Inner ear barotrauma: a retrospective clinical series of 50 cases

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Key words
Inner ear barotrauma, diving, ear barotrauma, treatment

Abstract

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A retrospective series of 50 cases of inner ear barotrauma is reviewed, as regards the diving and otological history, the clinical manifestations, basic audiometric investigations and treatments. This disorder may be predicted to some degree by a previous history of otological barotraumas, otological pathology and the use of inappropriate middle ear auto-inflation and diving techniques. The absence of tympanic membrane haemorrhage does not exclude the diagnosis, nor does a delay between the dive and the developing symptoms. The dive profile, symptomatology and investigations assist in verifying the site of the lesion and the likely damage, as well as differentiating inner ear barotrauma from various decompression sickness manifestations, including inner ear, cerebellar and cerebral. Treatment procedures and indications are reviewed.

Introduction

It is now 30 years since the term inner ear barotrauma (IEBt) was introduced and the first series described.[1,2] In the same papers, the pathology of round-window fistula (RWF) was verified surgically, and other pathologies considered.

The main reason for the initial series being accepted, against the prevailing belief that barotrauma did not cause permanent hearing loss, was the presence of pre- and post-incident pure tone audiometry (PTA).[3] This investigation was obligatory in the compulsory diving medical examination in Australia.

Similarly, the first group of cases with vestibular pathology from IEBt was presented and verified by the application of the electronystagmogram (ENG).[4] This not only allowed for the localisation of the lesion to a peripheral site in the eighth cranial nerve, as opposed to cerebral and cerebellar disease, but also indicated the degree of pathology.

This present, much larger, series was extracted in order to review and reassess the clinical observations and basic investigatory results with IEBt. This allows for a comparison with the major dysbaric differential diagnosis, inner ear decompression sickness (IEDCS).

Methodology

Fifty cases were analysed. The first 25 consecutive cases were extracted from the 1970s, and the last 25 consecutive cases from the 1990s. To ensure there was no likelihood of diagnostic confusion, the cases had to meet the following criteria:

- have occured in association with air diving (free or scuba) and result in enduring inner ear pathology, verified by PTA and/or ENG;
- have been exposed to single, shallow dives (usually less than 10 m) or with depth/duration profiles of less than half the US Navy no-decompression limits;
- and/or have a demonstrated RWF at surgery.

All were referred for assessment because of the injury to the Diving Medical Centre and/or the School of Underwater Medicine of the Royal Australian Navy. This was, therefore, a selected series.

Each case was documented using a pre-designed 'Ear Barotrauma Protocol' with a checklist for specific features of the history, clinical examination and investigations. Many were sent for further otological consultation by the same clinicians responsible for the original seminal papers on this subject.

To be designated as having a hearing loss, divers had to experience a decrement of at least a 20 dB in at least two frequencies, compared with their previous audiogram. Most losses observed were far in excess of this.

Treatment of sensorineural hearing loss was designated as successful if the audiogram returned to within 15 dB of the pre-incident audiogram. Partial success represented improvement equal to or greater than 20 dB, compared with the post-incident audiograms.

During the time period covering these diving cases, there were two other cases of IEBt from aviation exposure, which were not included. Another case, which did actually comply with the criteria, was excluded when the predominately conductive deafness was diagnosed as otosclerosis and successfully treated with surgery. This patient may also have experienced some IEBt.

Results

DEMOGRAPHY

These cases represented just under 1% of the diving problems encountered at the Diving Medical Centre. If one included all divers presenting with possible IEBt, but not meeting the above strict criteria, the prevalence was well over 1%.

Twenty six (52%) of the divers in this series were moderately experienced, having performed 6-50 dives. Thirteen (26%) were inexperienced or under training (1-5 dives) and 11 (22%) were very experienced (>50 dives). Although all were scuba divers, the incident causing injury occurred whilst free diving in five (10%). The female to male ratio was 3:7.

Presentation to a physician usually occurred in the first week (43 cases, 86%), with the remainder over the next two to three weeks. Medications prescribed included decongestants in four cases (8%) and aspirin in three (6%).

As regards hearing loss, the right side was affected in 20 (40%), the left side in 17 (34%), and three (6%) cases were bilateral.

PREVIOUS OTORHINOLOGICAL PATHOLOGY

Chronic otorhinological (ENT) pathology was present in 20 cases (40%), comprising allergic rhinitis, chronic sinusitis, previous surgery or other ear pathology, in order of decreasing frequency. This pathology was usually of a minor degree. There was a history of acute ENT pathology in 8% including infections (pharyngitis, otitis media, sinusitis). There was a past history of:

- recurring middle ear barotrauma (MEBt) of descent in 28 (56%);
- one episode of MEBt of descent in three (6%);
- MEBt of descent and ascent in three (6%);
- aviation MEBt in 12 (24%);
- IEBt in six (12%).

In performing middle ear equalisation (ME=) techniques, not one employed the recommended 'equalising ahead of the descent' technique, and only three (6%) used the feet first descent, as has been advocated.[5]

DIVE PROFILE

Although many initially claimed to have had no trouble with ME=, specific interrogation revealed that they had been obliged to undergo a slow descent (22 cases, 44%), or a 'yo-yo' (multiple interruptions of descents by short ascents) descent (22 cases, 44%) because of sluggish ME=. A few developed symptoms associated with uncontrolled rapid ascents or descents. Most of those who initially claimed no ME= problems, apart from the indicative dive profile, also had both symptoms and signs of MEBt.

CLINICAL FEATURES AND INVESTIGATIONS

In total, after more complete interrogation than simply asking if there was any problem with ME=, there was a clinical history consistent with MEBt of descent in 44 cases (88%) and five had MEBt of both descent and ascent.

Some symptoms were attributable to this associated MEBt, such as a blocking, fullness or crackling sensation in the ear (37 cases, 74%), persisting pain after the dive (11 cases, 22%) or slight epistaxis from the side of the MEBt (6 cases, 12%).

Grading of MEBt by otoscopy is traditional in diving medicine.[6] Physical signs of MEBt were not always evident by the time the subject was first examined by a diving physician, with 19 cases (38%) being Teed Grade 0. Grade 1 was evident in two (4%), Grade 2 in eight (16%), Grade 3 in 10 (20%) and Grade 4 in three (6%). The remaining eight showed some degree of haemorrhage, which was not graded at the time.

Many of those with a history of MEBt had a flattened tympanometry and sometimes a mild conductive deafness.

Inner ear symptoms attributable to cochlear damage included hearing loss (40 cases, 80%), tinnitus (43, 86%) and dysacusis (10%). The hearing loss was always confirmed as sensorineural, either high frequency at 4 kHz, 6 kHz and 8 kHz, or virtually total, involving the lower frequencies also. Four divers (8%) did not recognise their hearing loss, despite a definite high-frequency sensorineural deafness.

Vestibular symptoms occurred in 16 cases (38%) and always included vertigo. Sometimes these symptoms were mild, lasting only a day or so, but frequently they were severe, with nausea, vomiting, prostration and ataxia. Lying horizontal or sudden head movements caused aggravation in some. In four cases (8%) there was ENG evidence of pathology, despite there being no symptomatology attributed to the vestibular system.

Inner ear symptoms were noted either during the dive or immediately on ascent in most cases (31, 62%), although in 16 (32%) they developed over the next few hours and in three (6%) they developed during the night following the dive. Sometimes the vestibular symptoms were noted after the cochlear, usually when the diver tried to board the boat. In four cases (8%) the symptoms followed exertion (lifting the anchor or scuba tanks) or other causes of raised intracranial pressure (Valsalva, sneezing).

TREATMENT

Conservative treatment was administered in 33 cases (66%) with partial success evident in 21 (42%) and cure in three (6%). With conservative management, symptoms other than hearing loss were too variable to be accurately quantified.

Tinnitus tended to improve over hours, days, weeks or months, with or without treatment. Vestibular symptoms consistently improved in all cases over weeks or months. Often the only evidence of persisting vestibular pathology was from provocative ENGs.

Surgery was performed in 13 divers (26%), with nine (18%) showing improvement in hearing, two (4%) being cured and one worsened, at the time of discharge. Much more impressive was the often dramatic improvement in tinnitus and vestibular symptomatology, from the time of the operation. The longest time interval between incident and surgery was 10 weeks; surgery was performed because of the diagnosis of persistent RWF, and had excellent, immediate results.

Recompression therapy had been inexplicably undertaken in three cases, but did no evident damage, prior to more appropriate referral for surgical treatment. Drug therapy had previously been used in three cases (aspirin, nicotinic acid), without effect. Decongestants and anxiolytics were sometimes employed during symptomatic and conservative treatment. Because of the time delay, the remainder received no therapy.

Discussion

Clinical descriptions of IEBt are not available in the diving medical literature before 1972, as the disorder was not recognised until then. Thus much of the literature quoted before that time did not discriminate between decompression sickness (DCS), IEBt and non-diving aetiologies such as noise, gunfire and explosions. Thus, using this literature to support any position on the topic of inner ear problems from diving is somewhat misleading.

Since then there have been case reports and clinical series of diving otological disorders reported and reviewed, but still with some confusion regarding the manifestations of MEBt, IEBt, cerebral DCS and/or IEDCS.[6-12] The lack of specificity of diagnoses sometimes causes difficulty in extrapolating the clinical features, and differential diagnoses were based on assumptions that have not been tested.

For this reason, 50 cases of evident IEBt, uncomplicated by a decompression sickness likelihood, were extracted from the files of the Diving Medical Centre, and retrospectively assessed. Differentiation from MEBt was verified by PTA and ENG.

The strong association with MEBt was verified. Also, the frequent past history of MEBt, descent, ascent and/or aviation induced, was observed. Two aspects of MEBt should not be used to exclude the diagnosis of IEBt. The initial claim of 'no ME= problem', either in clinical notes or from the diver directly, needs to be carefully explored. It is often a euphemism used by divers, who are renowned for the use of denial as a psychological mechanism, to indicate that ME= problems were overcome successfully and did not require aborting the dive.
Unless the examiner has also specifically questioned the diver about the reasons for:

- slow descent;
- employing 'yo/yo' techniques;
- 'fullness' sensations in the ear following a dive;
- epistaxis,

it is likely that MEBt will be missed. If the diver also employs less effective ME= techniques, such as head-first descent, or waiting until middle ear pressure is felt before employing ME=, the likelihood of MEBt becomes greater.

The absence of tympanic membrane haemorrhages does not exclude MEBt and/or IEBt. It could be explained as follows:

- Haemorrhage into the tympanic membrane is not inevitable in MEBt if mucosal effusion in the middle ear or a perilymph leak dominates the pathology and contributes to ME=. This is recognised by the inclusion of Grade 0 (symptoms without membrane tympanic signs) in the Teed classification of MEBt, and haemorrhage would be less expected in very slow descents.
- MEBt manifestations often resolve within days, before otoscopy is performed by the physician.
- The most widely accepted pathophysiology of IEBt is the explosive effect of the raised intracranial pressure associated with the Valsalva manoeuvre.[11,13,14] It is the latter that is likely to be directly related to IEBt, not the MEBt per se.
- Most general otological cases of RWF have nothing to do with dysbaric barotrauma, but are related to sudden raised intracranial pressures in non-divers.

Thus, the failure to observe tympanic membrane pathology should not be used to exclude RWF.

Although the development of symptoms during the dive may be indicative of a diagnosis of IEBt, a delay between the dive and the appearance of symptoms does not exclude this diagnosis. Indeed, their development associated with après-dive activities may well be suggestive of membrane rupture as opposed to other pathologies of IEBt, such as haemorrhage.

Symptomatology of IEBt is easily confused with MEBt (which frequently co-exists), IEDCS and neurological (cerebral and cerebellar) DCS, if appropriate investigations are not performed. Sensorineural deafness, partial or total, is usually associated with tinnitus and occasionally with dysacusis. PTA, up to 8 kHz, is essential for accurate assessment, and a pre-incident PTA is immensely valuable.

ENG clarifies the existence of vestibular pathology, and its severity, as well as differentiating peripheral from central (cerebellar) lesions. It brings objectivity to the symptoms of dizziness and vertigo, and the assessment of nystagmus. For more detail on the use, versatility and value of ENG, conventional diving medical texts should be accessed.[6]

Although vomiting and ataxia are dramatic, they indicate only the acuteness and severity of the vertigo, not necessarily its origin.

Initially the tendency was to treat all cases with surgery, but it soon became evident that conservative treatment was often very effective. This included bed rest with head elevated, avoidance of all intracranial pressure increases (Valsalva manoeuvres, sneezing, nose blowing, straining at defaecation, lifting and other exertion). Repeated PTA is used to monitor progress.

Massive hearing loss or deteriorating hearing despite conservative treatment, are indications for middle ear exploration with plugging of labyrinthine windows even if the fistula cannot be visualised. Successful surgery often has immediate value in relieving symptoms. The 1970s contributed nine (18%) of the surgical cases, with the 1990s producing four (8%). Although verifying the observed trend, this is not statistically significant.

Our experience confirms that permanent inner ear damage predisposes to further damage if the provoking activity (diving) were to continue.[11] The high incidence of both MEBt and IEBt in the past medical history supports this belief. We thus advise IEBt patients with evidence of inner ear damage that not only should they discontinue scuba diving, but they should also avoid any hyperbaric exposure, such as with free diving. They are also advised regarding ideal ME= with aviation exposure.

Conclusions

1 IEBt may be predicted to some degree by a previous history of MEBt or previous IEBt, and is more likely in divers with ENT pathology and who use inappropriate ME= diving techniques.

2 The history of ME= needs to be carefully assessed, to be compared with the dive profile, and the absence of tympanic membrane hemorrhaging does not exclude the diagnosis.

3 The development of symptoms during the dive may be indicative of IEBt diagnosis, but a delay between the dive and the appearance of symptoms does not exclude this diagnosis.

4 The cause of the pathology (IEBt, IEDCS, neurological DCS) can be indicated by the dive profile and the presence of other symptoms of MEBt, IEBt or DCS. PTA and ENG are sometimes necessary to complement the clinical findings and verify the site and extent of the lesion.

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Australian standards committees

Dr David Smart was the successful applicant to replace Dr John Knight as the SPUMS representative on the Standards Australia SF-017 Occupational Diving Committee.

Dr Cathy Meehan is the current SPUMS representative on the Standards Australia CS-83 Recreational Underwater Diving Committee.