U. S. NAVAL SUBMARINE MEDICAL CENTER

Submarine Base, Groton, Conn.

REPORT NO. 450

AEROTITIS MEDIA IN SUBMARINE RECRUITS

by

Howard J. Alfandre
Lieutenant, MC, U. S. Navy

Bureau of Medicine and Surgery, Navy Dept.
Research Work Unit MR005.14-3100-1.05

Released by:
C. L. Waite, CAPT MC USN
COMMANDING OFFICER
U. S. Naval Submarine Medical Center

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SUMMARY PAGE

THE PROBLEM

To evaluate the predisposing factors, the results, and possible sequelae of aerotitis media as encountered among submarine recruits during their physical qualification for submarine training.

FINDINGS

Four hundred and thirty-two Naval personnel were studied as they underwent pressurization in a dry-cylinder tank. Among these men, 156, or 36.2 per cent, developed aerotitis following pressurization. Upper-respiratory infection was shown to predispose a man to development of aerotitis; also predisposing were the inability to insufflate the middle ear, and the presence of nasal allergies. The presence of adenoidal tissue and/or dental malocclusion did not appear directly related.

APPLICATIONS

Results of this study should be useful in improving the method of handling pressurized groups of candidates, and in reducing the incidence of aerotitis media in these candidates for the submarine service.

ADMINISTRATIVE INFORMATION

This investigation was conducted as a part of Bureau of Medicine and Surgery Research Subtask MR005.14-3100 (Physiology), under Work Unit No. 1 - Physiological Alteration in Free Diving. The present report has been designated as Report No. 5 on this Work Unit, and was approved for publication as a Submarine Medical Center Report on 29 May 1965. It had previously (March '65) been submitted by the author as partial fulfillment of requirements for qualification in submarine medicine.

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AEROTITIS MEDIA IN SUBMARINE RECRUITS

INTRODUCTION

The recent interest in the treatment of disease states, in which hyperbaric oxygenation may be a modality, has introduced additional hazards for the patient and medical staff. Utilization of this form of treatment requires that all individuals involved be subjected to increased ambient pressures up to three atmospheres absolute (66 ft.). Breathing 100% oxygen, at pressures greater than this markedly increases the incidence of oxygen toxicity.[1] During "pressurization", the pressure within gas-containing bone-surrounded cavities must be equalized with the ambient pressure. Failure to equalize gas pressure within these structures may result in pain referred from the involved area. The structures most commonly involved are the middle ear, the paranasal sinuses, and the teeth. Involvement of the middle ear is referred to as aerotitis media or "ear squeeze" among diving personnel. Other terms used are dysbarism (A. F.), aerosalpingotympanitis (Teed), otitic-barotrauma, oticbarotrauma, aerotitis media, and aviator's ear. The term "aerotitis media" is used in this report because it is the most descriptive and precise. This term at once defines etiology, appearance and location. The inability to insufflate the middle ear through the Eustachian tube results in the development of aerotitis media. It is necessary to perform certain maneuvers to open the nasopharyngeal orifice of the Eustachian tube to insufflate the middle ear. Among these maneuvers are the Valsalva maneuver, yawning, movement of the mandible from side to side, and swallowing. The objective findings of aerotitis media can be visualized by otoscopic examination as involvement of the tympanic membrane and middle ear to various degrees.

Teed, Shilling, and Schulte reported a 26.9%—30% incidence of aerotitis media in prospective submarine personnel.[2, 3, 4] It is important to emphasize that these prospective submariners met U. S. Naval physical standards by history and physical examination prior to the "pressure test". Acute upper-respiratory infection, nasal allergy, malocclusion, hypertrophic adenoidal tissue, and the inability to perform the Valsalva maneuver have been reported as predisposing factors to the development of aerotitis media.

In addition, since a relatively large proportion of those exposed to increased ambient pressure develop aerotitis media, it is necessary to determine its sequelae. Prospective submarine personnel provide an excellent source of material for study. As part of the physical requirements for submarine training, these individuals must successfully adjust to a pressure of 50 p.s.i. in a dry recompression chamber. Failure to do so results in elimination of the individual from submarine training.

The purpose of this study is to evaluate the predisposing factors, the results, and sequelae of aerotitis media. Among the factors studied were the effects of increased ambient noise levels and the effects of aerotitis media on the hearing acuity of individuals.

METHODS AND PROCEDURES

Prospective submarine personnel received complete physical examinations, phychological tests, chest x-rays, dental examinations and audiograms prior to the 50 p.s.i. "pressure test". Only those personnel who possessed physical requirements for the U. S. Naval Submarine Service were scheduled for the pressure test.[5] Four hundred thirty-two consecutive individuals undergoing pressure tests were studied. The study was carried out during the months of April and May 1964. Upon reporting to the chamber and prior to undergoing the pressure test, each man was examined otoscopically to assure that there were no findings that could mimic aerotitis media clinically. The men were assigned to groups. A maximum of eighteen was assigned to a group. Each group reported to a classroom and received a lecture on the effects of pressure and the correct method of performing the Valsalva maneuver in order to insufflate the middle ear. After this was accomplished, each man

completed Part I of the questionnaire (Appendix 1). Upon completion of Part I, the group reported to the recompression chamber (Fig. 1)* and occupied the middle lock of the chamber. The middle lock was isolated from the outer lock by a pressure-resistant sealed door. A qualified Navy diver-instructor accompanied the group and controlled the pressurization. If an individual was unable to equalize the middle ear or paranasal sinuses, as evidenced by pain or discomfort in the location of the affected structure, the pressurization was stopped. Frequently, when a subject is unable to insufflate the middle ear it is necessary to depressurize the chamber 1-2 psi. In many instances the individual is then able to equalize pressure. Pressurization is begun again and the subject is instructed to "stay ahead of the pressure" and pressurization is carried out at a slower rate. In the event the individual was unable to equalize, he was "locked out" and brought to the surface (Fig. 2, lockout procedure). Upon completion of the lockout procedure, the pressure test was resumed for the remainder of the group. Upon reaching 50 psi, the pressurization was stopped and the ascent to the surface began. Immediately upon leaving the recompression chamber, each individual was again examined otoscopically. In addition, an examination of the nose and throat was carried out. All findings were recorded. Part II of the questionnaire was completed (see Appendix 1). Every individual with clinical evidence of aerotitis media was ordered to report for a "post-pressure" audiogram and dental examination for further evaluation of malocclusion. Similarly, an equal number of subjects without clinical evidence of aerotitis media was ordered to report for the same evaluation. Following the "post-pressure" audiograms, both groups were ordered to report at 4 — 5 day and 14 — 20 day intervals for repeat audiograms.

The clinical classification of aerotitis media, as described by Teed (3), was followed. This classification is used throughout the U. S. Navy by diving designated hospital corpsmen and medical officers. The classification consists of a grading system 0 - 4, based on the clinical findings as follows:

Grade 0 — Normal

Grade 1 — Retraction of the tympanic membrane with redness in Shrapnell's membrane and along the manubrium.

Grade 2 — Retraction with redness of the entire tympanic membrane.

Grade 3 — Same as Grade 2 plus evidence of fluid or fluid and bubbles of air in the middle ear.

Grade 4 — Evidence of blood in the middle ear, perforation of the tympanic membrane, or both. [3]

The evaluation of malocclusion was performed by a Navy dental officer. At no time did the dental officer know to which group the individuals belonged. The criteria employed were those set forth by Kelly, Harvey, and Ingle. (6, 7, 8)

Audiometric Procedures

For the pre-exposure audiometric examination, the group procedure routinely used for all submarine candidates was accepted. This program tests 50 men at once, at octaves and half-octaves from 500-8000 cps, each ear separately, in 5-db steps from 30 to 0 db with respect to American Standards Association (ASA) 1951 audiometric "zero". It has been shown that the difference between this group version and standard clinical audiometry on the individual patient is less than 5 db at all frequencies — i.e., about the same variation as with test-retest on standard individual audiometry.[9] Such a diotic 8-frequency audiogram was collected for every subject in this study.

This program is inadequate, however, to serve as a baseline audiogram in an especially acute-hearing population where only slight (5 db) threshold shifts may be encountered as a result of a midly noxious exposure of some sort, for the reason that the program presents no tone weaker than the

* Three-Lock Owens Recompression Chamber, Dixie Manufacturing Company Inc., Baltimore, Maryland, Internal Pressure 200 lbs. per square inch.

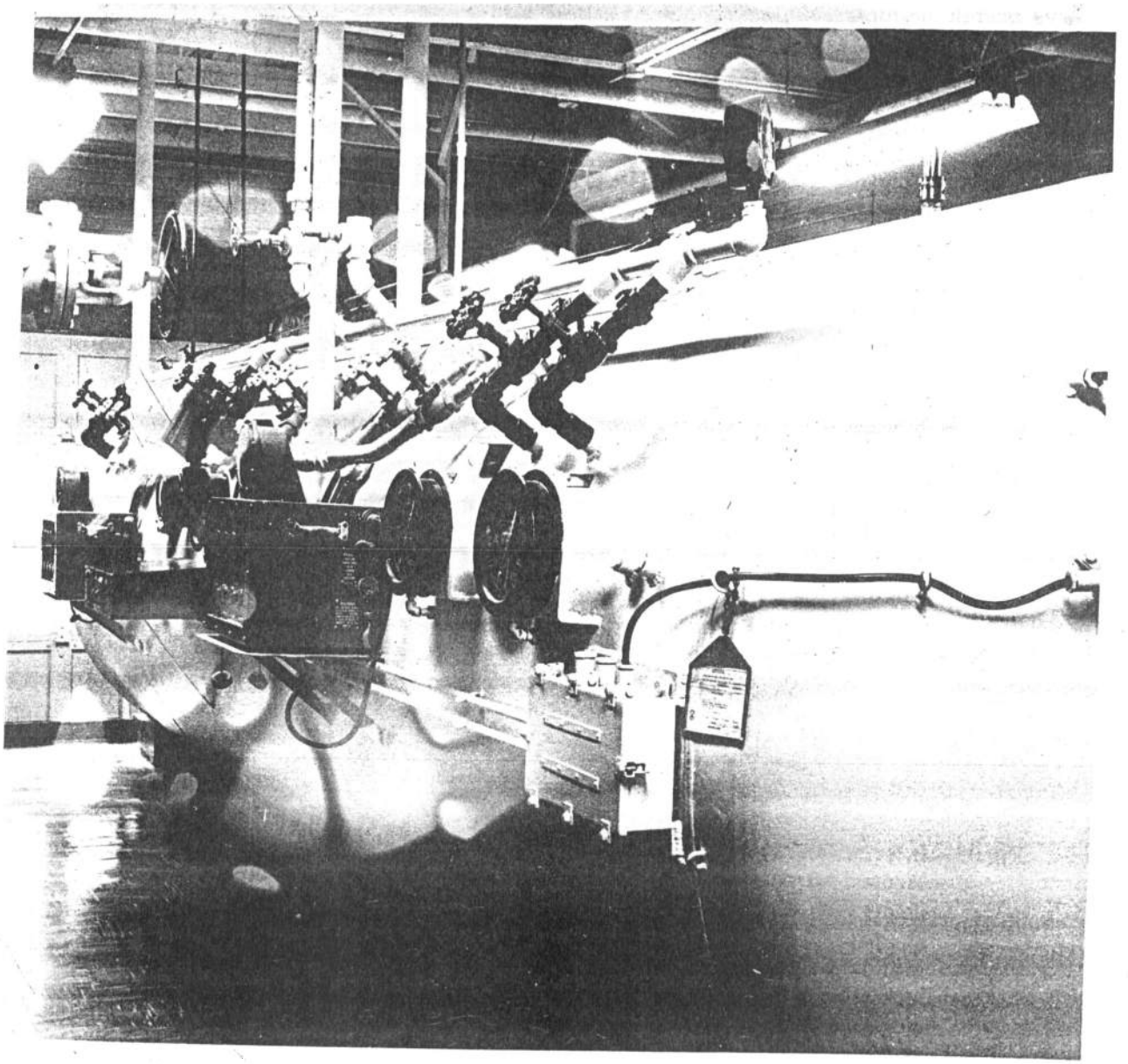

Fig. 1—Exterior View of Recompression Chamber Located on Ground Floor of Escape Training Tank.

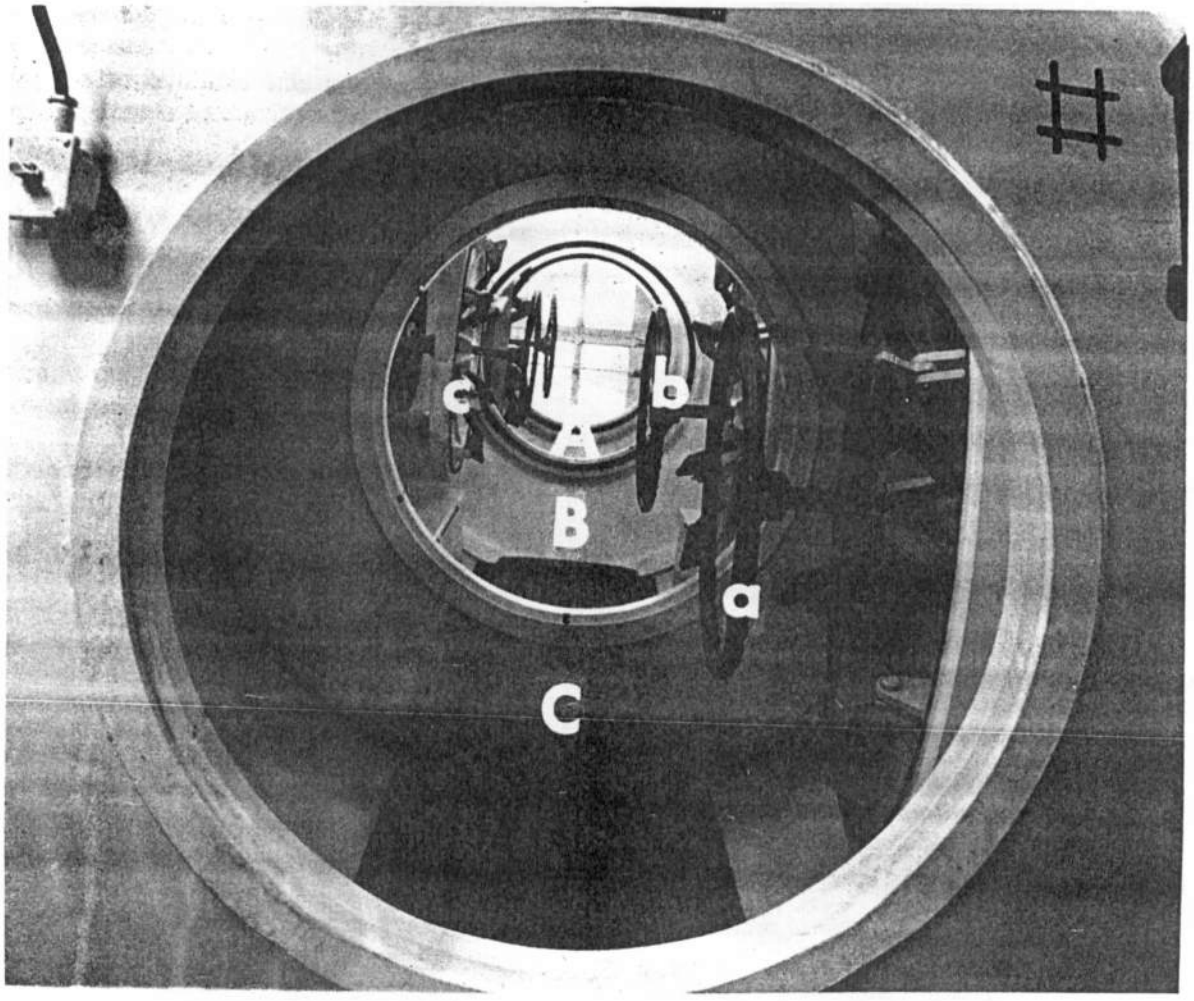

Fig. 2—Interior View of Recompression Chamber. Lockout Procedure: Medical personnel enter the "Medical Lock"—'a,' which contains emergency medical equipment. The lock is pressurized to equal the pressure in 'B,' and medical personnel can enter 'B.' 'B' is the Middle Lock—subjects undergoing the pressure test enter this lock from C. During the pressure test 'B' and 'C' are pressurized simultaneously and equally, with the door 'b' open and doors 'a' and 'c' shut. C is the Outer Lock—subjects enter the Middle Lock —'B,' through the outer lock. Subjects unable to equalize the ambient pressure move from the middle lock to the outer lock, door 'b' is then shut and the outer lock is depressurized. Upon reaching the 'surface,' the subject leaves the recompression chamber through door of 'a.'

"zero" audiometric level. It is not useful to do so in a group setup where noise in the room will mask weaker levels. It is well known that many of our sailor's ears can hear at the minus 5-db level, and a few even lower. Thus, in the case of an ear which can at pre-exposure hear the minus 5-db level, and which shifts 5-db after some noise, the audiometric data will indicate "zero" on both pre-and post-exposure tests, and the 5-db shift will have been unnoticed. Accordingly, for the post-exposure audiogram (c. 2-3 hrs, 4-5 days, 2 wks) the audiograms were collected from only 20 men at a time in a special soundproof chamber with a taped audiometric program which examined down to -10 db.

It was ascertained that 4-5 days after exposure virtually all men had returned to hearing levels which were not further improved if even more time elapsed. The 15-20 day post-exposure audiogram was therefore accepted, where available, as the best estimate of pre-exposure acuity, since it did not have the minor defects of the actual pre-exposure audiogram. The 4-5 day period to full recovery from any effects of the noise exposure (as one of the variables possibly contributing to threshold shift) is known to be conservative. Full recovery from noise was almost certainly accomplished in all subjects within less than 48 hours.

In a few cases the 4-5 day audiogram still shows some shift as compared to the 2-wk audiogram. In these cases one can state that the prolonged recovery could hardly have followed from the noise exposure, but from some minor pathological condition brought on by the pressure test.

Noise Measurement Equipment

A Bruel and Kjaer Model 2203 sound level meter was used to measure the noise levels within the chamber. It was calibrated on the day of use with a piston-phone which delivered 124 db at 250 cps to the microphone through a closed acoustic coupler. The calibration curve of the microphone showed it to be flat through 8000 cps. The sound level meter was then placed in the recompression chamber. Readings were recorded during pressurization and depressurization. The average time of exposure to these noise levels was computed from the log for the dives studied and is stated in connection with our results.

RESULTS

General Findings

The development of aerotitis media during escape training is a frequent occurrence in normal individuals. In this study 36.2% of the individuals exhibited clinical evidence of aerotitis media. No perforations of the tympanic membranes were encountered during this study. Table 1 summarizes these findings.

Table I—Clinical Grading of Aerotitis Media in 432 Male United States Navy Subjects

Distribution of clinical aerotitis media using the Teed Classification. Numbers in parentheses represent same findings except for grade ear injury reversal.

Right Ear	Left Ear	No. Subjects	%
0	0	275	63.6
0(1)	1(0)	26(20)	10.6
0(2)	2(0)	27(12)	9.0
0(3)	3(0)	3(2)	1.1
0(4)	4(0)	2(1)	.6
1	1	10	4.3
1(2)	2(1)	4(4)	.9
1(3)	3(1)	0(1)	.2
1(4)	4(1)	0(0)	.0
2	2	23	5.3
2(3)	3(2)	8(3)	2.5
2(4)	4(2)	1(1)	.4
3	3	6	1.2
3(4)	4(3)	0(0)	.6
4	4	2	.4

Aerotitis media was unilateral in 59.5% of the subjects, and bilateral in 40.5% of the subjects.

Upper Respiratory Infection

In an attempt to document the idea generally accepted among divers, that upper-respiratory infection predisposes the individual to the development of aerotitis media, we compared thirty-three subjects, in whom clinical evidence of acute upper-respiratory infection was found, with those who were normal on physical examination.

Table II depicts these findings.

Table II — Comparison of Subjects With or Without Clinical Evidence of Aerotitis and the Presence or Absence of Acute Upper Respiratory Infection.

	Diagnosis: Acute URI			Diagnosis: No URI	
	No. Subjects		%	No. Subjects	%
Grade	0	13	39.3	262	65.7
Ear Injury	1-4	20	60.7	137	34.3

In the group of subjects in which acute upper-respiratory illness (URI) was diagnosed clinically, 60.7% were found to have clinical signs of aerotitis media; whereas in the group diagnosed as no URI, 34.3% were similarly diagnosed. This finding is statistically significant (P.01), indicating that acute upper-respiratory infection predisposes toward the development of aerotitis media.

As previously noted, 40.5% exhibited bilateral aerotitis media. Table III bears upon whether acute upper respiratory infection contributes to this finding.

Table III — Comparison of the Presence or Absence of Acute Upper Respiratory Infection and Unilateral or Bilateral Otic Involvement

	Diagnosis: Acute URI		Diagnosis: No URI	
	No. Subjects	%	No. Subjects	%
Unilateral	9	45	84	61
Bilateral	11	55	53	39

Since the group diagnosed as Acute URI is so small, no definite statement can be made, but the data appear to indicate that there is a tendency toward bilateral otic involvement in the presence of an acute upper respiratory infection. Pain was the criteria employed to determine whether an individual was experiencing difficulty equalizing the pressure within the middle ear cavity. Of those individuals who complained of pain, 71.3% developed aerotitis. In subjects not experiencing ear pain, the incidence of aerotitis was 18.9%. Therefore, otic pain during pressurization appears to be a reliable index of the probability of developing aerotitis. In the group having acute upper-respiratory infection clinically, 57.6% complained of otic pain. This figure correlates closely with that percentage of subjects having upper respiratory infection who developed aerotitis. One subject in the acute URI group with aerotitis did not experience ear pain.

No relationship was found between the development of aerotitis and complaints of recent but resolved upper-respiratory infections. The salient factor in its development appears to be the presence of acute upper-respiratory infection at the time of the pressure test.

Symptoms of aero-sinusitis occurred in 19.5% of subjects diagnosed as having acute upper-respiratory infections. In the "no" upper-respiratory infection group, 4.8% complained of symptoms of aero-sinusitis. These values indicate that acute URI predisposes toward the development of aero-sinusitis. This group is small and no statistical significance can be determined.

Nasal Allergy

Nasal allergy has been implicated as a predisposing factor in the development of aerotitis media. [10,11] In the group studied, sixteen subjects (3.79%) had histories of nasal allergy. These included history of hayfever (13), allergy to grass pollen (1), animal dander (1), and wool (dust) (1). Because of the physical standards of the U. S. Navy, this group is, of necessity, small and the allergic manifestations mild. None had histories or physical findings compatible with allergic pulmonary manifestations, as this is cause for disqualification from the Submarine Service. In these men, seven (43%) exhibited clinical evidence of aerotitis media. Five of these men exhibited bilateral otic involvement. Only one man had clinical evidence of nasal allergy. He did not exhibit clinical evidence of aerotitis media. One man in this group complained of frontal sinus pain. He had clinical evidence of aerotitis media and an acute upper-respiratory infection. The remaining subjects had neither subjective symptoms of, nor clinical findings of, acute upper-respiratory infection. No attempt was made to treat this group prophylactically prior to the pressure test.

Ability to Perform Valsalva Maneuver

The inability to insufflate the middle ear

has been implicated as a predisposing factor in the development of aerotitis.[3] Prior to the pressure test subjects were asked if they were able to perform this maneuver successfully.

Table IV — Comparison of Ability to Subjectively Perform the Valsalva Maneuver Verses the Development of Aerotitis.

	Able to Perform Valsalva Maneuver		Unable to Perform Valsalva Maneuver	
Grades of ear injury	No. Subjects	%	No. Subjects	%
0	252	66.3	14	45.2
1-4	128	33.7	17	54.8

Table IV reveals that individuals who are unable to perform the Valsalva maneuver prior to exposure to pressure show a predisposition to develop aerotitis media, (P 0.02-0.05 Yates correction applied). It is interesting to note that 45% of those who were subjectively unable to insufflate the middle ear prior to pressure were apparently able to do so when subjected to pressure. This may be related to learning the proper Valsalva technique and the stress associated when one is introduced for the first time to increases in barometric pressure.

The two groups were compared on the basis of the presence of acute upper-respiratory infection. The results show that 8% of the subjects in the group who were able to insufflate the middle ear exhibited clinical evidence of acute upper-respiratory infection. In the group that was unable to insufflate the middle ear, 12% exhibited signs of acute upper-respiratory infection. There is no statistically significant difference between the two groups.

Hypertrophic Adenoidal Tissue:

In order to determine whether enlarged adenoids enhanced the development of aerotitis media, examination of the oropharynx was performed. No attempt to visualize the Eustachian Tube Orifice or Fossa of Rosenmuller was made. The purpose of this examination was to ascertain the relationship, if any, between midline hypertrophic adenoidal tissue and predisposition to aerotitis media.

Figure 3. — Relation of adenoid size to incidence of aerotitis media.

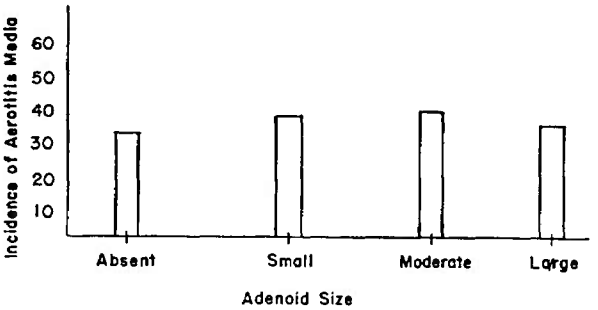

Figure 3 indicates that in this study the presence, absence and size of midline adenoidal tissue did not significantly alter the incidence of aerotitis media.

Dental Malocclusion

For the purpose of classifying the dental occlusion, three criteria were selected which were thought to be the most likely to lend to Eustachian Tube Orifice obstruction. They are: (1) Interference in lateral functional excursions, (2) Obvious mandibular shift when closing into centric occlusion, and (3) Presence of excessive overbite. Cases of lateral functional interference were determined in a manner similar to that described by Kelly,[7] the degree of overbite judged excessive corresponded to the grade "two plus" in the criteria used by Harvey[8] and the presence of a mandibular shift when closing to centric occlusion was determined by the method described by Ingle.[9]

Table V — Frequencies of Malocclusion in Relation to Ear Damage

GRADES of ear damage	0		1-4	
	No. Subjects*	%	No. Subjects	%
Interference in lateral functional excursions	82	91.1%	93	89.4%
Obvious mandibular shift present when closing to centric occlusion	26	28.9%	31	29.8%
Excessive overbite present	15	16.7%	18	17.3%

*Total Number Tested: 194

Chi square analysis revealed no significant differences between the occlusal factors considered in Table V and the development of arotitis media. This indicated that when applying the above criteria, susceptibility to aerotitis media cannot be determined on the basis of dental occlusal factors.

Auditory Acuity

During pressurization, high sound pressure levels (SPLs) are present in the recompression chamber. The level of sound intensity encountered is caused partially by the expansion of compressed air as it enters the chamber. These SPLs as measured by the Bruel and Kjaer sound level meter are summarized in Table VI.

Table VI — Sound Pressure Levels Measured (dB) In Octave-Bands Based On Center Frequencies (C/S)

	500	1000	2000	3000	4000	6000	8000
GAGE 0-8.9 (0-20 ft.)	110	112	113	110	106	103.5	103
Pressure 8.9-22.3 (20-50 ft) psig	106	110	113	115	112	111	109
Mean values during Pressurization	109.7	111.7	114.0	114.7	112.3	110.2	108.
Ventilation at 50 psig (112 ft.)	106	106	112	113	118	112	113
Ventilation at surface (0-psig)	84	112	128	130	130	130	132.

Similar SPLs were found during decompression. Coles reported a fall of pressure in the supply source of as much as 1200 psig when using high pressure air (2700 psi gage). This corresponded to a fall of 6 dB in the overall SPL.[12] The source of compressed air in this study was 3000 psig which was passed through a reducer which lowered it to 150 psig. This pressure remains virtually constant during pressurization and, therefore, no diminution in SPL was noted as pressurization proceeded. The mean exposure time to these SPLs was 8.2 minutes. Our SPL measures are comparable to those reported by Coles and are above the noise levels necessary to produce hearing loss if exposure time is of sufficient length.[12]

The procedure followed for the pressure test does not require that the recompression chamber be ventilated to revitalize the atmosphere within the chamber.[13] It can be seen that the SPLs during ventilation at 50 psi gage would increase the exposure time to noxious SPLs and likely would cause further loss of auditory acuity.[14] Therefore, the effects of SPL and of aerotitis must be considered together in evaluating loss of auditory acuity.

Since individual ears were tested, it is necessary to enumerate the number of ears on which the series of audiograms was completed. Grade 0:185; Grade 1:18; Grade 2:70; Grade 3:20; Grade 4:7. Figure 4 compares the results of the immediate post-pressure audiograms (i.e. within three hours following pressure) minus the baseline

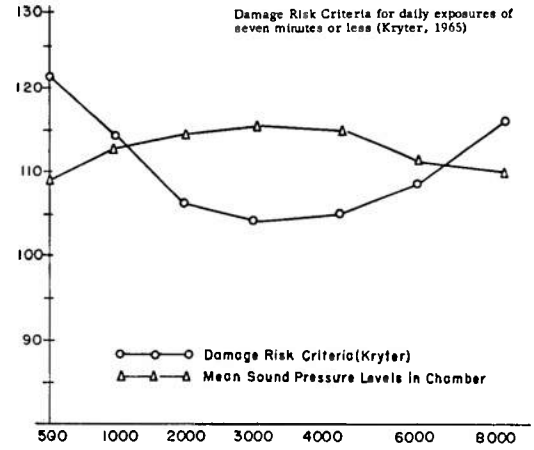

Figure 4—Center Frequency of octave bands (C/S). Ambient sound pressure levels within the recompression chamber and the Damage Risk Criterion.

audiograms, and the 4-5 day post-pressure audiograms minus the baseline values. It can be seen, that, immediately following pressurization (i.e. within three hours), the frequencies at which measurable losses occurred were 3,000, 4,000, 6,000 and 8,000 cps. At the higher frequencies, (6,000 and 8,000), the loss of auditory acuity follows stepwise increments as the severity increases, the exception being the Grade 4 ear injury at the 6,000 cps level. Four to five days following the pressurization, auditory acuity has returned to pre-exposure values regardless of the grade of injury that was incurred.

In order to present the extreme losses in auditory acuity the fifteenth percentile was selected for study (see fig. 5). This represents those ears in each grade category

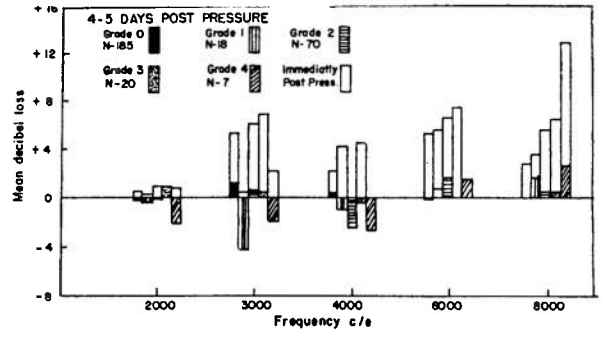

Figure 5—Ear in Each Grade Category which Sustained the Most Severe Audiometric Losses.

which sustained the most severe audiometric losses. The results show generally the same pattern as presented in Figure 4. The mean decibel losses are greater, and returned to pre-exposure value appears to be delayed in Grade 2 ears at 6,000 cps.

DISCUSSION

General.

The incidence of 36.2% aerotitis media which we have found is higher than that previously observed in prospective submarine personnel. However, it is in close agreement with results reported by Haslam, who found an incidence of 38% in divers, and of the U. S. Air Force, where a 34.7% incidence was observed.[15,16] While the method of pressurization differs between submarine personnel and aviation personnel (i.e. aviation personnel experience pressure changes from less than one atmosphere absolute) the differential pressures across the tympanic membrane in both cases are comparable and, of course, the mechanism of Eustachian tube obstruction is the same.

Teed[3] reported that 74% of those experiencing otic pain or discomfort enough to abort pressurization, did so in the initial 12 psig (27 ft) increase in pressure. This is to be expected since the greater relative pressure changes occur within the first atmosphere increment after leaving the surface. As pressurization continues beyond that point, the relative pressure changes become progressively less. In this study, 15 subjects in whom difficulty was encountered in insufflating the middle ear, as manifest by severe otic pain or discomfort of such magnitude as to preclude his continuing the pressure test, did so prior to a pressure change of 12.0 psig. Beyond that point in pressurization, five subjects encountered pain or discomfort of sufficient intensity to voluntarily abort the pressurization. This finding is in agreement with that of Teed.

The mechanism of Eustachian tube obstruction has been the subject of controversy for many years. That Eustachian tube obstruction is the primary factor in the development of aerotitis media has been generally accepted. Hartmann in 1879 clearly showed that the normal Eustachian tube is easily forced open by an overpressure within the middle ear. This mechanism is passive and does not require voluntary action by the individual.[17] However, the mechanism involved in the development of aerotitis media is the development of a negative pressure within the middle ear or, similarly, an increased extratympanic pressure. When a relatively negative pressure exists within the middle ear, the tube does not open passively and air cannot enter the middle ear without the active process of muscle action. Thus, in order to provide the appropriate muscle action the individual must perform the Valsalva maneuver, yawn, swallow, or move mandible from side to side. The muscles believed to effect this process are the tensor veli palantini, levator veli palatini salpingopharyngeus and the constrictor pharyngeus superior.

Most[19] stated that "the lymph vessels in the middle ear connect through the lymph capillaries of the Eustachian tube with those of the lateral pharyngeal wall, that they pass the rear circumference of the tube, through the cartilagenous wall and travel between it and the bone to the lateral pharyngeal gland which lies in the bucco-pharyngeal fascia. The lateral pharyngeal gland is the first and most important lymph node draining the Eustachian tube and middle ear." It follows that dilatation of these lymphatics, produced by the relative negative pressure, could contribute to compromising the lumen and lead to Eustachian tube obstruction. This alone would not appear to be of sufficient magnitude and proper location to cause complete obstruction of the tube.

Aschan[20] showed that the Eustachian tube is open for only short intervals. He demonstrated that active muscular opening begins at the pharyngeal end. Closure of lumen is due to relaxation of the muscles supported by elastic fibers in the tubal cartilage and begins at the aural end of the tube. Further, Aschan postulated that lumen closure was aided by the "capillary forces" between the damp surfaces of the mucous membranes of the tube. Armstrong and Heim[21] demonstrated that when the pressure differential is 90 mm. Hg. (1.76 psi) the Eustachian tube is "locked". At this

pressure differential, it is impossible for tubal musculature to open the tube since the membrane-cartilgenous wall has collapsed tightly. McKibbon[18] showed that the mechanism of this type of locking is purely mechanical "pressure occlusion". He postulated "vital occlusion" as a secondary effect which occured due to the changes of the mucous membranes of the tube.

The "pressure occlusion" as postulated by McKibbon[18] and Flisberg et al[22] is in agreement with experience in the recompression chamber. Flisberg et al[22] demonstrated that when a pressure of -20 to -30 mm Hg. (-35 psi to -.58 psi) exists across the tympanic membrane for fifteen minutes, transudation occurs within the middle ear. Once a pressure gradient of this degree has been established, it is readily understandable that vascular dilatation, plasma exudation, vascular rupture, and eventually tympanic membrane perforation may occur. These pathological processes are responsible for the clinical findings of aerotitis media.

The 71.3% of subjects experiencing otic pain during pressurization exhibited clinical evidence of aerotitis media, whereas 18.9% of those not experiencing pain had clinical aerotitis media. Because of the sensory distribution of nerve endings in the tympanic membrane and the pressure gradient across the tympanic membrane, it would be expected that a higher percentage with aerotitis media should have experienced otic pain. Three factors must be considered in discussing this finding. First, the subjects represent a high-motivation group. It has been shown that motivation can increase the pain threshold in certain individuals.[23] Whether this occurs in this situation is a matter for conjecture. Secondly, since this represents usually the first exposure to increased barometric pressure for these individuals, perhaps overzealous insufflation of the middle ear resulted in the clinical findings of aerotitis media, but otic pain did not occur because the subject was unable to provide a pressure grandient sufficient to cause pain. Armstrong and Heim demonstrated that from 15 to 30 mm Hg. positive pressure gradient in the middle ear produced discomfort or mild pain, while 30 mm Hg. or greater produced progressively severe pain.[21]

Lastly, subjects who have no difficulty in insufflating the middle ear may allow themselves to "fall behind" the pressure and only resort to Valsalva when a diminution in auditory acuity is noted. This occurs prior to recognition of pain and these individuals may have incurred clinical evidence of aerotitis media. Further study regarding these potential causes of aerotitis media is contemplated.

Role of Upper Respiratory Infection

Our data in Table II clearly indicate that acute upper-respiratory infections predispose to the development of aerotitis media. In addition, there is a tendency toward bilateral otic involvement in those subjects in whom the presence of an acute upper-respiratory infection was diagnosed clinically. The mechanism for this finding seems clearly defined. Flisberg et al[24] have shown that when an upper-respiratory infection (common cold) is present, the Eustachian tube orifice can be "locked" by a very small negative pressure. He demonstrated that when efforts are made to insufflate the middle ear by swallowing, further negative pressure spikes are produced due to the action of the muscular opening forces on the tube. In this manner the forces oppose themselves. It can been seen that these conditions in the nasopharynx indicate the presence of an upper-respiratory infection and provide a reasonable situation for the development of aerotitis media. In this case, the opportunity to abort the pressurization must be provided because the Eustachian tube once "locked" will tend to remain in that condition. Oral decongestants have not been employed to date at this activity. This mode of therapy may prove to be of benefit.

Ninety-nine subjects complained of acute URI (23%) at the time of the pressure test. Examination of all subjects revealed clinical evidence of an acute upper-respiratory infection in only 33 individuals. On this basis, it is believed that the subjective complaint of upper-respiratory infection is not an

adequate basis for predicting probability of development of aerotitis media.

In this study no relationship existed between the history of a recent, but not at that moment, acute upper-respiratory infection and development of aerotitis media. These findings are in agreement with those of Liebermann. He reports an incidence of 23.3% aerotitis media in those subjects with a history of a recent "cold". The incidence of aerotitis media was 26.6% in subjects without histories of recent colds.[25]

On the basis of these findings it is recommended that individuals anticipating pressurization should report the presence of an upper-respiratory infection to the chamber personnel. In turn, these individuals should be referred to the medical officer for evaluation. On the basis of this examination, the medical officer can provide the decision as to whether the individual should be exposed to increased ambient pressures.

Role of Nasal Allergy

In this study, seven men (43%) of subjects with a history of nasal allergy exhibited clinical evidence of aerotitis media. Gantt[11] reported a 40% incidence of aerotitis media in subjects having a history of nasal allergy. We found manifestations of acute nasal allergy in only one individual. This man did not exhibit clinical evidence of aerotitis media. Of the seven men with a history of nasal allergy and aerotitis media, clinically, five exhibited bilateral involvement. This incidence of bilateral involvement is higher than that found in any other group. The pathophysiology of nasal allergy (i.e. (1) edema of the mucous membranes, (2) spasm of smooth muscle, or (3) excess mucous secretious in the nasopharynx.) lends support to this observation.

Clein[10] used a somewhat different approach to this general problem. He studied fifty men who suffered painful ears during the pressure test and/or the escape training test (subjects pressurized to 22.22 psig). He found that 32% of these men revealed a definite history of perennial nasal allergy or hayfever.[10] The above findings support the hypothesis of nasal allergy predisposing toward the development of aerotitis media. Long term treatment of such cases with oral decongestants of such may be indicated, since as a group, the risk of developing aerotitis media is somewhat greater than expected in the general population.

Role of the Valsalva Maneuver

Teed[3] showed that insufflation of the middle ear can be demonstrated clinically at the sea level pressure. He believed that this served as a good criteria for determining individual susceptibility to aerotitis media.[1] Shilling et al[2] found that 56.2% of men who subsequently developed aerotitis media had no difficulty with insufflation before the pressure test. In his series the ability to insufflate the middle ear was determined clinically. Of those labeled good, 14.5% developed aerotitis media. In the doubtful category, 25.5% did so while in the group, rated as poor 18 of 21 (95.7%) were similarly diagnosed. Their conclusion was that unless the man was in the lower 2 - 3 percent in ability, a preliminary test to perform the Valsalva maneuver provides only a rough guide to a man's ability to pass the pressure test.

In this series no attempt was made to measure clinically. the ability to perform the Valsalva maneuver properly. We found 54.8 percent of those unable to do so developed aerotitis media, as compared to 33.7% in those in whom no difficulty with insufflation was noted prior to the presuure test. While this finding indicates that the inability to perform the Valsalva maneuver predisposes (P 0.02-0.05 Yates correction applied) to contracting aerotitis media, it does not provide sufficient information to predict whether the individual is susceptible to contracting aerotitis media.

Role of Hypertrophic Adenoidal Tissue

In considering obstruction of the Eustachian tube orifice by hypertrophic adenoidal tissue, an effort was made to determine the relationship between midline adenoidal tissue and the development of aerotitis media. Shilling et al[2] indicated that the relationship between adenoidal size and contracting aerotitis media is at best tenuous. They

reported a great deal of overlap in each category of adenoid size. However, their data indicated reliable tendency for damage to increase in severity as the size of adenoids increase. In our study, no relationship was found between the size of midline adenoidal tissue and an increased incidence of ear damage. In addition, no increase in severity was noted with increased size of adenoidal tissue. These findings indicate that the size of midline adenoidal tissue is not an index determining individual susceptibility to contracting aerotitis media.

Role of Dental Malocclusion

Willhelmy[26] was the first to investigate malocclusion as the cause of ear disturbances when increased pressures were applied. He inserted splints "to open the bite" in a small group of aviators with overclosure of the mandible. He observed relief from ear discomfort during rapid attitude changes. Lowery[27] studied thirty-three men with loss of intermaxillary distance. Of 26 men reporting, nine were symptom free, 14 felt some benefit from wearing the splints and three reported no improvement. Harvey and Morant[28] did not confirm these findings. They reported that malocclusion was found more frequently than normal occlusion and that the incidence of aerotitis was no greater in the group with malocclusion. The use of "teeth splints" failed to provide any improvement.

Kelly[29] postulated that the reasons for the divergent findings were that splints were used in cases where not indicated and by over-opening increased the already strained articular relations, thus aggravating the situation.

In his series, 50 men with clinical evidence of aerotitis media following pressurization were treated after a detailed functional analysis of the occlusion. In five cases, excessive intermaxillary distances were reduced by grinding or by extraction of third molar teeth and only in these cases were splints used to increase the intermaxillary distances. The chief point in Kelly's treatment was the elimination of lateral functional interferences.[29] Following these treatments the men were repressurized, and 46 exhibited no clinical evidence of aerotitis media.

The clinical impressions reported by Willhelmy and Kelly were not substantiated by the findings of our study. It must be recognized, however, that the experimental designs in these studies were different.

The procedure followed in the present study was to refer subjects with, and a similar number without aerotitis media to a dental officer for evaluation. The results of this study indicate that susceptibility to aerotitis media cannot be determined on the basis of dental occlusion factors alone.

Further investigation revealed that during the months of April, May and June 1964, 1,522 men successfully passed the pressure test and only 3 men were physically disqualified because of inability to equalize pressure. This group contains all subjects reported in this study. This indicates to this writer that many factors play a role in the development of aerotitis media and that given ample opportunity to recover from the previous aerotitis media and intercurrent upper respiratory infection, and given experience in performing the Valsalva maneuver, almost all individuals will successfully equalize pressure. This, however, is not meant to imply that these individuals will not develop aerotitis media when subjected to increased ambient pressure at some later date. Indeed, experienced U. S. Navy Divers have albeit infrequently, developed aerotitis media even to the extent of perforation of the tympanic membrane.

Effects on Auditory Acuity

(1) Sound Pressure Levels.—In discussing loss of auditory acuity in subjects exposed to increased ambient pressures in a recompression chamber, two factors that for the most part are inseparable must be considered. These factors are the effect of ambient SPLs present during pressurization followed by depressurization and the role of aerotitis media. For clarity, these subjects will be discussed separately. Coles measured SPLs in the recompression chambers at the Royal Navy Physiological Laboratory. Table VII lists the details of his findings.[12]

Table VII — Sound pressure levels as measured in recompression chambers at Royal Navy Physiological Laboratory.

Compartment	Air Inlet Pressure (psi)	Sound Pressure level (dB re 0.002 microbar) In octave bands of following center frequencies (C/S)							
		L.P.	125	250	500	1000	2000	4000	HP
Main Chamber	3800	116	115	120	127	126	128	122	122
Recompression Chamber	2800	110	108	114	123	124	124	128	121

Coles' conclusions were that the sound levels were clearly hazardous to hearing and must also be deleterious to mental concentration and communication. The SPLs measured in our recompression chamber are somewhat lower than those found by Coles. His results are higher by the values shown in Table 8.

Table VIII—Difference between SPLs in Royal Navy Recompression Chamber as Compared to U. S. Naval Chamber.

Compartment	Air Inlet Pressure (psi)	Sound Pressure levels (DB) mean difference In octave bands of following center frequencies (C/S)				
		500	1000	2000	4000	H.P.
R.N. Chamber minus U. S. Chamber	3800 2800	7.3	13.3	12	12.7	12.7

The inlet piping is so arranged in our cylindrical chamber as to allow the incoming air to follow the contour of the chamber wall and much turbulence is produced, thereby adding to the SPLs. No attempt has been made to install sound attenuating or absorbing material within the chamber.

Damage Risk Criteria for such an exposure has not been precisely delineated. Figure 6 reveals that the mean SPLs are above those necessary to produce a temporary threshold shift (TTS). This finding readily explains the common complaint of subjective hearing loss in the absence of clinical aerotitis media. It can be seen that the SPLs approach the Damage Risk Criteria (7 minutes) at 1000 and 8000 cps. At 2000, 3000, 4000, and 6000 cps the Damage Risk Criteria (7 minutes) is exceeded. The maximum duration of exposure to the SPLs encountered from 4800-9600 cps, according to Fig. 1 of Ref. 30 (See Fig. 4 - p. 8), is

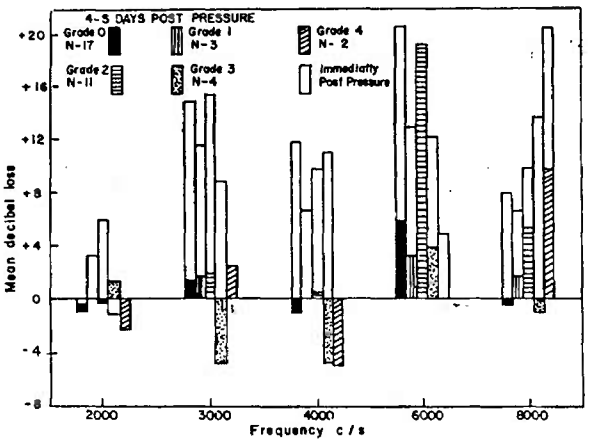

Figure 6—Ears in Each Grade Category showing Mean Decibel Losses Immediately Post-Pressure and 4-5 Days Post-Pressure.

seven minutes and in the octave band from 1200-2400 cps is five minutes. The mean exposure time for the subjects studied was 8.2 minutes. Thus, the recommended duration of exposure has been exceeded.

Mean TTS Data — No significant temporary threshold shifts (TTS) were noted below 2000 cps. Figure 4 shows that the immediate post-pressure audiograms (i.e. within three hours after the pressure test) revealed a TTS of significant degree at 3000, 4000, 6000 and 8000 cps. The expected time of recovery for the SPLs encountered is approximately 16 hours.[31] The mean audiogram for the entire group returned to baseline values by 4-5 days post-pressure regardless of grade of ear injury.

(2) TTS Data of 15th Percentile.

The audiograms for those sustaining the greatest TTS (15th percentile) showed no significant changes below 2000 cps. (See figure 5). Study of the 4-5 day post-pressure difference audiograms revealed that those with no clinical evidence of aerotitis media had returned to baseline values, with an exception at 6000 cps where a 6-dB TTS is noted. Of those with clinical evidence of aerotitis media, ears with Grade 2 injury showed a TTS at 6000 cps which did not show any improvement on the 4-5 day post-pressure audiogram, and the two ears with

Grade 4 injury at 8000 cps had improved to within 50% of baseline value. In the group with Grade 2 injuries there were two of eleven subjects who sustained losses of 45 and 55 dB. Ward[31] has demonstrated that recovery to pre-exposure threshold requires about 16 hours if the initial loss is less than 40 dB. However, if 40dB loss is exceeded, it may require more than 16 hours and even weeks for full recovery.

All subjects at the 15th percentile of loss had regained baseline values as seen in the 14-20 day post-pressure audiograms. None of the subjects with Grade 4 injuries exhibited TTS greater than 30 dB. On this basis one can postulate that the loss of auditory acuity in the two ears with Grade 4 injuries was due to aerotitis media.

Recommendations. In view of the above findings, the following recommendations are offered: (a) An effort should be made to reduce the SPLs within all U. S. Navy recompression chambers. This may be done by installation of a muffler-like device such as the British have successfully employed.[32] (b) Sound-absorbing material should be installed within the chamber. This material must of necessity be nontoxic, non-flammable, and must not require volatile adhesive agents. (c) Protective ear devices such as supra-aural muffs might prove to be of some value, but it might be that the disadvantages would outweigh the advantages. The disadvantages include the further deterioration of voice communication and in the case of plugs, the possibility of subjects contracting the Reverse Ear Syndrone.[33]

(3) **Aerotitis media.** Results of this study indicate that there is almost a step-wise increase in the TTS with increasing grade of injury at 6000 and 8000 cps. The increments are small, except for the Grade 4 injuries at 8000 cps., where a 12.7 dB loss was found immediately post-pressure. If one postulates that with increasing injury an increased negative pressure is present in the middle ear as well as hemorrhage and fluid, then the results are readily explainable on the basis of the findings of several investigators:

Crowe and Guild[34] stated that acute middle ear lesions in animals resulting from occlusion of the pharyngeal orifice of the Eustachian tube may cause greater impairment for the high tones than for the low tones. In this study only hightone impairment was found (i. e. 2000-8000 cps). Lock[35] reports that with slight negative pressures in the middle ear there was impairment of human thresholds at frequencies greater than 5793 cps. He found that as the negative pressure was further increased, thresholds for middle tones and low tones became progressively impaired. Campbell and Hargreaves[36] studied one individual by producing a negative pressure in the middle ear and found losses at the low frequencies only. Flisberg et al[22] produced negative pressures within the middle ear and found increasing loss of auditory acuity at all frequencies, but greatest for the frequencies less than 1000 cps. At -30 mm Hg they found a 12-14 dB loss for the frequencies greater than 1000 cps. They demonstrated that 60% of the tympanic membrane's maximum capacity for volume displacement, when measured from the resting position, has already been elicited by a middle ear presure of -10 mm Hg. From this point on, the middle ear assumes the characteristics of a rigid chamber. Further, they showed that with a negative pressure of -20 to -30 mm Hg, transudation within the middle ear can be demonstrated after 15 minutes. It seems reasonable to assume that in the Grade 3 and 4 injuries negative pressures greater than -30 mm Hg have occurred.

One would expect greater TTS in the lower frequencies, if ossicular movement was impaired. Fluid in the middle ear with an otherwise normal conduction mechanism causes a greater loss at the higher frequencies. This results from alterations in the mass of the conduction mechanism.[37] Consequently, the energy required to overcome this inertia in a given period of time is greater for the higher frequencies. In Figure 4, as previously noted, there was virtually no improvement in the Grade 2 injured ears at 4-5 days post-pressure. The clinical diagnosis of this grade of injury is evidence of retraction of the tympanic membrane and gross hemorrhage into it. By the mechanism of clot formation the

tympanic membrane would reduce its elasticity and increase in mass and thus interfere with the normal vibrations for higher frequencies.

The results of this study indicate that aerotitis media, while possibly responsible alone for loss of auditory acuity, exhibits shortlived sequelae of no consequence following acute exposure to increased ambient pressures.

SUMMARY

1. Four hundred and thirty-two male subjects undergoing pressurization in a dry cylindrical tank were studied. Ambient sound-pressure-levels within the recompression chamber during pressurization were 110-115 dB SPL in relevant octaves sustained for about eight minutes.
2. Out of 432 subjects, 274 or 36.2 per cent were found to have developed aerotitis following pressurization.
3. Of the factors studied, acute upper-respiratory infection showed the most causal relationship to the development of aerotitis media. Other factors that appeared to predispose an individual to the development of aerotitis were: Inability to insufflate the middle ear, and Nasal allergy. Factors that did not appear directly related to its development are: Size of midline adenoidal tissue, and Dental malocclusion.
4. The findings of this study indicate that no prolonged or permanent loss of auditory acuity results from either the sound-pressure-levels experienced or the contracting of aerotitis media following an acute exposure to increased ambient pressures of 50 psig.
5. Recommendations are made concerning medical handling of pressurized groups, and method of reducing the effects of accompanying noise. These are:
   (a) That individuals, prior to pressure testing and actual escape training, should be asked to report the presence of an upper-respiratory infection.
   (b) That individuals so reporting should be referred to the medical officer for evaluation and receive medical clearance before being subjected to increased pressures, wet or dry.
   (c) That an effort should be made to reduce the sound-pressure-levels (SPLs) within all Navy recompression chambers. This would include the use of air inlet mufflers and other practical sound-proofing methods that would not affect chamber safety adversely.

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APPENDIX I

MEDICAL QUESTIONNAIRE

MEDICAL QUESTIONNAIRE

Recorder..... Number.....

I. To be completed by the trainee before the presure test: Date.....

Name:..... Rate:.....

Date of birth:..... Ship/Station:.....

1. Have your tonsils and adenoids been removed surgically? Yes ( ) No ( )

2. Do you have:

   (a) Frequent colds: Yes ( ) No ( )
   (more than 5 per year)

   (b) Frequent sore throats: Yes ( ) No ( )
   (more than 5 per year)

   (c) Frequent sinus problems: Yes ( ) No ( )
   (more than 5 per year)

3. Do you have a cold now? Yes ( ) No ( )

4. Have you had a cold:
   (a) Within the past 7 days: Yes ( ) No ( )
   (b) Within the past 7 to 14 days: Yes ( ) No ( )

5. Have you had a sore throat:
   (a) Within the past 7 days: Yes ( ) No ( )
   (b) Within the past 7 to 14 days: Yes ( ) No ( )

6. Have you had a sinus problem:
   (a) Within the past 7 days: Yes ( ) No ( )
   (b) Within the past 7 to 14 days: Yes ( ) No ( )

7. Have you had your hearing test? Yes ( ) No ( )

8. Do you have any allergies? Yes ( ) No ( ) (Drugs, pollen, hayfever, sinusitis, etc.)

   (a) If yes, to what:.....

9. Do you grind your teeth at night? Yes ( ) No ( ) Don't know ( )

10. Do you chew most of your food on one side of your mouth?
    Yes, on right side ( ) Yes, on left side ( )
    No, I chew equally on both sides ( ) Don't know ( )

11. Does your jaw joint click or pop when you chew? Yes ( ) No ( )

12. Are you able to clear your ears now? (Yes ( ) No ( )

II. To be completed by the trainee after the pressure:

1. Time of pressure test .....

2. During the pressure test did you have pain in your ears? (mark one)
Right ( ) Left ( ) Both ( ) Neither ( )

3. Did you have a pressure sensation or pain in your (mark one)
Forehead ( ) Cheeks ( ) Eyes or behind them ( ) No pain ( )

4. Were you dizzy? (mark one)
For 10 seconds ( ) 10 to 60 seconds ( ) More than 60 seconds ( )
not dizzy ( )

5. Did you have pain or discomfort in your teeth? Yes ( ) No ( )

6. Do you have ringing in your ears NOW? Right ( ) Left ( ) Both ( )
No ( )

7. Do you think your hearing is affected now?
A little ( ) Moderately ( ) Greatly ( ) No ( )

III. To be completed by the examiner.

1. Grade ear injury—.....

2. Tonsils (size) Absent ( ) Small ( ) Medium ( ) Large ( )
Exudate ( ) Erythema ( ) Edema ( ) Acute U R I ( )

3. Adenoids (size) Absent ( ) Small ( ) Medium ( ) Large ( )

4. Nose

(a) Normal ( ) Red ( ) Exudate ( ) Boggy ( )

(b) Deviation of septum Yes ( ) No ( )