INVESTIGATION OF OTOLOGICAL DISORDERS IN DIVING

by

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1. INTRODUCTION

Clinical problems involving the ears are the commonest cause of morbidity amongst divers, especially during the initial stages of their training. The presentation of these problems may be very variable, and there is a relative paucity of literature involved in the classification and investigation of them. In the two most popular texts on diving medicine, one (Miles; 1966) deals with some disorders of the external and middle ear, but virtually omits the more serious disturbances of inner ear function, whereas the other (Bennett and Elliott, 1970) omits any reference to the subject.

This lack of information is partly attributable to the failure of communication between the specialists in underwater medicine and otology. Otologists tend to be called in after the accidents have happened, and without any pre-incident investigations being performed. It is the absence of base line recordings of otological function that has bedevilled the acquisition of information regarding these disorders. Although most pre-diving medical examinations include a general history of otological disorders, and a clinical examination of the ears, too often there is an absence of adequate audiometric testing, and frequently when an otological problem has occurred there is an inadequate investigation of the cause and the effect. Such all embracing terms as "aural barotrauma" or "aero-otitis" are used to cover a wide range of disorders with an equally varied range of presentation and sequelae.

This report is meant to highlight some of the facets involved in otological examination of divers, both as a routine prior to diving training, as a regular periodical check on otological function, and finally as a method of investigating otological disease. It also covers, en passant, some of the research performed in otological aspects of diving at the RAN School of Underwater Medicine. More comprehensive assessment of the results is obtainable elsewhere (Edmonds, 1970; Gray and Edmonds, 1970; Edmonds, 1971; Freeman and Edmonds, 1972). Case reports are given when these serve to illustrate the investigation required in an otological disorder associated with diving. Experimental results are also given when these serve to illustrate relevant investigations into general otological aspects of diving.
The commonest reason for failure in diving courses has been shown to be otological (Caille, 1969).

2. CLINICAL EXAMINATIONS

History

It is common practice to exclude from diving all those candidates who have a chronic or recurrent infective or inflammatory disorder of the upper respiratory tract. This includes sinusitis, rhinitis, tonsillitis, pharyngitis, otitis externa and media etc. These conditions tend to be aggravated by diving, and some predispose to difficulties in equalising pressures within gas spaces e.g. blocking the sinus ostia, eustachian tubes etc. Because of this latter effect, they may be instrumental in the production of barotrauma i.e. tissue damage due to pressure changes.

The candidate must also be able to perform the Valsalva manoeuvre without the use of excessive force. This is performed while the medical officer is inspecting the tympanic membrane with an otoscope. Any perforation of the tympanic membrane will exclude the candidate from diving, as will a tendency to recurrent perforations. These predispose to middle ear infection and vertigo (see later). Acute perforations heal satisfactorily in a month or so, whereas chronic perforations may require tympanoplasty.

It is not uncommon when dealing with diving candidates to observe large exostoses in the external canal. This is an occupational complication noted in subjects who spend a great deal of time surfing and diving. Although these exostoses are observed with great frequency, they have not caused any notable problems at this school. Not once have they totally obstructed the external canal, or prevented equalisation of pressures within this canal. It is also common practice to remove any large accumulation of cerumen, so as to prevent the occurrence of external ear barotrauma and to prevent unequal caloric stimulus from water in the external canals, causing vertigo (see later).

No recommendations are made to alter the above procedures.
The significance of hearing loss due to the pressure changes experienced in diving has recently been reassessed and verified (Harris, 1969; Gray and Edmonds, 1970; Edmonds, 1970; Freeman and Edmonds, 1972). The medical literature on the subject of hearing loss with diving had previously been questioned because of the absence of objective assessments carried out prior to the cochlear damage. This situation was overcome by performing audiometric assessments of all diving candidates, prior to their being exposed to hyperbaric conditions, and then repeating these examinations, usually on an annual basis. In this way serial audiometric estimations were obtained prior to the hearing loss resulting from diving accidents. With this information it was shown that a total or partial sensorineural deafness is possible following forceful Valsalva manoeuvres, when the diver attempts to equalise middle ear pressures. Cases 1 and 2 respectively demonstrate the sequence of events.

Case 1.

This diver had many pre-incident normal audiograms and had one audiogram a week prior to the dive. During this dive he experienced difficulty in clearing his left ear i.e. equalising pressure in the middle ear to relieve the sensation of pain as the middle ear space contracted during descent. The dive was performed in a recompression chamber. Four days later he attempted another RCC dive, again receiving severe pain in his left ear at approximately 25 feet depth, and was unable to successfully perform the Valsalva manoeuvre. He ascended to 15 feet with relief, and then descended to a depth of 66 feet. The total duration of the dive was no more than 5 minutes, and there were no symptoms during ascent. On leaving the RCC he noted tinnitus and presented to the medical officer on the following morning with complete deafness in his left ear. This was permanent. (See Figure 1, (a) and (b).)

Case 2.

This man had a normal audiogram prior to his diving accident. He attempted to descend to 20-25 feet, and noted pain in both ears. He developed tinnitus at that time, and decided to ascend. After surfacing, tinnitus and deafness were noted in the right ear, and audiograms carried out revealed a high frequency sensorineural deafness on the right side, which has persisted (Figure 2 (a) and (b)).
These two cases are typical of others which have been verified by the comparison of pre- and post-incident audiograms. The common factor appears to be the difficulty in performing the Valsalva manoeuvre, with over-forceful attempts at this. It occurs during descent i.e. associated with middle ear barotrauma. The result of this is the development of either a total or partial sensorineural deafness, of which the latter is often only able to be demonstrated if the audiogram extends to the higher frequencies viz 4000, 6000 and 8000 Hz.

Middle ear barotrauma of descent i.e. "middle ear squeeze" results in characteristic otoscopic changes in the tympanic membrane (McFie, 1964), and is likely to result in a number of subjective impressions which are described as deafness, fullness, blocked sensation, woolly hearing etc, but which have very little associated audiometric change. Previous workers (Shilling and Everley, 1942; Shilling, 1945; Haines and Harris, 1946) described the effects of compression on hearing acuity. Some of these findings were contradictory both with other authors and within their own reports, and thus a further investigation was performed (Gray and Edmonds, 1970).

Having separated the rare cases of internal ear barotrauma resulting in total or partial sensorineural deafness, these workers decided to prospectively assess the common effects of diving on auditory acuity. Experiments were conducted to demonstrate the following:

Experiment 1. The effects of repeated Valsalva manoeuvres on auditory acuity.

Experiment 2. The common effects of middle ear barotrauma of descent on auditory acuity.

Experiment 3. The endurance of any of the changes noted in Experiments 1 and 2.

Experiment 4. The auditory acuity of divers without specific otological disorders.

Four groups of divers were examined:

Group 1: This consisted of 50 candidates who successfully completed the compressed air diving course. The influence of repeated Valsalva manoeuvres on auditory acuity was assessed by audiometric testing of these candidates prior to and at the conclusion of the three weeks course. All candidates were under the age of 30 and were previously non-divers. Those who developed symptoms of aural barotrauma were selectively excluded from this group and placed into Group 2.
Result. The audiograms performed prior to the commencement of
the diving course showed no significant deviation (at the 0.05
level) from those performed at the end of the three weeks
course. The greatest impairment was at the 6000 Hz, where
there was an average of 1.5 decibel loss. This was only
significant at the 0.1 level (Figure 3).

Group 2. This group comprised patients with middle ear
barotrauma of descent i.e. pain and difficulty with equalising
the middle ear cavity via the eustachian tube, during descent.
Audiograms were performed on these divers both before their
course and within a few hours of sustaining the barotrauma.
Based on the subject's statement as to which ear was affected,
the group was subdivided into the effects on the subjectively
involved ear or the subjectively uninvolved ear. The pre-
and post-barotrauma audiograms on the affected ear (Figure 4)
showed a hearing loss throughout, with a loss at the 500, 1000
and 6000 Hz being significant at the 0.05 level, and the loss
at 4000 and 8000 Hz being significant at the 0.01 level.
The hearing loss was quantitatively small, being 6.3 decibels
at 8000 Hz, 4.1 decibels at 4000 Hz and 3.1 decibels at 500 and
1000 Hz. Audiometric changes in the unaffected ear demonstrated
a similar pattern and trend, but of a much less degree and not
statistically significant, even at the 0.1 level.

Group 3. This comprised the same subjects as Group 2, but
examined 1-3 weeks after the incident. The results were compared
with their pre-diving audiograms. None of the hearing loss
noted in Group 2 persisted.

Group 4. This involved full time divers who had never suffered
from documented episodes of decompression sickness or internal
ear barotrauma. This group's audiograms were compared with
those of diving course candidates i.e. those who had never
been subjected to diving. There was no significant detriment
noted in the divers' hearing, and in fact there was an
increased acuity over the 500 and 1000 Hz range.

Experiment 5. By comparing the standard deviations of:

- Pre- and post-diving audiograms of Group 1;
- pre and post aural barotrauma audiograms of
  Group 2;

.../pre-incident
Fig. 3

Fig. 4
pre-incident and post-recovery audiograms of Group 3;

audiograms of Group 4 and pre-course audiograms of Group 1;

there was no evidence of any greater variability in the post-diving than in the pre-diving measurements - calling into question the often quoted variability in auditory acuity following exposure to diving activities.

These results demonstrated that unless one develops middle ear barotrauma there is unlikely to be any appreciable change in hearing acuity due to diving. If middle ear barotrauma does occur then the hearing loss extends throughout the 500 to 8000 cps range, and although it is statistically significant it is quantitatively very small. This hearing loss is reversed over the ensuing 1-3 weeks. The temporary and mild impairment of hearing associated with aural barotrauma is conductive in type, with a possible sensorineural component.

Although permanent, and sometimes total, sensorineural deafness is possible following middle ear barotrauma of descent with forceful Valsalva manoeuvres, there is no evidence from the above work that such a change is either common or cumulative.

Perforation of the tympanic membrane, as has frequently been reported, results in a flat hearing loss of 5-10 dB, with a rapid restoration to normal as the perforation heals.

The hearing loss associated with decompression sickness, especially when this is due to helium, has been described recently (Harris, 1969). This is thought to be amenable to treatment by recompression, and is added reason for routine pre-incident audiograms.

Exposure to loud noise (compressors, recompression chamber environments) is also encountered in diving environments, requiring periodical audiometric assessments.

Recommendation

All diving candidates should be given audiometric testing with a 250-8000 Hz range, prior to commencement of the diving course. This assessment should be repeated periodically, and in the RAN School of Underwater Medicine, it is on an annual basis.
basis. Following any otological problems an audiometric assessment of a similar range should be made. In those cases of barotrauma which show a severe sensorineural hearing loss, specialist advice is indicated to assess whether surgical intervention is warranted. Such procedures are experimental at this stage. Because of the known history of subjects with this disorder to have subsequent episodes, it is probably wise to advise against further diving in those so affected. When there is a total sensorineural loss in one ear (as in Case 1) there should be no suggestion of further exposure to hyperbaric environments.

4. VERTIGO AND VESTIBULAR FUNCTION

The importance of orientation underwater has obvious relevance to the safety of the diver. Probably the most bewildering factor causing disorientation in diving is vertigo. This is a common disorder amongst divers and has been demonstrated in previous series (Rivera, 1964; Lundgren, 1965; Terry and Dennison, 1966). Each of these general surveys tends to stress the production of vertigo due to a specific disease entity, as opposed to discussing its varied aetiologies. The problem of the aetiology has recently been discussed and a classification presented (Edmonds, 1971).

CAUSES OF VERTIGO IN DIVING

1. Unilateral caloric stimulation from -
   a. Perforated tympanic membrane
      - middle ear barotrauma of descent ("middle ear squeeze");
      - underwater blast wave (e.g. being "finned").
   b. External auditory canal obstruction
      - cerumen;
      - otitis externa.

   ...

   2. Inner
2. Inner ear barotrauma from -
   a. External ear barotrauma of descent ("reversed ears")
   b. Middle ear barotrauma of descent ("middle ear squeeze") and over-forceful Valsalva manoeuvres;
   c. Middle ear barotrauma of ascent ("alternobaric").

3. Decompression Sickness.

4. Abnormal gas pressures:
   a. Inert gas narcosis
   b. Oxygen
   c. Carbon dioxide
   d. Others.

5. Unequal vestibular responses to bilateral stimuli.

6. Miscellaneous.

Those cases resulting from unequal caloric stimulation are readily diagnosable from history and otoscopic examination. Permanent damage would not be expected with correct management.

The cases of inner ear barotrauma require more than merely a history and physical examination. In each of these cases the stimulus is predominantly unilateral and the damage, permanent or temporary, is related to an excessive pressure gradient within one ear i.e. the stimulus to the vestibular apparatuses is unequal. Cochlear and vestibular function may both be affected and therefore must both be assessed. Such cases have previously been described (Edmonds, 1971) and the standard otological investigations of audiometry and electro-nystagmography, with caloric stimulation and positional testing, are employed to clarify the type and extent of damage.

Decompression sickness is also usually unilateral in its effects, and the standard tests for cochlear and vestibular function are applicable. Cases of hearing loss (Harris, 1969) and vestibular disorder (Edmonds, 1971; Freeman and Edmonds, .../in
in preparation) have been described. In many cases the damage is partly or completely corrected by adequate recompression therapy. Vestibular function can be monitored to demonstrate that adequate compression has been administered in order to correct the abnormality, or to demonstrate that the decompression does not result in a recurrence of vestibular damage. An ENG under hyperbaric conditions is performed in the following manner:

The typical ECG electrode plug of the RCC may be used to transmit the ENG information. To simplify the technique, the left arm lead of the ECG lead is replaced by the left eye lead of the ENG; the right arm lead of the ECG is replaced by the right eye lead of the ENG. The right leg lead of the ECG, being the indifferent electrode, is replaced by the central indifferent electrode of the ENG. Using this technique an ENG type tracing (but needing correction for speed and sensitivity) can be obtained from some ECG recorders using maximum sensitivity. Alternately an EEG recorder using a single channel and with a chart speed of 10 mm/second could be used. However the specially designed ENG recorder (Cardio-trace 3050) made by Watson Victor (Australia) is portable and able to be used wherever one can use an ECG). The silver stick-on electrodes are more applicable than the strap-on electrodes of the ECG, for obvious reasons. ENGs performed under compression are described and illustrated later.

Case 3

In October, 1969, this man was exposed to many ascents and descents, diving for 30 minutes with a 60% O₂ mixture on a rebreathing set, to a depth of 45 feet. He had difficulty in clearing his ears, and needed to perform forceful Valsalva manoeuvres. There was no difficulty noted during the dive. When he returned to the boat and climbed inboard, he noted that he had become "dizzy". He described this as a spinning sensation, and he was unable to stand. He obtained some relief by sitting and lying down. This disturbance persisted for only 10 minutes or so. He also noted left ear tinnitus, which persisted for some weeks. Audiograms demonstrated a marked sensorineural hearing loss on the left side; electronystagmograms showed spontaneous nystagmus, and caloric testing verified a peripheral type impairment of vestibular function.
function. Two weeks later the patient still had evidence of vertigo, noted especially when tilting the head, and lasting for a few minutes. He also noted vertigo when lying down, and although he could lie on his right side without any disturbance, it made it impossible for him to lie on his left side. Even at that date there was a rotatory nystagmus noted, and aggravated when he moved his head from the horizontal to the left position. Repeat audiograms and electronystagmograms performed two years later revealed a permanent sensorineural hearing loss but normal electronystagmograms with caloric testing.

In the investigation of vertigo due to the inhalation of abnormal gas pressures, there is little experimental or clinical evidence to conclusively incriminate any one specific gas. Some anecdotal evidence has been accrued to suggest the production of vertigo with inert gas narcosis, and also from alteration in the oxygen and carbon dioxide pressures. Because of the extreme paucity of information, experiments were designed in which the effects of different gas pressures within ranges encountered in diving practice were observed on the production of vertigo in normal subjects.

Experiment 6. 12 subjects were monitored with the ENG while lying in the supine position and with the head elevated to 30°. They were then given an anaesthetic bag and mask, which fitted tightly onto the face. 10% oxygen in nitrogen was administered for five minutes at a flow rate of 10 L/min. The mask was then removed, but the ENG monitoring continued for a further 5 minutes. Positional testing was performed during the latter period.

Result. No evidence of nystagmus due to this degree of hypoxia was obtained.

Experiment 7. With the same equipment and subjects, a 5% carbon dioxide in oxygen mixture was administered.

Result. No evidence of nystagmus was obtained with the subjects breathing 5% CO₂ in O₂, but there were subjective impressions of vertigo in 5 subjects following the withdrawal of the gas mixture. Some ENG support was obtained to support this symptomatology.
Experiment 8. Those subjects who demonstrated any symptoms or ENG abnormalities with 5% CO₂ in O₂ were then given 100% O₂ under identical conditions.

Result. No evidence of vertigo or nystagmus was obtained.

Experiment 9. One subject was given 5% CO₂ in O₂, at a flow rate of 20 L/min for 20 minutes, and monitoring continued for 5 minutes thereafter.

Result. No aggravation of nystagmus or vertigo could be elicited with this technique.

It appeared that there was no experimental evidence in favour of the production of vertigo or nystagmus with hypoxia or hypercapnoea within the ranges encountered in diving. There did appear to be some evidence for the production of these effects following the removal from a high CO₂ environment. This is consistent with the cases of "CO₂ off effect", described by submariners.

An attempt was made to assess the aetiological influence of high nitrogen pressures (inert gas narcosis, nitrogen narcosis) and high oxygen pressures (oxygen poisoning, oxygen toxicity) on vertigo. It was decided to use realistic and practical pressures, likely to be encountered in routine diving, and not extremes of nitrogen or oxygen tensions which would be likely to incapacitate the subjects. The following experiment was performed.

Experiment 10. Six subjects were exposed to a 180 ft "dive" in a RCC, following a compression rate of between 60 and 90 feet per minute. The subjects were in a supine position with the head elevated at a 30° angle to the horizontal. Positional testing was performed both at surface pressures and at maximum depth. Instructions were given to the subject by a series of auditory signals passed through the chamber. Throughout the descent the patient needed to equalise the middle ear by performing Valsalva manoeuvres. ENG monitoring continued throughout the 2-3 minutes descent and the 7-8 minutes at 180 feet, and during the ascent to 60 feet at a rate of 1 foot per second.

.../During
During this ascent the diver would have experienced the escape of gas bubbles through the eustachian tubes. On arrival at the 60 feet stop, he was given 100% oxygen for a period of 10 minutes. During that time positional tests were also performed and the ENG monitoring was continuous. After this time at the 60 feet stop the subject ascended to the surface at a rate of 10 feet per minute. The subject remained immobile following the ascent, with the ENG monitored for a further 5 minutes (see Dive Profile, Figure 5). During this whole procedure the diver is thus subjected to:

- the effects of descent ("middle ear squeeze") with Valsalva manoeuvres
- high nitrogen pressures ("inert gas narcosis") during the stay at 180 feet
- middle ear gas expansion of ascent (alternobaric effect) during the ascent to 60 feet
- high oxygen pressures (oxygen toxicity) at the 60 feet level
- further alternobaric effects from 60 feet to the surface, and
- the oxygen "off effect" during and subsequent to the final ascent.

Result. It was demonstrated that nystagmus did seem to predominate at the 180 feet stop, supporting the concept of inert gas narcosis aggravating, if not producing, vertigo. There was no consistent effect noted during descent or ascent, and no definite evidence of nystagmus aggravation with the inhalation of oxygen, either under high pressure, or following its removal. (Figure 6A - F). Other workers (Unsworth, personal communication) have demonstrated nystagmus as part of the oxygen toxicity syndrome when subjects are exposed to prolonged high oxygen tensions and have other manifestations of toxicity.
In those cases of divers developing vertigo from either unilateral caloric stimulation, inner ear barotrauma or decompression sickness, the inference is that the major stimulus is unilateral. In the case of the vertigo aggravated by abnormal gas pressures, it is difficult to make such a proposal. It is possible that the effects may be more central, or that the stimulus may be equal in both peripheral vestibular apparatuses, with some otherwise unimportant inequality of vestibular responses. This concept of unequal vestibular responses resulting in vertigo is a source of much potential research in diving medicine. There are many stimuli in the undersea and hyperbaric environments that may be capable of producing vertigo, especially if an underlying inequality of vestibular responses is present. The stimuli which may well act equally on both vestibular systems, but may produce vertigo if vestibular sensitivity is unequal, include:

- caloric effects
- barotrauma effects (both descent and ascent)
- abnormal gas pressures.

It seems important that in the investigation of cases of vertigo in diving, the conditions under which the vertigo occurred are simulated as closely as possible. Cases 4 and 5 demonstrate the occurrence of vertigo and nystagmus in divers who had a previously demonstrated abnormality of inner ear function.

**Case 4**

This very experienced diver had never had any episodes of vertigo prior to him developing decompression sickness involving the vestibular division of the internal ear, at a depth of 140 feet after being incorrectly decompressed from 515 feet. He was severely incapacitated with vertigo, vomiting and prostration for many days, but was not given the recompression therapy needed to ameliorate or cure his condition. Subsequent to this episode he was noted to have a positional vertigo of the peripheral type with total hypofunction of one vestibular apparatus, demonstrated on caloric testing (Figure 7). While being compressed to a depth of 60 feet with ENG monitoring, he developed unsteadiness with a brisk nystagmus (Figure 8). Although he had no trouble in equalising his middle ear pressures...
either then or previously, the increased nystagmus now associated with this activity demonstrated a vestibular disturbance due to bilateral stimulation of unequal vestibular systems.

**Case 5**

This man noted left sided hearing loss when he exposed himself to excessive rifle fire. Tinnitus and partial deafness (e.g. to hearing his watch ticking) persisted from that time. Three months later he attempted diving, but noted a severe vertigo during each ascent. No problems were encountered with performance of the Valsalva manoeuvre. Otological investigation revealed a partial sensorineural loss in the left ear with a typical gunfire dip at the 4000-6000-8000 Hz (Figure 9). ENG performed during descent to a depth of 60 feet showed no abnormality, but during the ascent the subject experienced vertigo and showed a typical ENG pattern of nystagmus during this time (Figure 10). Both the clinical and ENG manifestations were diminished if he assumed the supine position during the ascent (Edmonds, 1971). This case demonstrated vertigo due to expansion of gas in the middle ear cavity, causing a stimulus to the vestibular system on both sides, but with an unequal vestibular response. The anatomical implications of the required positioning were not well understood.

If there is some minor inequality of the vestibular response on each side, then the stimuli associated with diving, especially the caloric stimulus, would be expected to cause vertigo in these divers. In practice, the commonest presentation of vertigo is in those divers who have obtained a set depth and proceed to swim in a horizontal manner (but often with the feet elevated, due to buoyancy effects of equipment and fins). This vertigo is especially noted in the 5-15 minutes period, and is reproducible when the subject attempts a similar swim under similar conditions.

In many cases the investigation of vertigo may require not only a typical otological investigation including audiometry and vestibular function assessments, but also an appreciation of the environmental conditions to which the divers are subjected. Such factors as diminished visual cues are particularly common in certain dives e.g. night diving in muddy water or with a high degree of phosphorescence. Under these conditions it is presumed that any tendency to vertigo would be augmented, although still some basic inequality must exist, either in the stimulus to each vestibular system, or the response.

... /There
There is therefore a requirement to assess the vestibular function of every diver presenting with any evidence of vertigo or disorientation underwater. The performance of ENG gives some objective evidence of this disorder, especially when combined with positional testing, caloric stimuli, compression and decompression exposures, and occasionally the alteration of gas pressures. Although not available in this unit, the presence of a Barani chair, optokinetic drum etc., may be of value in evaluating certain cases. The subjects with correctable lesions can be assessed and tested prior to the resumption of diving; whereas those divers with demonstrable vestibular damage, or recurrent vestibular disorders precipitated by positional, caloric, gaseous or barotrauma stimulation, should be excluded from further diving. The use of the ENG as a research tool in hyperbaric and aquatic states has yet to be fully explored.

5. **EUSTACHIAN TUBE FUNCTION**

Although a crude assessment of the patency of the eustachian tubes can be obtained by otoscopic examination during the performance of the Valsalva manoeuvre, such an examination is of little quantitative value. There is a requirement for an instrument which will measure the pressure changes required before the tubes become patent, both during ascent and descent. Although there is no such equipment available at this unit, the use of the sonomanometer appears to be of value in assessing the quantitative measurements of the pressures required to inflate the middle ear i.e. analogous to the descent conditions of diving. This equipment, presuming there are few technical difficulties, could be well adapted for use in diving medical units. It could well be of value in predicting those subjects likely to suffer from middle ear barotrauma of descent, and in assessing the value of techniques and drugs used in assisting this equalisation of the middle ear pressure. Evaluation of this equipment is anticipated in the near future.

6. **EQUIPMENT**

In the investigation of otological disorders of diving, the routine otological examination tray is required. Rarely such techniques as catheterisation of the eustachian tube, politzerization and use of the Segal speculum may be required.
A wide range audiometer (250-8000 Hz) is essential, as are the conditions required for its use e.g. a trained technician, soundproof booth, bone conduction, masking, SISI and recruitment capability. At the RAN School of Underwater Medicine, the equipment used is a Kamplex DA11 audiometer.

An electronystagmogram with facilities for accurate positional testing, caloric stimulation of the Hallpike type, and the ability to monitor continuously the ENG during compression and decompression of the patient. At the RAN School of Underwater Medicine the equipment used is a Philips 8 channel Cardiopan recorder for multiple recordings and a Watson Victor Cardiotrace series 3050 ENG Recorder for single recordings. For those diving medical units who have only an electrocardiogram attachment, crude tracings can be obtained by using standard lead I (left arm goes to left outer canthus, right arm to right outer canthus, and the ground lead to the central forehead) with maximal sensitivity, however a preamplifier stage with a 20 db gain ahead of the ECG input would enable standard ECG instruments to produce tracings comparable to commercial electronystagmograms. The patient must be able to be positioned within the chamber so that he can assume whatever position is related to the production of the clinical vertigo while diving. If caloric tests are attempted under hyperbaric conditions, the mechanics of the situation and the difficulty in maintaining constant temperatures of water, make the use of the iced water calories much more practicable. In these cases 1cc of iced water per second, for 20 seconds, is irrigated into each ear.

The sonomanometer will probably be of considerable value in giving a quantitative assessment of the patency of eustachian tubes, at least during descent. Reference has been made to this equipment in La Physiologie de la Trompe e'Eustache, by R. Rui et al, 1966.

The value of such equipment as the acoustic impedance meter, operating microscope etc., will rely on the presence within the diving medicine establishment of highly qualified otologists. Surgical repair of some of the more serious otological disorders of diving is still within the experimental field. It is likely to be of considerable value in the future management of these accidents.

.../7. CONCLUSION
7. CONCLUSION

This report discusses some isolated aspects of investigations into otological disorders of diving. It combines within the one paper, information on recent investigations into both hearing and vestibular damage, and discusses the possible methods of demonstrating these abnormalities. The basic requirements considered necessary for the initial data, periodical measurements and investigation of otological disorders of diving, includes:

1. Full clinical ENT examination.
2. Audiometer with 250-8000 Hz range, bone conduction, masking, SISI and recruitment capability.
3. Electronystagmogram, with caloric, positioning and the compression/decompression stimulation, as described in this article.
4. Sonomanometer.
5. Specialists in otology, who also have a practical appreciation of diving conditions.
8. REFERENCES


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