annoyance level at 1 kHz to be about 100 dB above threshold, much as it is for AC hearing. Above 165 dB all Ss reported some distortion of the visual field. A diver's hood created about a 10-dB attenuation, but a hole in the hood as small as 2 inches at the forehead destroyed this protection.

Harris (1960) established that an U/W level of 125 dB re  $2 \times 10^{-4}$   $\mu$ bar for certain sounds was loud but not noxious. Smith (1964) stated the distances a swimmer with or without a wetsuit hood

might safely approach a particular sound source of 175 dB re  $2 \times 10^{-4}$   $\mu$ bar (see also Smith and Linaweaver (1966)).

## V. U/W AUDITORY DISCRIMINATIONS

Differential thresholds for frequency and intensity have not been collected under water (though see Thomas, 1973, for data in hyperbaric conditions); but intelligibility for waterborne speech has been assessed. It is common knowledge that near-perfect U/W speech communication is easily achieved with high-quality equipment and good environments.

Brandt and Hollein (1968) with a Navy J9 underwater loudspeaker found 95% correct responses for monosyllable intelligibility at 30 dB re the weaker intensity for 50% correct response. The latter point was achieved at 80 dB re  $2 \times 10^{-4}$   $\mu$ bar, about 13-15 dB more than the U/W thresholds for pure tones at the speech frequencies: this U/W puretone vs speech difference is about the same as the difference in AC hearing.

Such good intelligibility could not arise if U/W hearing suffered much greater distortions than AC hearing. But the safety factor in voice communications is such that at least usable communications can be achieved with quite "noisy" systems. Alexander (1970) summarizes the state-of-the-art for shouting directly into the water or for the use of impedance-matching devices and transducers. Many commercial voice communications units are now available for a variety of U/W situations. Hollein and Coleman (1969) evaluated four such units with two divers 30 ft apart at 30 ft depth. Per cent

monosyllables correctly heard were from 57-64%; i. e., usable but not good.

Unprocessed voice communications by U/W telephone (AN/UQC-1) through the water between two submerged submarines is usable over a range of several miles (Murry and Strand, 1970).

Of course, if the frequency is lowered so that transmission distances may be increased, two debilitating effects occur: (1) a single path through the water is subjected to reflection from many facets of the medium, such as irregular bottoms or layers, and a transmitted pulse will be "smeared" out in time so that a single word will be stretched out for an appreciable fraction of a second; and (2) there may be several such paths, so that a smeared word would be repeated three or more times. As to (1) the smear through a single channel, Sachs et al. (1969) simulated time smears of up to .665 sec for single words. Intelligibility of 92% at 0 smear dropped to a plateau of 75% at a smear of .2 sec, but the authors predicted a much more serious drop in intelligibility for connected discourse if the smear should exceed .100 sec.

A problem in U/W talking is the obvious restriction of the diver's mask and mouthpiece. Several companies have attempted to solve these problems, some with proprietary solutions. Morrow and Brouns (1971) give by far the most sophisticated measurements and discussion (see also Hollein and Doherty, 1970; McCrory and Jenkins, 1967; Ioffe and Dmitriyev, 1972).

Much has been written on the "Donald Duck" quality of speech uttered with a

HeO<sub>2</sub> breathing mixture, and helium-speech unscramblers are now commercially available. A symposium on processing HeO<sub>2</sub> speech was held (Sergeant and Murry, 1972). A number of papers have looked specifically at the interaction of depth and HeO<sub>2</sub> breathing (Gerstman et al. 1966; Fant et al. 1971; Murry, 1971b). Hollein et al. (1973) found that helium speech at 1 ATA was reduced about 50% for each 200 ft of simulated water depth, down to 600 ft. Furthermore, the most intelligible talkers at depth were not necessarily the most intelligible on the surface.

## VI. LOCALIZATION OF U/W SOUNDS

All swimmers have known that U/W sound localization is poor, and many authors have concluded it to be impossible because it would seem all those binaural cues are removed which alone allow us to localize sounds in air. But in the first place it has been known since 1900 that monaural localization in air can be very good indeed in many situations, and in the second place it is known that sea-going mammals have very good directionality, so that it would seem odd if the human head was not capable of similarly compensating in some manner.

Ide (1944) fitted some swimmers with helmets isolating the two ears and reported some success in all ten subjects in homing on sounds. Discouraging comments on the possibilities of localizing sound under water were made by later workers (Reysenbach de Haan, 1956; Hamilton, 1957; Howard and

Templeton, 1966) but Feinstein (1966 a, b) found minimum audible angles of about 10° - 30° in one bare-headed diver (in this man the meati filled with water), though another could not distinguish 45° L from 45° R. However, he corroborated Ide that when he fitted the latter man with a 1/2-inch thick helmet, leaving apertures over the ears, the subject experienced a binaural sensation and could then point to an U/W sound source. In this case the minimum audible angle (m.a.a.) was ± 10-15°. The diver had air bubbles in the meati.

Bauer et al. (1965) arranged two directional pickups attached to the diver's head. Each pickup was split and led to the earphone on its proper side, and to the earphone on the opposite ear after a time delay tailored to the case of AC hearing, and reduced in intensity to simulate the head shadow effect in air. The prototype at least sounded promising. Bauer and Torick (1966) immersed two microphones in water and created the effect of back-to-back cardioid pattern receptors 10 cm apart, then shifted phase through the cross-coupling network; a person listening with earphones to the binaural outputs could tell the orientation of an underwater sound projector.

Feinstein (1966a) found that one bare-headed diver could furnish a m.a.a. of only 10° for U/W clicks in a relatively echo-free tank, and only 5° in a reverberant tank. Another man could approach this performance only if he wore a neoprene helmet with small ear-holes. Again, Feinstein (1969) reported that 4 divers with a hood and ear-holes yielded average m.a.a. of 15° for a faint 3.5-kHz tone, while two

bare-headed divers yielded m.a.a. of  $5.0^\circ$  and  $2.5^\circ$  for pulsed white noise.

Hollein (1969, 1971) had 6 fairly experienced divers immersed in water at 50 ft with bodies fixed but heads free to move. Underwater loudspeakers were energized with trains of pulses at  $0^\circ$ ,  $\pm 45^\circ$ , and  $\pm 90^\circ$  from midline, at an intensity at least 40 dB over threshold. Ss indicated which speaker was energized. Where 20%-correct is chance, mean score was 24.0 at 6 kHz, 37.3 at 1 kHz, 52.7 at 250 Hz, and 57.3% for broadband noise. Most of the errors were no more than  $45^\circ$  in extent. Evidently some underwater localization capability existed.

Andersen and Christensen (1969) used 7 normal-hearing skindivers without hoods and with ear canals filled with water. When immersed they judged the second of two 1-sec pulses "L" or "R" of the first. Results were much the same in the open sea at 6 m head depth and in a more reverberant harbour enclosure (porpoise pen) at 3 m head depth. Mean correct responses were hardly above chance at 2 kHz, even when the two transducers were  $\pm 90^\circ$  from midline, but at 1 kHz there was evidence of localization, while at 4, 8, and 16 kHz there was slowly increasing ability with frequency. The best diver achieved 70%-correct at 1, 8, and 16 kHz even when the transducers were only  $\pm 10^\circ$  off midline ( $20^\circ$  minimum audible angle).

Leggiere et al. (1970) immersed 6 divers and reported a weak directionality, namely, a standard deviation of pointing error of  $58^\circ$ , better at low frequencies (600, 800 Hz).

Hollein et al. (1970) amplified Hollein's (1969) pilot experiment with 17 divers who yielded mean correct scores of 50% at 250 Hz, 39% at 1 kHz, 34% at 6 kHz, and 53% for broadband noise. Later, 7 Ss repeated the runs with their heads fixed, with quite similar results except that a drop to 37% occurred at 250 Hz. Knowledge of results on each trial in a later procedure increased these scores by over 30%. Hollein (1971) has himself summarized these and other studies, indicating that some U/W localization does exist in the bare-headed human.

Norman et al. (1971) arranged 7 underwater projectors in  $30^\circ$ -steps around persons submerged in a reverberant pool. Where localization of clicks at chance was 14%, two persons yielded 42% correct with bare heads, and 40% with hoods with ear-holes, but only 27% with small neoprene ear-patches fitted over the pinna. One S with only 1 ear-patch fell to chance performance. The authors speak of a mental cue for localization. In a fuller account of his Canadian work, Feinstein (1973) submerged 8 normal-hearing divers 9.1 m down in water 45 m deep with a soft mud bottom (i. e., highly non-reflective), and collected L-R judgments for bursts of 3.5 and 6.5 kHz and white noise. Even after training, 2 Ss could not operate at better than chance performance (the author surmises this was for reasons of personal anxiety) but of 4 Ss who completed a training period, surprisingly good uniform ability was found, with mean m.a.a. of  $11.3^\circ$  and  $11.5^\circ$  for 3.5 and 6.5 kHz respectively, and  $7.3^\circ$  for white noise. He points out that with an



inter-cochlear distance of 10 cm,  $\Delta t = 11 \mu\text{sec}$ , or just about the inter-aural discriminable time, and feels this weighs against the suggestion of Norman et al. (1971) that meatal conduction is essential for U/W localization.

Smith, et al. (1973) immersed 5 divers to a head depth of 5 m in the open sea, with transducers movable along a chord. They were asked to judge whether the second of two 1-sec pulses of narrow-band noise centered at 1 kHz was "L" or "R" of the first pulse. S/N was about 19 dB. Enough trials were achieved at various angular separations that true minimum audible angles could be computed. M.a.a. on the average varied from  $5.72^\circ$  to  $3.86^\circ$  over 4 independent sessions; across all sessions the divers achieved means of 2.70, 3.93, 4.63, 6.45, and  $8.55^\circ$  (overall mean of  $5.25^\circ$ ). These values compare very favorably with the  $1^\circ$ - $3^\circ$  reported for air (Mills, 1958; Harris, 1972).

In the face of such results, the conclusion is inescapable that U/W m.a.a. can be precise enough to be quite usable. Here, as is often the case in psychoacoustics, data has outstripped theory. Till now, theory has predicted that little or no U/W localization is possible. Many control experiments still must be run to determine just which aspects of the stimulus the listener actually uses in U/W localization.

The speeds of sound in water, bone, and body tissues and the longer wavelengths make it seem unlikely that timing (and of course phase) cues are processed. However, Neuss (1969) made some calculations on a stylized skull

considering speeds of sounds through bone and through water, showing that some localization of sounds under water could exist on the basis of times for a sound impinging upon the skull to reach the cochleae. Assuming a sound through the water to the R of the immersed human head, Neuss calculates a differential delay through water to the far (L) side of  $10 \mu\text{sec}$ , but points that "this value...lies in the boundary range of the resolving capacity of sound localization." His sketches are not very convincing since he assumes that underwater sound reaches the L cochlea, for example, from the L mastoid rather than from all points on the skull. But if we can extend his sketch to the case of all the ensemble of paths from the whole half of the skull nearest the sound source reaching the R ear first, by as little as  $5 \mu\text{sec}$ , a directional time cue would in fact be available.

There are equally likely to be differences in intensity at the two cochleae which could be differentiated. Isele et al. (1968) showed that there were differences of 1-8 dB in transmission through to the cochlea from five points of attachment of a BC vibrator to the skull, and that these points interacted with frequencies from .05 - 4 kHz. Now inasmuch as the cochlea is indifferent to where on the labyrinth acoustic pressure is applied, we may say that all the energy over the whole skull converges on and is in some way summed at the basilar membrane on each side. It would not be surprising, then, if the total energy impinging on one cochlea were of the order of 0.5 dB or more different than the total on the other cochlea. Such an interaural difference is quite adequate to affect the two auditory

nerve codes to yield a sensation of dimensional space. Thus either time or intensity cues, or both, may yield some U/W directionality.

## VII. EFFECTS ON THE EAR OF AMBIENT PRESSURE

### A. Hyperpressure in the External Auditory Meatus.

If the external auditory meatus is occluded, as with an earplug or by a diving hood, a diver who has "cleared" his ears under water will have a negative relative pressure in the ear canal, and a "cupping" effect may lead to an otitis externa. In diving circles this is known as "reverse ear." Jarrett (1961) showed that a depth of 30-50 ft (120-150 mm Hg) would cause "reverse ear." There are cases in which an external ear barotrauma has occurred on even a shallow dive (5-6 ft). As the bubble gets smaller with pressure, the eardrum bulges outward, with possible hemorrhage and otalgia. Treatment is simple and conservative, and divers should simply make sure their canals are open.

### B. The Effect on the Audiogram of Static Pressures Equalized Across the Eardrum.

(1) Human Material. References cited in the introduction make it clear that a pressure-release mechanism for the middle ear cleft exists in the nasopharyngeal (Eustachian) tube. Yet even where this tube is patent and adequately ventilates the middle ear, changes occur in the conductive aspects of hearing when the atmospheric pres-

sure is altered. If it is decreased, as in an airplane ascent, or the atmosphere is rendered less dense by substituting helium for air, some slight changes in hearing may occur. If it is rendered much denser, as in hyperbaric chambers or by water immersion at depth, more drastic changes may occur. In this regard, as in others, the problems are common to subaquatic and to hyperbaric chamber medicine.

Obviously the density of the bubble of air into which the eardrum looks will partially govern the eardrum's response and thus the hearing of the individual studied. Adolfson and Fluor (1965) (See also Fluor and Adolfson, 1966) took audiograms on 26 experienced men exposed to 11 ATA in a hyperbaric chamber. BC audiometry with a vibrator affected in no way by the pressure revealed no sensorineural shifts, but AC audiometry (the earphone response carefully corrected for the pressures used, with a Bruel and Kjaer microphone itself calibrated for pressures) showed conductive losses increasing rather regularly with pressure and amounting to 30-40 dB in the speech frequencies.

Oliver and Demard (1970) found that two divers on HeO<sub>2</sub> had reversible losses especially at lower frequencies at 12 ATA. Miller (1971) collected cochleagrams from cats, finding 15-20 dB losses at 2-8 kHz at 10 ATA, less at 5 ATA. A puzzling finding was a slight (7 dB) loss in BC sensitivity.

Farmer, Thomas, and Preslar (1971) studied 6 divers down to 19.2 ATA breathing a HeO<sub>2</sub> mixture. AC and BC hearing levels were measured

from .25 - 4 kHz at 5 different depths during the 12-hr descent, twice during the 6-day stay at 600 ft, and at 6 depths during the 7-day decompression. Pressures were always equalized across the eardrum. No BC changes were seen, and no AC changes at less than 100 ft, but at 600 ft conductive losses averaged  $14 \pm 11$  dB for .25, .5, and 1 kHz, but  $25 \pm 13$  dB for 2 and 4 kHz. Data were less variable after 6 days on the bottom, but the mean losses at .25, .5, and 1 kHz increased to  $26 \pm 8$  dB. The authors explain such changes as due to an upward shift of the ear resonant frequency and greater impedance mismatch with helium.

Appaix and Demard (1972) collected Bekesy pulsed-tone AC and BC audiograms before and during exposures on very experienced divers in a dry chamber during saturation dives to 41 ATA (1312 ft). A Telephonics TDH-39 earphone was used, calibrated in a Bruel and Kjaer Type 4152 artificial air at various pressures in O<sub>2</sub>-He up to 60 bars. Calibration corrections were made from 10-18 dB to the AC audiometric data. In a saturation dive at 26 ATA (820 ft) four divers showed low-frequency AC-only (i.e., conductive) losses of 15 to 25 dB at 1 kHz and at lower frequencies, but at 1.5 kHz and higher frequencies there was no change in AC hearing, nor were there any changes at any frequency in BC hearing. However, in a saturation dive at 41 ATA the low-frequency conductive losses were accompanied by a high-tone dip again of 15-25 dB at 3-6 kHz, sensorineural in nature (i.e., BC losses of that magnitude also developed at 3-6 kHz). At first glance this latter dip looks like TTS to chamber noise,

often intense during deep dives (see Summitt and Reimers, 1971; Murry, 1971; Lauderdale, 1973) but the authors do not comment on this possibility.

Thomas, Summit, and Farmer (1972) took AC and BC hearing levels on 33 divers on 1 saturation dive to 300 ft, 4 dives to 600 ft, 2 dives to 850 ft, and 1 dive to 1000 ft, all on helium-air mixtures. The audiometer was permanently wired into the hyperbaric chamber, and the earphone was one carefully calibrated by Thomas, Preslar and Farmer (1972) down to 1000 ft (31 ATA) in helium-air. Sensorineural loss and also TTS were ruled out by noting that BC never changed under any hyperbaric condition. The shallowest depth at which audiograms were collected was 100 ft, by which depth significant conductive losses (ca. 15 dB) appeared at 3 and 4 kHz, so that the authors conclude that a conductive loss may develop at even shallower depths, perhaps at only 1 ATA. Fig. 9 (their Fig. 3) shows the development of mean losses taken at various depths from the surface to 1000 ft and back again. A trend exists at .5, 1, 3, and 4 kHz for mean hearing level to decline, and then return, depending on depth. No trend existed for hearing to improve or to worsen during 200 hrs at 600 ft.

The mean losses at 1000 ft are somewhat less than the 30-40 dB losses reported by Adolfsen and Fluor (1965). The improvement in hearing level at 6 kHz and the lack of change at 2 kHz certainly mean that the losses found have a rather complicated explanation in terms of more than one effect, involving impedance changes plus shifts in resonant frequencies.

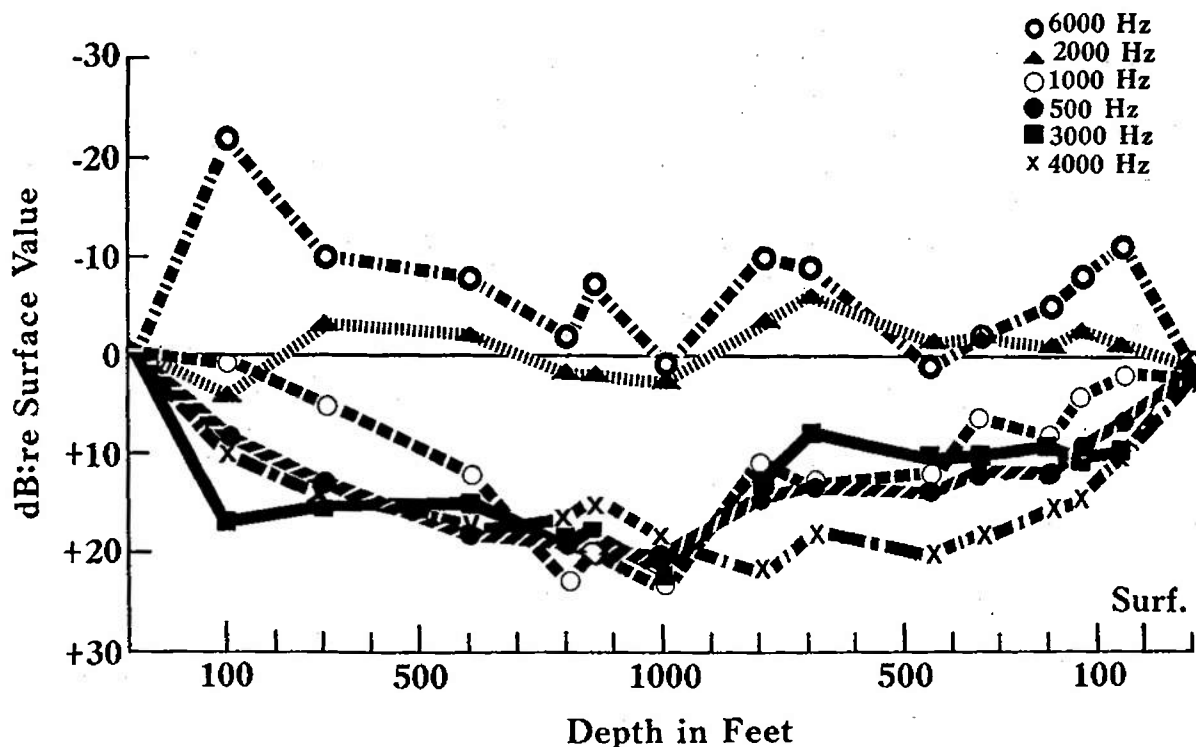


Fig. 9. Mean thresholds for six different frequencies as a function of depth. All thresholds are measured relative to surface threshold. (From Thomas, Summit, and Farmer, 1972.)

In all these exposures the conductive hearing losses were moderate, and of little consequence. However, this is unfortunately not always the case, as we shall see below.

(2) Animal Experiments. Nourrit (1970) submitted 16 guinea pigs to compression-decompression cycles typical of those his hypacusic divers had received. The organ of Corti and Reissner's membranes were intact, with no hemorrhage in scala media, not even in stria vascularis. No success was achieved in a search for intravascular air bubbles. However, lesions of a hemorrhagic nature were seen after rapid ("brutale") decompression, usually in the basilar membrane itself, and in the peripheral attachment of the

round window. Hemorrhage was also seen in the middle ear, for example at the tensor tympani and its canal. Most striking were changes in the tectorial membrane: it appeared more or less dislocated throughout its length, its fibrillar structure had disappeared and it appeared in part made up of a hyaline substance, which most likely, it was stated, presaged the disintegration of the membrane itself.

McCormick, Higgins, Clayton and Brauer (1971) subjected guinea pigs to compression-decompression in HeO<sub>2</sub> down to depths below simulated 2500 ft. In some animals, prior myringotomy prevented eardrum barotrauma. Cochlear microphonics were assessed pre- and post-dive. All animals showed

hearing loss, much of it due to eardrum barotrauma, but some with myringotomy may have had inner ear barotrauma. One animal definitely had a severe neurosensory hypacusis. McCormick, Higgins, Daugherty and Johnson (1971) also subjected guinea pigs with myringotomies to simulated 300 ft, using compressed air. Cochlear microphonics exhibited a general finding of loss, presumably neurosensory, beginning at 10-15 min postdive, increasing to "severe" loss at 1 hr, stabilizing at 2-5 hr, and again becoming more severe at 6 hr postdive.

McCormick (1973) exposed 24 guinea pigs to 80-90 ATA. There was no barotrauma, but vestibular systems did appear. Sixteen days later, one animal was completely deaf by CM test; there had been hemorrhage in the inner ear on that side. There was also profound deafness in another animal. He then induced decompression sickness in a group of guinea pigs; progressive hearing damage was noted in these animals. He also thought that administration of heparin protected these animals from a hearing loss.

### C. Effects of Altered Pressures in the Middle Ear.

(1) The Eustachian Tube: Structure. The characteristics of the Eustachian tube are of especial importance in sub-aquatic and hyperbaric medicine. All standard texts describe its structure and function (see, e.g., Graves and Edwards, 1944, and Simkins, 1943). Its bony portion arises near the bottom of the middle ear cleft, proceeds medially at about a 45° angle from the midline plane and downward at about a

30° angle from the horizontal plane, narrowing to an isthmus (2-3 mm high, 1-1.5 mm wide) after 12 mm and blending into a cartilaginous portion which bends downward somewhat more (about a 40° angle), enlarging its lumen and opening on the nasopharyngeal wall right behind the nose. The opening, the pharyngeal ostium, is on the lateral wall, and to the rear there is a firm prominence, the torus tubarius. Behind the ostium is a deep recess, the fossa of Rosenmuller. From the bottom of the torus the salpingopharyngeus muscle (special visceral efferent innervation from the Xth nerve) proceeds downward. A second muscle, the dilator tubae, arises from the lateral wall of the cartilaginous portion of the tube, and blends below with the tensor veli palatini (innervated by the V nerve). It is usually stated that action of the salpingopharyngeus and (largely) the tensor veli palatini tends to open the lumen of the tube, but the action of the levator veli palatini, which originates partly from the medial wall of the cartilaginous portion of tube, also has an effect especially on the orifice, however, the levator does not have the same innervation as the tensor (it has innervation similar to the salpingopharyngeus).

### (2) The Eustachian Tube: Function.

a. Normal Conditions. Normally the tube is closed, and is often said to operate as a "flutter-valve." Since the O<sub>2</sub> in the middle ear is always being absorbed by the mucosa, there is usually a slight negative pressure of -20 mm Hg in the tympanum with respect to the nasopharynx (Pohlman and Kranz, 1923; Perlman, 1943). Riu et al. (1969) state that the absorption of O<sub>2</sub> can lead to

negative pressure of the order of 1 cm H<sub>2</sub>O in 10 min. They found that in the usual waking state the Eustachian tube opens about once per min, about once per 5 min in sleep (see also Elnor et al., 1971).

Swallowing, yawning, or phonation are usually said to open the tube, but Riu et al. (1969) measured by sonomanometry the pressures in the nasopharynx, and simultaneously took electromyograms on the pharyngeal muscles, with the surprising finding that phonation did not open the Eustachian tube, only swallowing or yawning. See also Muenker, et al. (1972) for measurement of Eustachian tube activity by manometry in the external ear canal.

b. Artificially-Created Positive Pressure in the Tympanum. When the relative pressure increases in the middle ear, by way of decreasing the ambient pressure (as by ascent either in air or under water), the trapped gas is forced out the tube when the overpressure reaches 15 mm Hg (Armstrong and Heim, 1937), or ca. 20 mm Hg (Flisberg et al. 1963), or 4-10 mm Hg (Coles, 1964), or 20-25  $\mu$ bars (Riu et al. 1969). In the anesthetized monkey (Chang, et al. 1950) this occurs at 52 mm Hg  $\pm$  24, but when the tube is thus forced open, the pressure does not drop to 0, but remains at ca. 20 mm Hg, which is the same figure noted for the human by Pohlman and Kranz (1923) and Perlman (1943), and probably represents the resistance of surface tension offered by the tube. After an initial opening, as decompression continues, the tube requires somewhat less overpressure to open again. Much depends on the speed of decompression.

Williams and Cohen (1972) give some data on threshold for sensing pressure change, on 4 men subjected to "sudden" (time characteristics unspecified) pressure change. Threshold pressure change was .040 psi (an altitude change of ca. 75 ft in air). This may represent eventually the sensitivity of the eardrum to passive motion, but the pressures in the ear canals were unknown, more especially the volume velocity of the eardrum to these pressures.

c. Artificially-Created Negative Pressure in the Tympanum. Since the Eustachian tube is normally closed, then unless it is opened by muscular action it cannot be opened by overpressure alone in the nasopharynx until the overpressure reaches about 33 mm Hg (Chunn, 1960), being quite variable across subjects. However, at -80 to -90 mm Hg (Armstrong and Helm, 1937; Keller (1958) says -90 mm Hg) the flutter valve becomes "locked" so tightly that the subject's muscular action is now insufficient to "clear" the ear. Flisberg et al. (1963) put these values at -30 to -50 mm Hg, and Riu et al. (1969) put the values at -60 to -80 bars. For trained divers, Coles (1964) gives the figure of -100 mm Hg.

(3) Pressures at Pain and Rupture of Eardrum. When negative pressures are building in the tympanum without relief by the tube, the eardrum is progressively drawn inward. Severe pain is experienced at -60 mm Hg (Armstrong and Heim, 1937; Keller, 1958). Shilling and Everly (1942) found that 152 of 2,751 subjected to a hyperbaric

chamber run had to be released because of pain, usually before the differential pressure reached 10 lbs.

As the pressure differential increases, the eardrum will rupture, at -100 to -500 mm Hg (Armstrong and Heim, 1937), -200 to -400 mm Hg (Perlman, 1943), 27-30 lbs/sq inch (Vail, 1929) and -25 lbs/sq inch (Keller, 1958). In cat, Wever et al. (1948) found rupture at -89 to -140 mm Hg.

(4) Effect on Audiogram of Mild Differential Pressures Across the Eardrum. By means of a needle inserted through the Eustachian tube into the middle ears of cats, and recording cochlear potentials, Thompson et al. (1934) showed that not until  $\pm 5$  mm Hg differential pressure across the eardrum was reached was there any hearing loss, but that conductive losses then increased progressively to the pressure limit used of  $\pm 30$  mm Hg.

When Wever, Lawrence and Smith (1948) introduced differential pressures of  $\pm 50$  mm Hg across the eardrum in the cat, a loss (presumed conductive) of 20-40 dB was found, especially in the middle frequencies. At lesser pressures, the data for 2 kHz was exceptional, in that a gain was seen, not a loss. This finding foreshadowed the audiograms under pressure of Thomas, Summit and Farmer (1972) in which 2 kHz was also anomalous in not showing changes with pressure.

Close and Ireland (1961) showed that at sea level a differential pressure of +10 inches H<sub>2</sub>O across the eardrum created "conductive" losses of about 15

dB at frequencies up to 1-2 kHz, and at all frequencies with negative pressure of -10 inches; and about the same when the absolute pressure was changed to simulated 30,000 ft. No one has extended this experiment to hyperbaric conditions.

#### D. Effects in the Inner Ear.

From studies cited on the reversible audiometric losses either from differential pressure across the eardrum, or the onset of severe aerotitis media, it would appear that the conductive hearing losses found so commonly in hyperbaric conditions are moderate, almost always reversible, and of no medical consequence. Obviously, the audiogram will change if the impedance into which the eardrum looks has increased with pressure even with an equilibrated ear, and certainly if a gas other than air is being breathed, cavity resonances (which contribute to the audiogram) will change frequency. However, the distinct possibility of inner ear damage also exists. A general review was given by Edmonds and Thomas (1972).

(1) Intracochlear Vascular Accidents (Microhemorrhage, Vascular Spasm, Emboli, Bubbles). Coles and Knight (1960) carefully studied 62 very experienced Navy divers. Many of these had sensorineural hypacusis, but in every such case history the sensorineural losses could have been the result of previous acoustic trauma. On searching the literature at that time, furthermore, they uncovered no case of sensorineural loss in divers which was clearly supported by pre- and post-exposure audiograms as being due to barotrauma. More recently, Coles.

(1973) agrees that this conclusion must now be supplemented by cases of sensorineural loss which must be attributed to barotrauma; he has suggested even that some losses may be due to CNS damage in auditory tracts above the level of the cochlea nuclei, which may mimic peripheral (conductive) loss. There is no general agreement on that point, but there is now universal acceptance of not-rare cochlear hearing loss under hyperbaric conditions.

Vail (1929) was one of the first to report deafness resulting from nitrogen bubbles in the internal ear.

Nourrit (1961) distinguished a unilateral or bilateral perceptive deafness which may arise upon the patient's emergence from a compression-decompression cycle, or it may appear some hours or even days later; it may or may not be accompanied by vestibular symptoms. The author's clinic treated 10 subjects with acute hypacusis, and 15 with continuing hypacusis.

Of the 10 sudden-loss cases, 8 were perceptive (4 bilateral), 2 were mixed conductive-perceptive. Of the 15 hypacusics of longer standing, 3 had had prior accidents during decompression; 10 were perceptive; 5 mixed.

MacFie (1964) gave three cases of sensorineural loss in which, while pre-exposure audiograms were not obtained, there was nevertheless a good presumption of normal hearing prior to the diving incident.

Harris (1971) reported 10 cases seen by Dr. Summitt in the U.S. Navy, and reviewed the broader problem of

sudden deafness as it applied to diving medicine.

Plante-Longchamp, Maestracci, and Nicolai-Harter (1970) report never having seen an isolated cochlear barotrauma, such symptoms always being accompanied by (usually severe) vestibular symptoms also. They feel that a compressional lesion of the vestibule would lead simultaneously to a cochlear loss, given the continuity of the labyrinthine fluids. They predict that one will meet rather frequently with deafness or vertigo of which the vascular mechanism is proved by the efficacy of treatment by vasodilators and antispasmodics, and they question whether on ascent a formation of bubbles in the labyrinthine liquids does not aggravate the effects of the arterial spasm.

Stucker and Echols (1971) give a brief discussion of the possibility during decompression of bubbles of gas released as emboli to occlude the terminal twigs of the internal auditory artery, and recommend rapid recompression to avoid permanent damage from progressive necrosis of cochlear structures.

Farmer (1973) reported 12 more such cases of sensorineural loss, 3 of them with vertigo, during decompression. In no case were there any other classical symptoms of decompression sickness. Decompression, however, was certainly the major causative agent, as proved by the fact that recompression was an efficacious treatment.

Plante-Longchamp and Blanchi (1972) saw in 1970 two cases of isolated cochlear/vestibular lesions following dives, and upon searching the literature



were surprised to find such episodes were not unknown. Two more cases were seen, one showing a hearing loss: a man aged 34 after a dive to 20m, lasting 30 min. This led to vertigo and nausea, but treatment (by vasodilators and antivertiginous drugs) was begun only after three days. There remained a hearing loss confined to the high frequencies. It was suggested that the special nature of the arteries in the labyrinth (see Lawrence, 1966) might make it possible for a spasm to isolate the labyrinth fluids, so that bubbles could form even during a dive of short duration. Another young man after a short dive to 25m had vertigo and vomiting, and when this persisted for 10 hrs, he was seen by one of the authors who noted nystagmus and prescribed recompression. All subjective symptoms disappeared at 30m. The authors felt both cases raised the possibility of an intratissular bubble, either in endo- or perilymph. It appeared unjustified to evoke barotrauma or any direct mechanical lesion in these cases. It was also said possible that gas formations in the inner ear might have a more or less harmful effect mechanically, on the various membranes, so that even recompression might not be completely successful.

Gehring and Buhlmann (1972) reported four cases of hearing loss in men exposed repeatedly to HeO<sub>2</sub> at simulated depths to 690 ft. Three recovered immediately upon recompression, leading the authors to diagnose true decompression sickness. One diver was permanently hypacusic.

Farmer (1972) summarized the literature involving 20 cases of vestibular

and/or auditory problems during sub-saturation dives. Some had had some difficulty with auto-inflation. There were 6 cases of pure hearing loss (no vestibular symptoms), occurring during deep Helium dives. Two of these improved with recompression therapy; the remaining 4 did not have such treatment, and all developed a sensori-neural loss. One case developed a persistent unilateral loss at 56' from a dive to 400'. Six cases had combined vestibular-auditory symptoms, with sensorineural loss ascertained by audiometry (3 cases) or by tuning fork (3 cases). All 6 showed rapid and good recovery upon recompression. Farmer felt that the relief by recompression strongly supports an etiology of labyrinthine symptoms from bubbles. See also Demard, et al. (1969).

(2) Loss of Perilymph (Round Window Rupture). Pullen (1972) repaired a ruptured round window in a man who noticed loss of hearing three days following a dive. Seven weeks later hearing was normal, presumably when the normal volume of perilymph was regained.

Freeman and Edmonds (1972) presented in some detail five cases of quite reduced sensorineural loss generally not recovered, one of them bilateral, three of them of high tones only, all occurring during immersion to depth, and in such circumstances that a diagnosis of decompression sickness was stated not to have been possible. Two of the five had symptoms of vertigo. In each case there was impaired ability to "clear" the ear on the affected side, and Freeman and Edmonds concluded the pressure

differential between the middle and the inner ear was considerable, and the stress to which the stapes was put was relatively violent as these divers, all in some pain, were striving to "clear" their ears. They felt that there was no reason to postulate intracochlear hemorrhage or vascular spasm, etc., and recommended that upon any aural pain during ascent the diver should rise to a depth at which he could easily equilibrate his tympanum, or at least pause, before again descending.

Edmonds (1973) presented 5 cases of inner ear damage from barotrauma. Both audiograms and nystagmograms were collected. He showed that the effects of middle ear barotrauma of descent may have either temporary or permanent damaging effects on the cochlear and/or the vestibular apparatus. Three of the 5 cases were shown to have surgically correctable lesions by repairing round window perforations. The histories of the dives were consistent: there was initial difficulty in equalizing pressures within the middle ear cleft during the ascent, and usually there was an excessive attempt at forceful Valsalva maneuvers. Following the dive, there was usually a history of tinnitus, with or without vestibular and cochlear damage. In the cases of round window fistuli, the cochlear and vestibular damage progressed following the dive which was corrected only after the surgical repair was performed.

Edmonds et al. (1973) presented three cases of rupture of the round window attributed to barotrauma. On exploration of the middle ear, a fistula was observed in each case, with perilymph passing through the rupture

into the middle ear cleft. Fat tissue grafts were applied, with symptomatic improvement paralleling the improved otological functioning, as assessed by audiometric and ENG recordings.

If there is a presumption that round window rupture has occurred, this must be ascertained by exploratory middle-ear surgery and reconstruction begun at the earliest date if permanent hearing loss is to be avoided.

(3) Gas Saturation of Cochlear Fluids. There is still another possibility for inner ear damage: In a helium dive, the helium in the middle ear cleft can readily diffuse across the round window and the inner ear fluids can reach saturation before those tissues receiving gas normally through the circulation. (In the cat, the cochlear microphonic voltage remains for as long as two hours, fed by oxygen through the round window; but this "death potential" disappears immediately if the middle ear is flooded with helium.) Thus the usual gas dynamics need not apply to the labyrinth.

(4) Occluded Eustachian Tube. In certain patients with chronic occluded Eustachian tube, high-tone hearing loss presumably sensorineural has been noted. Mygind (1931) suggested the organ of Corti might become edematous, and Galloway (1940) that toxic substances collected in the undrained middle ear might enter the inner ear by way of the round window. However, Lock (1942) closed off his own tubes with a balloon inserted through the nose and inflated. After 75 min, hearing loss at 2-10 kHz had

progressed irregularly to 15-20 dB, all returning to normal with deflation of the balloon. Evidently tubal obstruction first affects high tone conduction. Since it is most unlikely that the inner ear is changed in any way, a mild high-tone loss cannot always be presumed non-conductive in nature.

(5) CNS Involvement. Fructus and Ricci (1970) state that a too-forceful Valsalva maneuver can lead to a "brutale" variation of the alveoli capillary gradient with stress at the level of the smaller vessels leading to the formation of pathogenic bubbles at the level of the alveoli, these bubbles passing in the left ventricle by the pulmonary veins, possibly leading to cerebral embolisms of very wide distribution.

Carlotti and Plante-Longchamp (1972) present the case of an experienced diver who after one 18-min dive to 40m suffered a variety of symptoms on leaving the water. At the beginning of his ascent, the diver had repeatedly executed the Valsalva, and finally rose slowly with numerous Valsalvas. He was recompressed with total recovery, and dived again within a week. Carlotti himself had been with the patient throughout the dive and could state no particular etiologic factor. The authors concluded it was a clearcut case of central neurologic accident, and agreed with Fructus and Ricci (1970) as to its probable origin. It was evidently such cases as these that Coles (1973) had in mind in referring to CNS involvement.

Other cases of inner ear dysfunction as a result of barotrauma are cited by Picard et al. 1959; Picard and Nourrit, 1961; Bouche et al. 1961; Pagano, 1961;

Appaix, Picard, and Nourrit, 1961; Appalix, Grinda et al. 1961; and Grinda and Canavese, 1962).

## VIII. GENERAL MEDICAL EFFECTS

### A. Incidence.

(1) Among Swimmers Generally. Hitschler (1949) in a survey of 900 college graduates found no greater incidence of reported hearing loss among the half of his sample who were trained swimmers than among his controls. There seems nothing about fairly frequent exposure to water as such (especially, clean sea water) to lead to auditory problems in those who are trained in the necessary hygiene.

In some persons who have swum for years, there may develop exostoses in the external auditory meatus, and the suggestion was made many years ago that there may be associated middle ear changes resulting in some conductive hearing loss. However, in a series of 10 hypacusic swimmers of long standing with exostoses, Adams (1951) determined that their hearing loss was of a perceptive type, while in 3 controls without exostoses the hearing loss was also perceptive, and concluded that meatal osteomata are not necessarily accompanied by similar growths in the tympanic cavity. There were ample reasons given in the extensive case histories for the hypacusis (e.g., 4 of the 10 had deafness in the family).

(2) Among Amateur Skin- and SCUBA-Divers. Especially with the recent advent of the Self Contained Underwater Breathing Apparatus, the

otolaryngologic problems of U/W exposure have mushroomed. The paper of Hitschler (1949) has been mentioned. Taylor (1959) gave a general discussion from the point of view of the otolaryngologist advising those performing frequent skin and scuba diving. All the auditory problems he mentioned are conductive in nature rather than sensorineural.

Wilke and Stefel (1965) and Dubs (1967) discussed hearing among sport divers. A recent rather extensive review was given by Stucker and Echols, 1971 (see also Ottoboni et al. 1966; and Wilson, 1972).

(3) Among Caisson Workers. It has been long known that caisson disease often has an inner-ear/labyrinthine component. Smith (1873) coined the term "Caisson Disease" and mentioned hearing as the most prevalent problem. Alt et al. (1897) note auditory problems with pressure changes, and present histological material on such cases in both the human and the animal. Papers far too numerous to mention have reported labyrinthine (including auditory) dysfunction among caisson workers. Full reviews representative of those which summarize the status at successive dates are Smith (1873), Van Renssalaer (1891), Curnow (1894), Snell (1896), Alt et al. (1897), Lester and Gomez (1898), Heller et al. (1900), Phillip (1907), Berruyer (1908), Keays (1909), Hill (1912), Boot (1913), Vail (1929), Lestienne (1933), Chiappe (1939), Shilling (1941 a,b), Almour (1942), Behnke (1947), Catchpole and Gersh (1947), Kerekes (1948), Bertoin (1953), Pagano (1953), Coleman (1953),

Haymaker and Johnston (1955), Kooperstein and Schuman (1957), Bruzzi (1958), Nourrit (1959), Pagano (1959), Rozsahegyi and Gomori (1961), Maspotioli et al. (1965), McCallum (1967), Demard (1968), Behnke (1969), and Lang et al. (1971).

Poli (1909) states that of 205 caisson experienced workers exposed to 0.7-1.5 A, 14.6% had hearing difficulties, and of 186 less-experienced men, 7.5% had such problems. Thirty workers (22 inexperienced, 8 experienced) were considered unfit for caisson work, only 3% by reason of poor hearing, the rest for tinnitus and vertigo.

Pagano (1959) studied 40 cases of caisson disease, dividing them into losses of conduction (35%), of perception (50%), and mixed (15%). In a later series of 104 workers, 77 had perceptible losses. Of these, 52 had AC losses exceeding 50 dB and BC losses exceeding 30 dB at all frequencies. In 25 cases the perceptible loss was judged by oto-audiological tests to be retro-cochlear.

Lang et al. (1971) examined 432 young (under 40 yrs) caisson workers extensively with a battery of oto-audiological tests, with the rather alarming finding that 60% had hearing defects, far more than would have been predicted from the careful measurement of the noises to which these men had been exposed. Two-thirds of the men 20-29 yrs old suffered a hearing defect after only 6 mo in compressed air. The authors feel that decompression sickness potentiates the ear's response to noise.

(4) Among Inexperienced Hyperbaric Chamber Subjects. A wide range of incidence has been reported of aerotitis media among inexperienced persons. If pressurization is too rapid, all ears will undergo the symptoms of middle ear barotrauma. Teed (1944) studied men subjected for the first time to a 50-lb pressure test (13 ATA, simulated 100 ft depth) occupying from 3-10 min, and found 25-30 per cent had some degree of aerotitis media. He felt this could be reduced to 2-3 per cent if only those men able to perform the Valsalva maneuver were pressurized. Shilling et al. (1946) in the same pressure chamber found 26.9 per cent of 6,149 men contracted more or less severe symptoms. About 6-10 per cent of all men were unable by reason usually of severe pain to complete the pressure test. Again in the same chamber Alfandre (1965) found an incidence of 36.2 per cent of 432 men given the 50-lb test.

Bayliss (1968) reported the incidence of aural barotrauma in 530 diving trainees, all medically fit; 20.4 per cent of these men reported "ear trouble" either in the recompression chamber (58.5%) or in the water (41.5%) and were examined by the author himself. On the 1-5 MacFie scale, only 1 ear ruptured (Grade 5), none showed Grade 4 (free blood in middle ear), 2 ears had Grade 3 (gross hemorrhage confined to the eardrum but no blood in middle ear), while the remainder who complained of ear trouble showed Grades 0-2, about half of whom recovered completely in 3 days or less with oronasal decongestants and antibiotics. The relatively low incidence of serous barotrauma was attributed to rigorous physical selection

and appropriate management during the early stages of diving training. (For other systems used to classify the symptoms of aerotitis media see Teed, 1944, and Seeman, 1964).

On descent from airplane flights of course the pressure differential cannot exceed 1 ATA; yet this is enough to cause aerotitis media, but seldom in more than 10% of persons (see Wright, 1945). The problem of aerotitis media in real and simulated flight has been extensively treated (see, e.g., Armstrong and Heim, 1937; McGibbon, 1942; Dickson et al. 1943; Kos, 1944; Hyde, 1952).

Liebermann (1946) subjected 167 aviation candidates to simulated heights of 35,000 ft at room temperature, of 30,000 ft at -40°F, and a "free fall" from 30,000 to 18,000 ft in 50 sec, a total of 330 individual "flights;" 43 men reported episodes of aero-otitis media, a percentage of 16.6%. However, otoscopy revealed an additional 37 cases where the men had not complained, which yields a total of 27.9%. Only 3 men reported symptoms but were rated normal by otoscopy. In the worst case (free-fall), 50.3% showed aero-otitis media by otoscopy. Room temperature as such had no effect, neither did report of recent upper respiratory infection ("colds"), nor flying experience. Examination of 129 men with a nasopharyngoscope yielded 12 with obvious occlusion of the ostiums of the Eustachian tube, of whom 50% had aero-otitis media, whereas the incidence was only 13.7% in those with nonobstructed ostia. Evidently even in flight conditions, generally

less severe than in U/W or simulated U/W conditions, very substantial percentages of men are affected.

(5) Among Professional Divers. It is a commonplace that the conditions under which diving takes place are ideal for the creation and furtherance of otitis externa. In warm, especially tropical, environments there is a need for frequent bathing, and an increase in activity of the sudatory and sebaceous cells in the external auditory meatus. In this debris any infective organism, of which there are many, finds a hospitable medium. There is often local damage to the epithelium in attempting to remove water or cerumen from the meatus, in using ear plugs, etc., which may allow organisms an entry. There may be secondary infection following dermatitis or eczema, and even rupture of the eardrum in otitis media. Allergic eruptions may occur, and fungi are common in tropical waters.

None of this etiology is of course confined to diving or hyperbaric chamber operation, and every physician is familiar with the symptoms and the conservative treatment indicated, namely, sterile cleansing of the area and topical applications, including local and systemic antibiotics, plus specific measures to control any underlying problem (fungi, allergy, etc.).

Sperati and Perfumo (1967) studied otitis externa rather exhaustively in 50 divers. Of these, 23 showed the disorder on periodic inspections, as against only 3 of 50 controls. Of 34 ears, 16 were nonsuppurative, 9 exhibited sterile, and 9 purulent exudate. Cultures showed in 53% the presence of E. coli, but

there were frequent case of staph and strep strains. In controls, only 6% showed E. coli. However, the authors claim that even though the disorder in divers is more common than usual, it presents no other especial characteristics peculiar to that activity, and treatment is the same as for any case.

Symptoms of barotrauma are either often subclinical or else working divers are reluctant to report aural problems. Prazic and Salaj (1971) carefully administered auditory and caloric tests to experienced divers who reported no hearing or equilibrium disorders. 38% of these had hearing disorders or lack of thermal sensitivity in various degrees.

In those who dive repeatedly, a high incidence of auditory complications can be expected. The Ama, the diving women of Korea and Japan (Hong and Rahn, 1967), who inspire to 85% of their inhalation capacity and then descent to depths of about 100 ft as many as 60-90 times per day, experience otorhinolaryngological problems of all kinds as their chief occupational disorder (see Yamamoto, 1936, and Kataoka, 1954), with chronic otitis media, opaque eardrums, non-patent Eustachian tubes, sinusitis, rhinitis, and hearing loss.

In Yamamoto's material, permanent hearing losses at the speech frequencies (250-2000 c/s) occurred at an incidence from about 30% of cases in the youngest divers (16-20 yrs) to about 50% in the older (51-60 yrs). During heavy diving months, greater losses (usually recoverable) occurred. In Kataoka's material, 36 of 74 divers

(49%) had severe hearing losses as against 28% of non-divers from the same villages. Anzai (1960) compared 88 Ama vs 86 controls, and found that only auditory problems distinguished the groups medically, though only 20% of these particular Ama reported loss of hearing (2% in the controls).

Harashima and Iwasaki (1965) performed a complete medical study of 22 Ama aged 29-59, with 12-43 yrs of deep diving experience. Of these, 14 complained of tinnitus, 12 of unilateral eardrum rupture, and 6 of spontaneous otalgia. About half reported otitis media, and about half said they did not dive if they had a cold. The authors feel the chief problem of these divers is chronic otitis media leading to deformities and, often, rupture, of the eardrum.

Zannini et al. (1972) studied 160 professional divers for some time, and find also that there is cumulative insult to the middle ear apparatus, and further that much conductive loss presages sensorineural loss, mainly expressed as high-tone loss from lesions in the basal turn of the cochlea. Edmonds and Blackwood (1971) discuss the otological and audiological personnel and tests which should be available to a population of working divers.

#### B. Further Remarks on Aerotitis Media.

(1) Etiology and Treatment. Any number of acute and chronic infectious processes, nasopharyngeal stenosis, cleft palate, or malocclusion can lead to aerotitis media. When a serosanguinous fluid collects in the middle ear, the condition is termed serous otitis media. Occasionally the invagination of the

eardrum ruptures that membrane and the serous phase never develops. Only when fluids in the middle ear impede eardrum motion is the audiogram appreciably depressed from a single episode (Haines and Harris, 1946). All such episodes recover usually within a few days with conservative (i. e., no) treatment. The initial suffusing of the epithelium lining the whole middle ear cleft, including the eardrum, usually subsides with no treatment whatever, but secondary infection often follows, due to the presence of warm liquids (water, serosanguinous fluid) as an ideal culture. There may be tinnitus, some fever, earache, and a measure of conductive hypacusis. In treatment, decongestants and vasoconstrictors are given, and usually penicillin. Anything is done to drain the middle ear and render patent the Eustachian tube.

It is surely unnecessary at this date to reiterate the caution of Lampe (1950) against using radium application to the nasopharynx to render the Eustachian tube easier to open; but as late as 1958 (Duffner), 1958 this was still being advocated by ill-informed persons.

Lovelace and Boothby (1939) give rather more than anecdotal evidence on 16 cases of barotrauma in whom the symptoms of pain, etc., were alleviated or stopped by breathing 80%-20% HeO<sub>2</sub> mixtures during or after flight. It was suggested that the lighter gas was more effective because it passed more readily through the Eustachian tube (at sea level the mean velocity of the He molecule is 1.202 km/sec, while that of O<sub>2</sub> and N is of the order of .4). Hall (1940) however in a fuller test did not corroborate this suggestion.

Since the tympanic mucosa will absorb oxygen about three times faster than nitrogen, a person breathing pure oxygen for an appreciable time may suffer a chronic and seemingly delayed barotrauma (Behnke, 1944) from an unusually rapid reduction of pressure in the tympanum. However, Chang et al. (1950) believed that the patients observed by Behnke were rather suffering from a prolonged period of low differential pressure which can also lead to barotrauma.

Chang, Margaria and Gelfan (1950) were able to explain why in their monkeys with barotrauma the tympanic vessels often ruptured, perhaps at only -300 mm Hg, while the eardrum did not. They pointed out that the tympanic vascular system was subjected not only to the "cupping" effect of the low air pressure in the tympanum, but also to a high intravascular blood pressure, both forces tending to dilate the vessels.

Farmer (1972) gave a full description of aerosinusitis, pointing out that the etiology and symptoms are similar to those for aerotitis media, but of course involving different drainage tracks. (See also Bateman, 1945; Pagano, 1951; Bertand, 1945; Campbell, 1960; and Weissman, et al., 1972).

(2) Mechanisms Available to the Diver to Open the Eustachian Tube. For a basic discussion, see Ingelstedt et al. 1967.

a. Valsalva Maneuver. A forced expiration, mouth and nose closed. Riu et al. (1969) by electromyography showed

that the pharyngeal muscles which can open the tubes were not involved, and sonomanometry showed that gas could be forced up the tube by pulmonary and thereby nasopharyngeal pressures which are 130 cm H<sub>2</sub>O on the average.

(a) Special Caution on the Valsalva Maneuver. It seems clear that a strenuous effort to clear the ears should be avoided. Consider a diver at some depth, say 6 ft, at which the volume of air in the middle ear is reduced about 20%. The eardrum is here forced inward, the stapes likewise, and the round window is forced outward about as far as it will go. Now on a strenuous Valsalva maneuver, air is let in, and the round window is suddenly forced inward about as far as it will go. No doubt its elastic limits are occasionally exceeded. It is conceivable that the oval window and the annular ligament might similarly be affected.

Lee, Matthews and Sharpey-Shafer (1954) showed with indwelling sensors the more precise relations during a Valsalva maneuver among systemic and pulmonary arterial pressure and right atrial and pulmonary capillary pressures. Right atrial pressure and pulmonary capillary pressure rose as much as 20 mm Hg. The high pressures which in the nasopharynx can reach 190 cm Hg produce an obstruction to the return of venous blood which can lead to cephalic venous congestion and to cephalic and even general arterial hypotension. There could follow syncope (see Duvoisin et al. 1962) from pulmonary nociceptive reflexes, venous



cephalic congestion, or arterial hypertension.

Armstrong (1961) felt that prolonged Valsalvas should be prohibited in flight. Lamb et al. (1958) implicate the Valsalva in cardiovascular arrhythmias induced by pulmonary stretch reflexes in aircraft accidents.

The hazards of the Valsalva maneuver while weightlifting were discussed by Alvis (1971), with generalization to divers (see also Langer and Mansure, 1971).

b. Frenzel Maneuver. There is a non-traumatic way to ventilate the middle ear by way of positive pressure in the nasopharynx, now called the Frenzel maneuver (Frenzel, 1938) in honor of its propounder: voluntarily closing the glottis, closing the mouth and nose, while contracting the muscles of the floor of the mouth and the superior pharyngeal constrictors.

With the nose, mouth, and glottis closed, the elevated tongue can be used as a piston to compress the air behind it in the nasopharynx and thus up the Eustachian tube. In a well-executed Frenzel, the mass of the tongue is very strongly driven backward. Note that no remotely dangerous pulmonary pressures are generated, the intrathoracic pressure being practically unchanged. Moreover, probably since the tubal muscles are active, it takes less nasopharyngeal pressure (only 6 mm Hg instead of about 33) to force air up the tube than with the Valsalva; the nasopharyngeal pressures created by the Frenzel maneuver are higher than those possible with the Valsalva; and finally,

the Frenzel can be performed at any stage of inspiration - the Valsalva usually only after a sharp inspiration. Thus the Frenzel maneuver is superior on several important counts; unfortunately it is difficult to learn and harder to teach, though this is possible in most subjects (Chunn, 1960).

c. Activation of the Pharyngeal Muscles by Raising the Velum. Experienced divers can use isolated contractions of the velum (soft palate) without swallowing, or certain movements of the jaws. Riu et al. (1969) studied such movements by X-ray movies and found these maneuvers always had the effect of raising the velum; the muscles of the pharynx were thus shown to be active. These active muscular openings of the tube must therefore not be effective when the differential pressure in the middle ear exceeds -60 to -80  $\mu$ bars, and more forceful maneuvers are necessary to force gases into the tympanum.

Riu et al. (1969) found by survey that while beginners nearly always use the Valsalva, experienced divers rarely do, relying upon isolated contractions of the velum together with jaw movements. They suggest that these often unconscious movements point to a conditioned reflex.

d. Toynbee Maneuver. This consists of swallowing with the mouth and nose closed; it is the inverse of the Valsalva and is of some limited use in relieving hyperpressure, rather than hypopressure, in the tympanum.

## IX. SUMMARY AND CONCLUSIONS

Underwater (U/W) hearing is shown to be a special case of bone-conducted

hearing in which acoustic energy enters the cochlea at all points on the bony capsule. Since the cochlea is quite indifferent as to where acoustic energy enters, U/W hearing is about as good as in air for the analysis of complex sounds such as speech. For the frequencies 15 kHz - 100 kHz, U/W hearing sensitivity is markedly superior to that in air since in the usual air-conduction route the middle ear acts as a high-frequency-reject filter.

In terms of sound pressure level, hearing by air conduction exceeds U/W hearing by 30-60 dB, somewhat greater at the higher frequencies. However, the displacement of the skull to threshold underwater acoustic energy, about  $10^{-10}$  cm, is also the order of magnitude of the movement of the ossicles at threshold by air conduction. From the latter point of view it is not correct to think of hearing U/W as less efficient than in air.

Well-controlled studies from two laboratories concur that the threshold sensitivity of the submerged ear is about 70 dB re  $.0002 \mu\text{bar} \pm 5$  dB from .125 kHz through 4 kHz, falling off about 5-10 dB at 8 kHz. This was true whether or not the external auditory canal contained a bubble of air and did not vary appreciably between depths of 35-105 ft.

Recent studies, in contradistinction to earlier studies, have shown a modicum of localization ability in the submerged human for sound sources. Some divers have yielded minimum audible angles in the horizontal plane of 10 and even 5°. Analysis of U/W acoustic energy coming from the whole of one side of a submerged head to the

near cochlea vs the far cochlea, leads to intercochlear differential arrival times (about 5  $\mu\text{sec}$ ) and intensities (about 0.5 dB) known to be on the verge of affecting localization of sounds in air. Thus a theory for U/W localication can perhaps be worked out not greatly different from that for air. The precise effects of acoustic frequency, of water depth, of distance to the source, of verticality, of learning, and of many other features of the task are still to be delineated.

When ambient pressure is changed, as in airplane flight or in hyperbaric chambers, a reversible change in air-conduction hearing occurs even when the pressures across the eardrum are equalized by opening the Eustachian tube. These conductive (i. e., not sensorineural) losses may amount to 30-40 dB at the speech frequencies, though some report only about 20 dB even at simulated water depth of 1000 ft. However, in guinea pigs with eardrums lanced so that otitis media would not occur, inner ear barotrauma occurred in some animals even at only simulated 300 ft depth.

In the human, the normally-closed "flutter valve" of the Eustachian tube opens only occasionally, upon swallowing or yawning (not phonation), and  $\text{O}_2$  is absorbed in the middle ear continuously, so that a negative pressure of about -20 cm Hg is very often the case. If ambient pressure is increased, a sensation of fullness in the ear also increases, with severe pains at about -60 mm Hg, and rupture of the eardrum at -100 to -500 mm Hg. Unless the muscles which open the tube are activated, it takes about 33 mm Hg overpressure in the

nasopharynx to force an opening. When the overpressure reaches -80 to -90 mm Hg, the flutter-valve is "locked" and even experienced divers cannot "clear" their ears by muscular action alone, and must use auxiliary maneuvers (Valsalva, Frenzel) to increase nasopharyngeal pressure. For reasons of efficiency and of medical safety the Frenzel is superior to the Valsalva maneuver and should probably supplant it.

In all persons repeatedly exposed to subaquatic and dysbaric environments, otological problems are rife and among the most common complaints. Otitis externa, infections, fungus, etc., from water immersion are the rule. Otitis media in some degree is universal from barometric changes too sudden to allow compensation or too slow to be immediately sensed; in those inexperienced or unable to ventilate the middle ear; or even in highly experienced persons incautious enough to expose themselves when they have even a slight upper respiratory disorder. There are now cumulating scores of cases of inner ear disorders some temporary and mild but others up to and including sudden and permanent deafness, traced directly to dysbarism. A distinction can be made between cochlear barotrauma of descent, when ambient pressures are increasing, and of ascent, when they are decreasing. In cochlear barotrauma of descent there may be vascular accidents, treatable with vasodilators or anti-spasmodics, and even rupture of the round window sometimes attributable to a too-forceful Valsalva maneuver (against which several cautions are raised). The round window rupture should be visualized and repaired at once by an

otologist. In cochlear barotrauma of ascent there may be intratissular bubbles leading to necrosis of the organ of Corti and/or the tectorial membrane, causing severe and, in the absence of treatment, often permanent hypacusis, with perhaps no other symptoms of decompression sickness. In these cases, immediate recompression is often effective in restoring normalcy. Obviously, widespread gas emboli from too-rapid decompression can also attack any auditory center in the CNS.

Most otorhinolaryngological problems are the same among any professional group exposed to dysbaric conditions. Among aviators the severity of symptoms is usually less, due to the limited pressure differentials. But among caisson workers, for whom the literature is rather full for the past century, and among bareheaded divers such as sponge and shellfish gatherers, SCUBA divers, deep saturation divers, and hyperbaric chamber users generally, among whom another population explosion is now only in the beginning, the incidence of auditory problems both of structure and function is scheduled only to increase with increasing depths and relaxed personnel selection. All such persons should be very familiar with the auditory physiology of dysbarism, techniques to avoid medical incidents, and in a general way with treatments available. The present report is one tool in this direction.

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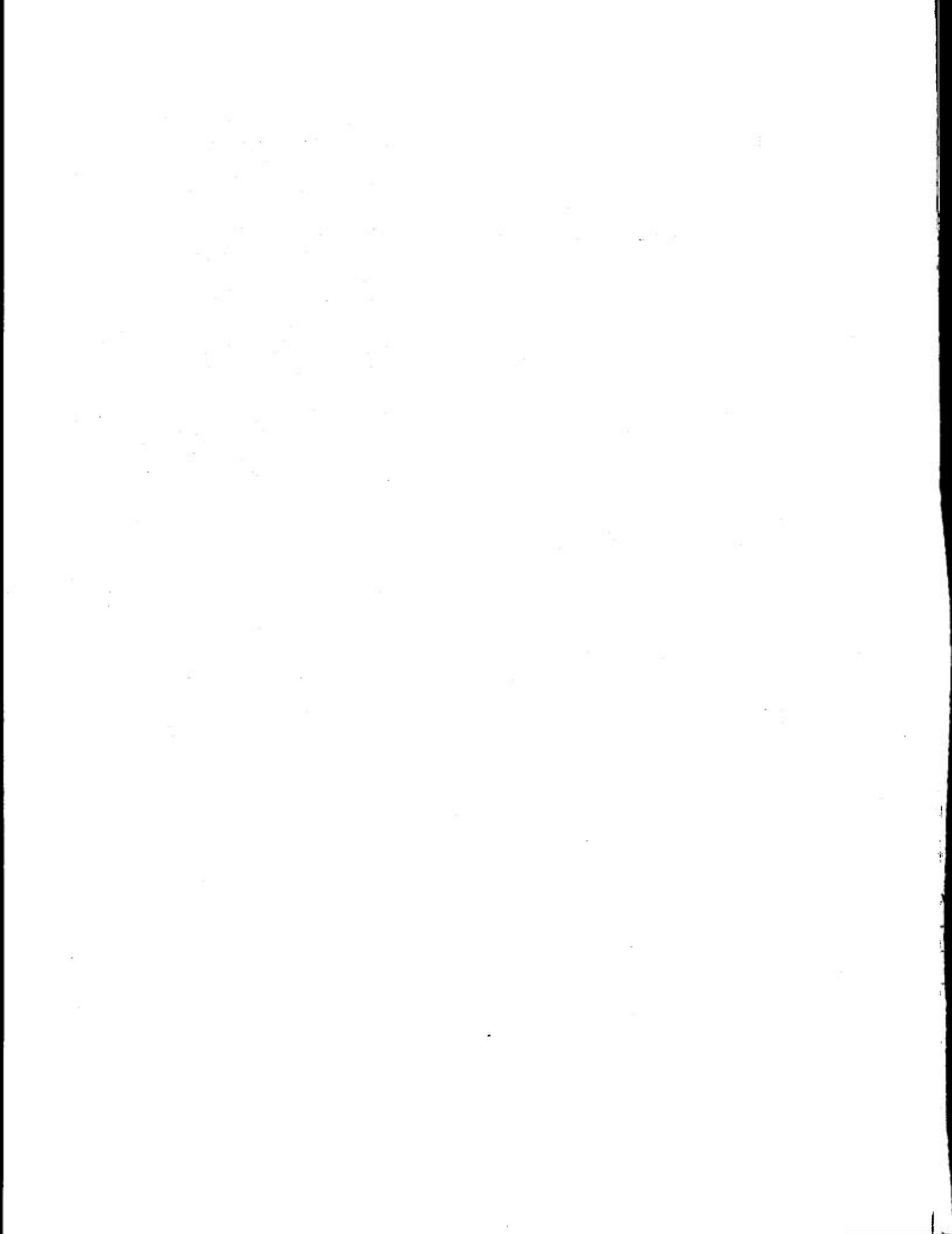
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Security Classification

DOCUMENT CONTROL DATA - R & D		
<i>(Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)</i>		
1. ORIGINATING ACTIVITY (Corporate author)  NAVAL SUBMARINE MEDICAL CENTER Naval Submarine Medical Research Laboratory		2a. REPORT SECURITY CLASSIFICATION <b>UNCLASSIFIED</b>
		2b. GROUP
3. REPORT TITLE  THE EAR AND HEARING IN AQUATIC AND DYSBARIC ENVIRONMENTS		
4. DESCRIPTIVE NOTES (Type of report and inclusive dates) Interim report		
5. AUTHOR(S) (First name, middle initial, last name)  J. DONALD HARRIS, Ph.D.		
6. REPORT DATE 29 May 1973	7a. TOTAL NO. OF PAGES 47	7b. NO. OF REFS 250
8a. CONTRACT OR GRANT NO.	9a. ORIGINATOR'S REPORT NUMBER(S) NSMRL Report Number 746	
b. PROJECT NO. M4306.03-2090DXC9.01		
c.	9b. OTHER REPORT NO(S) (Any other numbers that may be assigned this report)	
d.		
10. DISTRIBUTION STATEMENT  Approved for public release; distribution unlimited.		
11. SUPPLEMENTARY NOTES		12. SPONSORING MILITARY ACTIVITY Naval Submarine Medical Center Box 600 Naval Submarine Base New London Groton, Connecticut 06340
13. ABSTRACT A non-quantitative theory of human hearing is sketched for the case of the head immersed in water, based upon what is known of hearing in air and hearing by bone conduction. A consideration is given of four routes by which acoustic waterborne energy is transmitted to the fluids of the inner ear. Quantitative data are presented from recent studies of the thresholds of underwater auditory acuity, and of the relations between airborne and waterborne thresholds. Sound levels possibly hazardous to free swimmers are suggested and the intelligibility of speech passed through water is discussed. Studies on the ability of the human to localize sound under water are discussed. The acute otorhinological problems of the outer, middle, and inner ear are discussed in both aquatic and dysbaric environments. A bibliography of 250 items is presented which constitutes a valuable reference tool.		

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S/N 0102-014-6600

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14. KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Underwater hearing						
Underwater speech						
Waterborne acoustic hazard						
Underwater acoustic orientation						

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