Review article

The assessment and management of inner ear barotrauma in divers and recommendations for returning to diving

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Abstract

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Inner ear barotrauma (IEBt) constitutes a spectrum of pressure-related pathology in the inner ear, with antecedent middle ear barotrauma (MEBt) common. IEBt includes perilymph fistula, intralabyrinthine membrane tear, inner ear haemorrhage and other rarer pathologies. Following a literature search, the pathophysiology, diagnosis, and treatment of IEBt in divers and best-practice recommendations for returning to diving were reviewed. Sixty-nine papers/texts were identified and 54 accessed. Twenty-five case series (majority surgical) provided guidance on diagnostic pathways; nine solely reported divers. IEBt in divers may be difficult to distinguish from inner ear decompression sickness (IEDCS), and requires dive-risk stratification and careful interrogation regarding diving-related ear events, clinical assessment, pure tone audiometry, a fistula test and electronystagmography (ENG). Once diagnosed, conservative management is the recommended first line therapy for IEBt. Recompression does not appear to cause harm if the diagnosis (IEBt vs IEDCS) is doubtful (limited case data). Exploratory surgery is indicated for severe or persisting vestibular symptoms or hearing loss, deterioration of symptoms, or lack of improvement over 10 days indicating significant pathology. Steroids are used, but without high-level evidence. It may be possible for divers to return to subaquatic activity after stakeholder risk acceptance and informed consent, provided: (1) sensorineural hearing loss is stable and not severe; (2) there is no vestibular involvement (via ENG); (3) high-resolution computed tomography has excluded anatomical predilection to IEBt and (4) education on equalising techniques is provided. There is a need for a prospective data registry and controlled trials to better evaluate diagnostic and treatment algorithms.

Key words

Inner ear, barotrauma, diving, pathophysiology, treatment, ENT, review article

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Introduction

Inner ear barotrauma (IEBt; see Table 1 for a range of acronyms related to this topic and used in this article) encompasses a spectrum of pathology in the inner ear resulting from pressure injury. This includes perilymph fistula (PLF), intra-labyrinthine membrane tear, inner ear haemorrhage, and other rarer pathologies (Figure 1). PLF is a subset of IEBt where an inappropriate communication occurs between the perilymph fluid in the inner ear and the middle ear via the labyrinthine structures of the round and oval windows.[1–4] A communication may also occur within the semicircular canals, vestibule, or cochlea via the intra-labyrinthine structures of the Reissner's, basilar and tectorial membranes, resulting in mixing of the endolymph and perilymph.[3,5–7]

Anatomy

The oval window receives sound vibrations directly from the stapes footplate, converting them into waves which travel through perilymph to the organ of Corti for sound detection. The round window is also located in the vestibule of the membranous labyrinth, inferior to the oval window. Its function is to compensate for the changes in pressure in the unyielding fluid by stretching.[8] If a deficit involves either labyrinthine window, perilymph fluid leak occurs from the semicircular canals and/or cochlea into the middle ear (see <http://www.dizziness-and-balance.com/disorders/unilat/fistula.html>).[2,7–10]

Reissner's and the basilar membranes are located in the perilymphatic space between the internal structures of the cochlea, separating the three scala layers. Reissner's membrane divides the scala vestibuli and scala media; the basilar membrane separates the scala media and scala tympani and the tectorial membrane sits in the scala media.[11,12] The fluid in the scala vestibule and tympani is perilymph, with endolymph contained in the scala media above the organ of Corti (Figure 2).[11,12] Each has a significantly different ionic composition. All membranes are responsible for propagation of sound waves via the incompressible inner ear fluid.

Diving-related barotrauma, hearing loss and IEBt

Aural barotrauma has been identified as the most common cause of long-term morbidity in divers, experienced by 52% in a sample of 709 experienced recreational divers.[13] It is estimated that 0.5–1.1% of divers will suffer IEBt in their lifetime.[13,14] In the absence of ear injury or noise exposure, diving per se may not cause hearing loss. No significant difference was found in pure tone audiometry (PTA) of 60 sport divers compared with controls; neither

Figure 1
Types of inner ear barotrauma

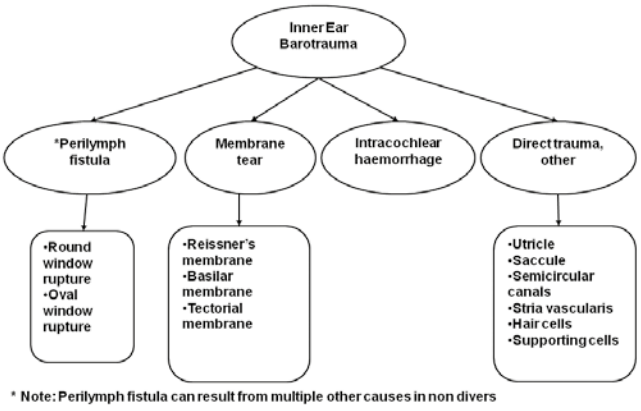

Figure 2
Cross section of the cochlea, illustrating the Organ of Corti <http://en.wikipedia.org/wiki/File:Cochlea-crosssection.svg>[12]
(reproduced with permission of *Wikipedia*)

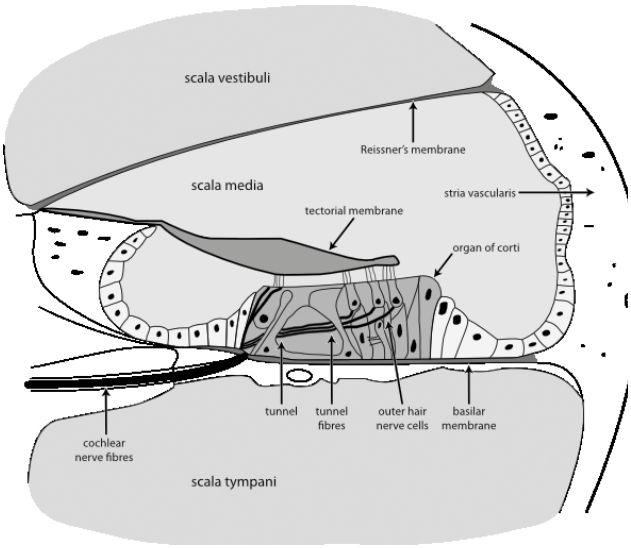

Figure 3
Dissection of the inner ear <http://www.britannica.com/EBchecked/topic/175622/human-ear/65040/Cochlea>[32]
(reproduced with permission of *Britannica*)

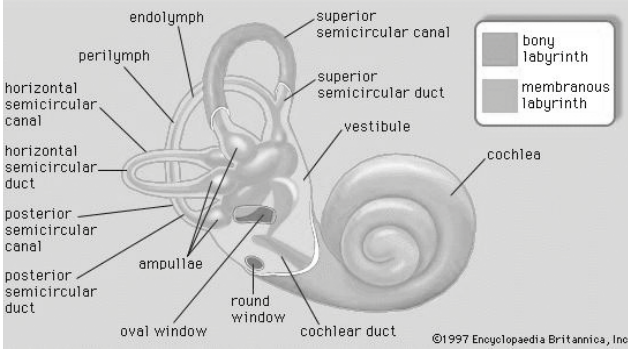

Table 1
Acronyms for otorhinolaryngological and other terms relevant to inner ear barotrauma used in this article

Acronym	Medical term
ABR	Auditory brainstem response
BPPV	Benign paroxysmal positional vertigo
CCG	Craniocorpography test
CHL	Conductive hearing loss
ENG	Electronystagmography
ENT	Ear, nose and throat
HRCT	High-resolution computed tomography
IEBt	Inner ear barotrauma
IEDCS	Inner ear decompression sickness
MEBt	Middle ear barotrauma
MRI	Magnetic resonance imaging
OAE	Otoacoustic emission
OWR	Oval window rupture
PLF	Perilymph fistula
PTA	Pure tone audiometry
RWR	Round window rupture
SCD	Superior canal dehiscence
SHA	Smooth harmonic acceleration
SNHL	Sensorineural hearing loss
TM	Tympanic membrane
URTI	Upper respiratory tract infection
VEMP	Vestibular evoked myogenic potential

group had a past history of significant noise exposure or IEBt.[15] Another study comparing occupational divers with offshore workers also did not detect a higher incidence of hearing loss in divers.[16] In 64 entry-level occupational divers studied over six years, the only detected change in PTA was secondary to noise exposure. None suffered IEBt (or inner ear decompression sickness, IEDCS), although middle ear barotrauma (MEBt) was common.[17,18] One-hundred-and-twenty Navy divers had greater hearing loss than 116 non-diver controls.[19] Although cases were not split by cause of hearing loss (IEBt, IEDCS or industrial hearing loss), this study suggested that continuing to dive with known hearing problems may exacerbate hearing loss over time.[19] Any study of aural barotrauma or hearing loss in divers presents challenges – the spectrum and severity of injuries is broad. In addition, detection of injury may be retrospective via hearing and balance deficits, with no clear traumatic event recognised by the diver.

Hence, the true incidence of IEBt is difficult to calculate owing to minor symptoms not precipitating medical review, spontaneous healing and diagnostic uncertainty. Following either conservative or surgical treatment options, the diving specialist faces the challenge of assessing fitness to return to diving. There appears no current consensus to guide occupational divers returning to employment following IEBt, and this review was unable to identify guidance from learned societies, such as the South Pacific Underwater Medicine Society (SPUMS), the Undersea Hyperbaric Medical Society (UHMS) or the European Underwater and Baromedical Society (EUBS).[7,12,20–26]

Our aim was to review the literature on the pathophysiology, diagnosis and treatment of IEBt in divers and to develop best-practice recommendations for diagnosis, treatment and returning to diving.

Literature search

A literature search from January 1972 to December 2012 was conducted using search terms 'inner ear barotrauma', 'perilymph fistula', 'round window rupture', 'oval window rupture', 'diving', 'SCUBA', 'diagnosis' and 'treatment'. The search was initially restricted to human studies only. Search engines employed included Ovid, Medline (PubMed), CINAHL, EMBASE and Google Scholar. In addition, a hand search was conducted of texts in diving medicine that covered IEBt, as well as proceedings and workshops from SPUMS, UHMS, EUBS and the International Congress on Hyperbaric Medicine. Finally a search was undertaken of references identified in papers located by the initial search providing access to other relevant articles.

SEARCH RESULTS

Sixty nine papers/texts were identified, with 54 papers/chapters accessed. One paper was rejected as it was unable to be translated.[27] Six other papers were included for essential pathophysiological or anatomical information and the Australian/New Zealand standards.[28–31] Six webpages were accessed for supportive information and images.[2,8,11,12,32,33] Four key animal studies (dogs, cats, rabbits and guinea pigs) were included describing the historical development of pathophysiological understanding of IEBt.[34–37]

Pathophysiology of IEBt in divers

Understanding pressure physiology is essential for an understanding of IEBt. Pressure increases by one atmosphere (101.3 kPa) for every 10 metres' sea water depth (msw). According to Boyles Law, during descent to 10 msw, an air space halves in volume, unless equalised by additional gas. The greatest rate of change in volume occurs in shallow water, hence divers may experience significant barotrauma even at shallow depths.[14,23,24]

From historical data on intraoperative assessments or forensic findings, three injury patterns were identified from IEBt in isolation or combination in scuba divers:
- fistula of the round or oval window;
- intralabyrinthine membrane tear;
- inner ear haemorrhage.[23]

PERILYMPH FISTULAS IN DIVERS

A key factor in diving-related IEBt is the direct link between the intracranial cerebrospinal fluid (CSF) and the perilymph. IEBt results from unbalanced pressure changes between the structures of the inner ear and middle ear and/or CSF. The relationship between changes in pressure from the intracranial space to the perilymph was first identified in rabbits in 1879, and further clarified in the mid-twentieth century.[35–38] An association was demonstrated between thoracic and intra-abdominal pressures, occlusion of neck blood vessels and CSF pressure.[38] Citing many early animal studies, it was concluded that changes in perilymph pressures correlated with intracranial pressure (ICP), and occlusion in the cochlear aqueduct inhibited this reflex.[39] However, it was not until the 1960s that the link between raised CSF pressure, perilymph pressure and PLF was observed in humans.[39] This coincided with the advent of stapes surgery. IEBt was first confirmed in divers in 1972 using pre- and post-incident audiograms.[40]

IEBt in diving may result from 'explosive' and/or 'implosive' forces on the vestibulocochlear apparatus due to pressure differences caused by a blocked Eustachian tube.[3,6,7,26,41–44] Explosive barotrauma is transmitted through the CSF to the perilymph of the cochlear aqueduct, vestibular aqueduct, and scala tympani.[6,43,45,46] The final endpoint of this pressure wave is the round or oval window. Implosive forces act from external sources upon the inner ear. Middle ear pressure changes may be transmitted to the labyrinthine windows, causing rupture and vestibulocochlear injury. Round window rupture (RWR) appears to be associated with barotrauma, whereas oval window rupture (OWR) is more commonly due to external forces to the head or auditory apparatus.[43]

Diving-related IEBt is thought to be due to pressure gradients on the tympanic membrane (TM) and middle ear during descent. From case histories, the main antecedent cause appears to be inability to pressurise the middle ear.[10,21] When the pressure differential between the middle ear and nasopharynx is > 90 mmHg (12 kPa), the Eustachian tube closes and 'locks', resulting in an inability to equalise the middle ear.[10,46] The TM bulges inwards, pushing the stapes into the oval window, displacing perilymph towards the round window, pushing it outwards into the middle ear cavity.[3,7,43] As perilymph and endolymph are incompressible fluids, pressure changes are transmitted to the weaker labyrinthine windows (Figure 3).[7,32,43] If a sudden pressure wave is transmitted to the perilymph, a RWR may result with leakage of fluid into the middle ear or an influx of air into the perilymph.[3,20,47] The above mechanism is a combination of implosive force (on the stapes and oval window), and explosive force due to the Valsalva manoeuvre. The process is augmented by a pre-existing negative pressure in the middle ear. This causes a sensorineural hearing loss (SNHL) and vestibular symptoms which may be progressive or fluctuating owing to leakage of perilymph.[3]

Excess pressure in the perilymph resulting from other processes, such as coughing, sneezing, straining or lifting, may also cause a RWR, OWR or tear of the basilar or Reissner's membranes.[3,43,48,49] Stresses are further amplified by external negative pressure (e.g., when removing a diving

hood).[1,6,44] Non-diving case series frequently include such mechanisms of injury. Most literature focuses on RWR and OWR which are subsets of IEBt. Both can occur as single pathologies, combined or occasionally bilateral. In the setting of diving barotrauma, RWR almost exclusively occurs and OWR is very rare.[21,25,48,50]

MEMBRANE TEARS

Other possible injuries from IEBt are tears and/or haemorrhage of the Reissner's, basilar or tectorial membranes.[7,23] These are termed intralabyrinthine or intracochlear tears and may be associated with MEBt.[20,21,23,26] It has been postulated that intracochlear ruptures are responsible for SNHL with additional interruption to the striae vascularis, hair cells and ancillary cells.[10] Injury of Reissner's membrane (also known as the vestibular window) may cause SNHL through a direct effect on the cells of hearing in the organ of Corti, or through perilymph/endolymph mixing, which changes the ion concentrations around the structures of hearing.[7,23,39,45] Basilar membrane damage can result in long-term SNHL by compromising the organ of Corti.[7] Membrane tears can be diagnosed clinically on PTA by a lingering SNHL isolated to the frequency that corresponds to the anatomical location of the membrane tear.[23,26] Membrane tears may also result in vertigo and nausea.[7] Symptoms do not appear pathognomonic for a specific membrane injury.[11] There also exists a 'double membrane break theory' that proposes a RWR or OWR may be combined with an intracochlear (i.e., Reissner's, basilar, tectorial) membrane tear.[6,39]

INNER-EAR HAEMORRHAGE AND GAS

With IEBt, there also may be direct physical damage to the hair cells or their blood supply caused by expanding gas in the inner ear, in particular the scala tympani and scala vestibuli from air forced from the middle ear through a PLF (pneumolabyrinth).[7,10,44,47] Clinical presentation is similar to RWR and OWR, with SNHL of varying severity and/or transient vestibular symptoms.[23,26] Inner ear haemorrhage tends to have a more sudden onset.[3] Isolated hearing deficits appear to be predominantly due to intracochlear membrane pathology and associated structures of hearing rather than RWR or OWR.[39]

Predisposing factors

Divers suffering IEBt may report difficulty in equalising on descent or ascent, or application of an internal or external force on the ear while diving or immediately post dive (e.g., forceful Valsalva, wave trauma, removing a wetsuit or hood, or lifting heavy equipment).[3,16,21,22,25,40,51] In a retrospective review of 50 Australian divers sustaining IEBt, 24 had acute or chronic ear, nose and throat (ENT) pathology.[21] This suggests that IEBt in divers results from problems in equalising, supported by a New Zealand case series in which a history of difficulty in ear clearing and/or respiratory infection was identified in three-quarters of the divers.[25]

Some individuals appear prone to recurrent PLF, suggesting anatomical predisposition; one study identified recurrent IEBt in two of 44 divers.[16] Post-mortem histological examination of temporal bones has demonstrated an association between PLF and enlarged vestibular and/or cochlear aqueducts, permitting greater CSF-perilymph communication.[6,45,52] These anomalies may be detected on high-resolution computed tomography (HRCT).[16] This contrasts with the proposed hypothesis that a small cochlear or vestibular aqueduct could make an ear more prone to implosive or internal pressure changes.[39]

Aplasia or malformations of the cochlea, and weakness or malformation of the bony structures, otic capsule, or inner ear membranes may also predispose to IEBt.[45,52,53] Other congenital causes of IEBt (OWR) include weakness of the annular ligament of the stapes, and abnormalities of the stapes bone.[6,23,39] Anatomical abnormalities of the windows may also pose a risk for IEBt.[39] None of the aforementioned factors would be detected during routine physical examination. In a non-diving series of 44 cases of PLF, half had no clear precipitating cause, suggesting possible spontaneous PLF in some individuals.[51]

IEBt is a rare diving injury; hence screening for congenital risk factors is not appropriate at the time of health screening. There is merit in identifying divers who have difficulty with ear clearing or greater risk of MEBt, because the evidence is that it increases the risk of IEBt.

Diagnosis of IEBt in divers

ONSET OF SYMPTOMS

The timing of symptom onset in relation to a diving event is a key element in identifying IEBt. Symptom onset (vestibular dysfunction or hearing impairment) after difficulty equalising on descent is a consistent finding in many IEBt case series.[14,21,23,24,26,40] IEBt may also become manifest during ascent; secondary to pneumolabyrinth and gas expansion, injuring the cochlea or vestibular apparatus.[12] The symptoms of IEBt may even occur some days post dive, particularly if provoked by lifting or straining.[1,54]

Unfortunately symptom onset may be vague, particularly if SNHL is not accompanied by vestibular symptoms. IEBt has a spectrum of severity, depending on the anatomical site of insult and degree of injury. Tears in Reissner's or the basilar membrane (within the cochlea) without RWR may present as isolated SNHL. Some resolve spontaneously within days.[6] These cases are thought to be under-reported because of their less dramatic presentation and self-resolution. The existence of subclinical IEBt has been suggested, which may be detected as minor degrees of SNHL in divers measured by

otoacoustic emission (OAE) testing.[54] However, this study was limited by the absence of a non-diving control group.[54]

The major differential diagnosis of IEBt is IEDCS. The two conditions pose a diagnostic dilemma, sharing similar clinical presentations: nausea, vertigo, nystagmus, SNHL, and tinnitus.[14,21,46,55] The original descriptions of five divers with IEBt described SNHL occurring after very low-risk dives.[40] Dive risk stratification remains a significant part of clinical algorithms when assessing new vestibulocochlear symptoms in divers. A provocative dive profile, uncontrolled ascent or missed decompression stops, onset of symptoms after the dive, other manifestations of decompression illness (DCI) or mixed gas diving indicate a greater likelihood of IEDCS.

IEDCS commonly occurs in divers using gas mixtures other than air (e.g., heliox) during their dive, although IEDCS in air divers is being reported in increasing numbers.[20,31,46,56] Up to 48% of divers with IEDCS have other symptoms of DCI.[56] In contrast, low-risk, shallow dives, sudden onset of symptoms during ear clearing manoeuvres, a past history of ear barotrauma, symptom onset in relation to Valsalva/straining (during or post dive), and coexistent MEBt all suggest IEBt.[20,21,31,46,54–56] It is important to note however, that individuals with IEBt may have normal tympanic membranes.[21,46]

It has been suggested that recompression could theoretically exacerbate the symptoms of IEBt, whereas IEDCS symptoms should improve.[9,31,46] This assertion has not been confirmed. In the retrospective series of 50 divers, three were recompressed without incident.[21] Other studies also have found that recompression can be conducted on suspected PLFs without adverse effects, provided tympanostomy tubes are present.[3,55] The risks of tympanostomy in an already injured ear must be carefully weighed against any theoretical benefit. It has been hypothesised even that IEBt may be improved by recompression because gas that has entered the inner ear via a window rupture may be redistributed or removed during recompression.[31]

Other differential diagnoses include alternobaric vertigo, and non-diving medical causes. Alternobaric vertigo is due to differences in middle ear pressures between the right and left ears resulting in disequilibrium and vertigo which usually manifest during ascent.[57,58] MEBt was reported by 24 of 67 occupational divers, in whom transient dizziness and vertigo developed during (24 divers) or soon after (10 divers) working dives.[57] Symptoms were short-lived post-dive (hours only) and not associated with demonstrable hearing injury or long-term effects.[57] Persistent symptoms should raise the possibility of IEBt. Non-diving differential diagnoses include Meniere's disease, benign paroxysmal positional vertigo (BPPV), or vestibular neuritis.[18,51] Where symptom onset coincides with diving, diving pathology should be considered and/or treated before assigning a non-diving diagnosis.

CLINICAL PATTERN OF SYMPTOMS

Vestibular injury is characterised by dizziness, constant dysequilibrium, positional vertigo, positional nystagmus, imbalance, ataxia, nausea and possibly vomiting.[1,7,51] These symptoms typically worsen with activity and loud noise (the 'Tullio effect'), and are improved or relieved with rest.[51] Disequilibrium caused by IEBt may fluctuate, provoked by actions that increase ICP, e.g., sneezing or straining.[51] Vestibular symptoms neither rule in nor rule out PLF, but do appear amenable to surgical repair.[1,14,47]

Hearing deficits may be perceived as aural 'fullness', 'muffled' hearing, tinnitus, hyperacusis, or complete hearing loss.[1,24] Often, impaired speech discrimination is noted.[22,41,59] The severity of the symptoms correlates with the extent of the injury and is a prognostic indicator for recovery.[1] The pattern of SNHL loss demonstrated at PTA for IEBt may be variable. The critical issue is that it is recognised. A pre-incident audiogram assists to define the degree of injury.[21,40]

THE CHALLENGE OF PRECISE PATHO-PHYSIOLOGICAL DIAGNOSIS

Given that the exact injury from IEBt may be microscopic and deep within the inner ear, it is challenging for the clinician to identify the specific pathophysiology in any affected individual. Diagnoses of PLF secondary to RWR or OWR dominate case series, and originate from the surgical literature. The exact cause may only be discovered during tympanotomy or at autopsy, or occasionally by high-resolution imaging techniques. Case series describe membrane tears and haemorrhages as diagnoses of exclusion, based on clinical findings, or when PLF has been ruled out at tympanotomy.[23] It is clinically difficult to differentiate between trauma to the cochlea or vestibular apparatus, haemorrhage in the inner ear and PLF.[3,23]

Investigation of IEBt

When assessing the usefulness of investigation of IEBt, data are available from both diving and non-diving case series. Some mixed-aetiology series include only one or two divers, whilst most non-diving series originate from the surgical literature and focus on identifying PLF. A comprehensive table of all studies from our literature search is available from the authors. However, in Table 2, we document only those clinical series focusing solely on IEBt in divers.

Initial assessment of suspected IEBt should include a general head and neck examination, otoscopy, Rinne and Weber tests, cranial nerve testing and cerebellar testing, including sharpened Romberg and Hallpike manoeuvres. Where there is clinical suspicion of IEBt, numerous tests are available for otoneurological assessment (Table 3). Essential tests for IEBt include air and bone (if available) PTA to diagnose and quantify SNHL, and the fistula test to clinically assess

inducible vertigo. Positional PTA has been used to assist in the diagnosis of PLF in non-divers.[47] A positive test was defined as a hearing gain of at least 10 dB in two or more frequencies when the subject lay supine with the affected ear uppermost.[47]

AUDIOMETRY

Audiometry findings may be variable in IEBt. Non-divers with PLF present with all three patterns of audiometric abnormalities: ascending curves 22.5%, descending curves 40% and flat global curves in 37.5%.[60] Both global and descending curves have been reported in divers with IEBt.[40] SNHL involving the higher frequencies (descending curve) has been associated with RWR.[43] The traumatised area in the membranous labyrinth can be mapped using audiometry in increments of 100 Hz, which demonstrates isolated damage to the cochlea in a narrow frequency range.[6] Audiograms are also useful to monitor recovery from the injury.[41] Hearing loss appears more likely to improve if low to mid frequencies are affected (250–1,000 Hz).[3] This is useful functionally as it reflects the speech frequencies.

PTA is currently required prior to clearance for occupational (and recreational) diving medicals in Australia (AS/NZS 2299 Standards and AS4005.1).[61,62] This assists in the assessment of diving-related ear injuries by providing a reference baseline for comparison.[21,40] The Australian recreational industry recently abandoned AS 4005.1, which will result in a diagnostic challenge of IEBt in recreational divers as they will be less likely to possess reference audiograms in the future.

FISTULA TEST

The fistula test has been classically used to detect RWR or OWR. The test involves a variable pneumatic pressure applied to the external ear with the aim of inducing vestibular symptoms or nystagmus. A positive test led to exploration in a number of series; however, its limited sensitivity and specificity have prevented the test becoming a 'gold standard'.[4,41,51,60]

ELECTRONYSTAGMOGRAPHY AND COMPUTER TOMOGRAPHY

ENG has been used to diagnose and quantify vestibular injury.[4,6,21,22,41,49,51,52,57,63,64] ENG may be combined with a pneumatic test (fistula test, Valsalva), or positional changes to increase its sensitivity.[5]

HRCT of the temporal bones has been recommended for IEBt to identify individuals at risk of an enlarged CSF-perilymph communication via a widened cochlear aqueduct orifice and enlarged internal auditory canal.[10] Two of 44 divers with recurrent IEBt were found to have anatomical abnormalities, and HRCT was stated to assist assessment in returning to diving.[10] It was not recommended for initial diagnosis of IEBt, but may have potential in identifying pneumolabyrinth.[44]

A key conundrum is whether or not to undertake surgical exploration and repair of the subset of IEBt that is due to PLF. It is difficult to develop clear guidelines from the non-diving literature regarding investigation of PLF in divers. Many of the series are of mixed aetiology including external trauma cases in non-divers.[1,4,5,47,48,60,65] A number of series report the findings of tympanotomy, but only loosely report the selection criteria for performing the procedure.[1,4,41,47,48,51,53,60,65] Clinical algorithms included PTA; however, there was variable reporting of other investigation even in recent series.[1,48] Most of the series are retrospective, introducing selection bias.

SURGICAL EXPLORATION

In 26 of a series of 51 cases, a fistula was demonstrated intraoperatively.[4] No diagnostic tests confirming PLF preoperatively could be identified.[4] Using clinical criteria to diagnose barotraumatic PLF, combined with PTA, nine suspected cases were exposed surgically, with eight having PLF confirmed intraoperatively.[1]

IEBt vs. IEDCS

Two clinical series of divers have documented diving events that produced cochlear (hearing or tinnitus) and/or vestibular (dizziness, balance, vertigo) symptoms, where there was a low risk of DCI.[21,14] In one, a significant percentage of divers who had problems with equalisation had subsequent onset of vestibulocochlear symptoms occurring within 24 hours of the dive, but mostly on ascent.[21] ENG demonstrated subclinical vestibular injury in four of 50 divers.[21] In the other, ENG, auditory brainstem response (ABR) and the vestibulo-ocular response smooth harmonic acceleration (SHA) test were used to differentiate central from peripheral causes of vestibular abnormality in divers.[14,46] The algorithm used was not well explained, making it difficult to separate the groups clinically other than via diving risk stratification and ear symptoms.[14,46]

In another series, clinical criteria were used to differentiate IEDCS from IEBt; however, once again the precise algorithm was not described.[55] From these data, 18 divers diagnosed with IEDCS appeared to have vertigo as a major symptom, and 26 diagnosed with IEBt had mainly hearing impairment (although nine had vestibular symptoms).[55] Only five of the 18 divers in the IEDCS group had audiograms, which makes interpretation difficult.[55] Large right-to-left cardiac shunts were detected in 15 of the 18 divers with IEDCS using bubble contrast echocardiography.[55] Unfortunately the study was weakened by failure to perform this test on divers identified as IEBt. Video oculography also was used in their diagnostic cache, but too inconsistently for analysis.[55]

Table 2
Summary of case series of inner ear barotrauma in divers (see Table 1 for definition of acronyms)

Author (Year)	Study	Diagnostic criteria	Indication for surgery	Treatment	Outcome
Freeman P, Edmonds C 1972[40]	Series of 5 Navy divers with SNHL	Clinical criteria associated with diving; IEDCS ruled out by risk stratification of dives; audiometry pre and post incident.	No surgery	Conservative: all	5/5: divers: persistent SNHL 2/5: global hearing loss 3/5: high frequency SNHL
Parell J, Becker G 1985[23]	Series of 14 divers	Clinical criteria associated with diving; PTA, fistula test and ENG.	Vestibular symptoms and severe hearing loss	Conservative: 12/14 Surgical: 2/12 tympanotomy + repair	Conservative: 7/12 improved Surgery: 2/2 improved, 1 fully
Shupak A et al 1991[46]	Series of 5 recreational divers	Clinical criteria; cases separated from IEDCS by risk stratifying the dive + ENG, ABR and SHA test. Algorithm not clearly defined.	Persistent vestibular symptoms	Conservative: all for at least 72 h initially Surgical: 2/5 1 confirmed RWR + PLF, patched; 1 not confirmed; both windows patched	Conservative: 3/5 cases, 2 improved hearing, 3 improved vertigo Surgery: Both improved vertigo and hearing, 1 fully
Parell J, Becker G 1993[24]	Long-term follow up of 20 divers (7 professional, 13 recreational) with confirmed IEBt who continued diving; may include some of 1985 series)	History ear injury/symptoms associated with diving; clinical criteria + PTA, fistula test and ENG. (all SNHL, 10 vestibular +/- tinnitus, 7 tinnitus only)	Vestibular symptoms and/or severe hearing loss	Conservative: 16/20 cases (not PLFs) Surgical: 4/20 cases	Conservative: 9/16 improved Surgery: 2/4 improved Long term outcomes: 19/20 remained stable (median follow up 54 months; 8–2,000 dives completed post injury)
Roydhouse N 1997[25]	Series of 20 divers; 19 RWR, 1 OWR (16 cases personally managed by author)	Clinical criteria – onset vestibular symptoms +/- deafness during/after diving.	Positive fistula sign or worsening signs/ symptoms	Conservative: 6/20 cases Surgical: 14/20 (4/20 managed by surgeons other than author)	Conservative: unclear Surgery: 8/10 PLF confirmed; 10/16 continued diving without issues (loose follow up) Surgical candidates stated to have better prognosis but not supported by presented data
Sheridan M et al 1999[26]	Series of 3 divers	Clinical criteria – onset during/ after diving of vestibular symptoms +/- deafness + fistula tests.	Worsening vestibular symptoms	Conservative: 1/3 (suspected intracochlear haemorrhage) Surgical: 2/3 cases (1 OWR, 1 RWR); fistula tests equivocal or –ve in surgically diagnosed PLFs	Conservative: hearing improved (resumed diving) Surgery: improved vestibular symptoms and hearing (neither resumed diving)

Table 2 (continued)

Shupak A et al 2003[14]	Series of 43 recreational divers (24 IEDCS, 19 IEBt); 9 IEBt cases were followed up long term	Same methodology to separate IEDCS/IEBt as Shupak (1991), but clinical criteria unable to distinguish IEDCS/IEBt. Follow up: mean 28 months, range 1–72 months	Deterioration of hearing or severe vertigo; 4/9 vestibular symptoms, 2 cochlear, 3 cochlear + vestibular	Conservative: 7/9 Surgical: 2/9 tympanotomy with both windows repaired (these cases were not specifically identified)	Conservative vs. surgical outcomes unable to be identified Cohort outcomes reported only: 6/9 fully recovered, 2/9 residual SNHL, 1/9 residual vestibulocochlear symptoms
Edmonds C 2004[21]	Series of 50 cases IEBt	Occurrence in low-risk-DCI air diving; clinical criteria (40 had SNHL, 43 tinnitus, 16 vertigo, 4 dysacusis); PTA (≥ 20dB loss in ≥ 2 frequencies), or ENG +/- confirmed PLF at surgery	Persistent tinnitus and vestibular symptoms	Conservative: 33/50 cases Surgical: 13/50 cases 4/50 cases: unable to identify treatment	Conservative: 3/33 full recovery, 21/33 improved tinnitus and vestibular symptoms Surgery: 2/13 cured, 9/13 improved, 1/13 worsened
Klingmann, C et al 2007[55]	Series of 44 divers with 46 cases of acute inner ear disorders 26 divers diagnosed IEBt	Not stated how separated IEBt from IEDCS; appeared to use clinical criteria (vertigo precluded diagnosis of IEBt)	SNHL > 40 dB or severe vertigo with nystagmus	Conservative: 23/26, including high-dose prednisolone, decongestants and rheologic therapy Surgical: 3/26 tympanotomy + RWR closure	Conservative: 18/23 persisting vestibulocochlear symptoms Surgery: 1/3 complete recovery, 2/3 no hearing improvement

Treatment of IEBt

The traumatic aetiologies of PLF have been classified into head trauma, acoustic trauma (external trauma), trauma caused by physical exertion, and barotrauma (internal trauma).[66] Surgical trauma due to stapedectomy would fall under the first group. It may not be valid to generalise outcomes from treatment of external traumatic causes to barotrauma-induced injury. The internal trauma group is applicable to diving barotrauma and, where relevant, outcomes from clinical series of mixed aetiology are included in this section.

CONSERVATIVE MANAGEMENT

Conservative management is recommended by a number of authors as initial treatment for IEBt because of its broad pathology.[1,24,26,45,46,51] Generally bed rest is recommended, with the head elevated to 30–40 degrees for one to two weeks, and avoiding significant physical activity for two weeks.[39,53,67] Conservative interventions also include; resting prone, taking decongestants and vestibular suppressants and avoiding activities that provoke an increase in CSF or ICP (e.g., heavy lifting, straining, nose blowing, coughing, sexual intercourse, ear clearing, air travel, high-speed elevators, and loud noises).[14,20,39,45,49,51,53,67] Even subtle changes in ICP caused by moving from a supine to erect position produce some stretch on the round window membrane.[39] Ear plugs have been used to protect against loud noise, and external ventilation tubes also known as 'ear planes' may aid by damping changes in air pressure in the external canal.[2]

Steroids have been used concomitantly with conservative management. The logic behind steroid treatment is its anti-inflammatory properties and to reduce swelling.[30,65] Steroids are recommended management for sudden SNHL and vertigo of possible vascular aetiology.[30] Prednisolone 60 mg for 10 days has been recommended for occupational divers within three weeks of injury but high-level evidence is lacking.[54] Trans-tympanic application of steroids has also been trialled.[30,54] To date, there are no studies supporting or refuting the efficacy, precise dosing or period of administration of steroids in the treatment of IEBt.[1] Vasodilators, anticoagulants (aspirin and NSAIDs) and nicotinic acid are contra-indicated in the treatment of suspected IEBt.[21,39] Other medications (anti-emetics, decongestants, sedatives), may be prescribed 'as required' for symptom control, and may reduce episodes of raised ICP.[22,54]

TYMPANOTOMY

The justification to undertake an exploratory tympanotomy is supported by clinical suspicion of PLF, persistent vestibular symptoms, and severe hearing loss. A positive fistula test may support the decision.[6,20,25,44,46,53,60,65] If vestibular symptoms are debilitating or strongly suggestive of a PLF, relatively urgent surgical repair is recommended by various

Table 3
Summary of the available diagnostic tests and features consistent with inner ear barotrauma

Investigation	Indication	Features/limitations
Rinne test	512 Hz tuning fork used to determine a conductive hearing loss (CHL). For IEBt the response will suggest sensorineural hearing loss (SNHL); both bone conduction and air conduction will be impaired.[29]	Useful if bone conduction PTA is not accessible.
Weber test	For SNHL, the vibrations from 512 Hz tuning fork are perceived louder in the non-affected ear (for CHL they are louder in the affected ear).[29]	Mixed pathology of SNHL and CHL (e.g., associated MEBt) causes difficult test interpretation.[29] SNHL with IEBt may present as a normal ear as affected frequencies can be higher than 512 Hz.[29]
Fistula test	Aims to induce nystagmus (Hennebert's sign) or subjective dysequilibrium (Hennebert's symptom). Positive and/or negative pressure is applied against the eardrum, commonly with a finger on the tragal cartilage, with a rubber bulb or pneumatic otoscope.[11,13,51,63,64] PLF is likely if symptoms are produced.[41]	Accurate in reportedly < 50–70% of cases with PLF.[51,52] Fistula tests may be a useful non-operative diagnostic technique if positive; not sensitive nor specific.[51]
Pure tone audiometry (PTA)	Detects and quantifies SNHL. Air and bone conduction audiograms are the best way to differentiate between MEBT, 8th cranial nerve involvement in IEDCS and IEBt, with IEBt presenting with a mostly high tone hearing deficit (compared to a global SNHL).[20,26] MEBt demonstrates CHL.[57] IEBt and PLF may demonstrate SNHL in the higher frequencies (4,000 Hz plus) or all frequencies.[20] Speech audiometry may demonstrate difficulties with word recognition in PLF.[66] Can aid in anatomically isolating SNHL to the cochlea (secondary to IEBt) or the auditory nerve when compared with PTA.[29] Useful in monitoring recovery. Successful treatment of IEBt = improvement in hearing to within 15 dB of the pre-incident audiogram.[20]	Bone conduction audiometry is not as available or as sensitive as air conduction testing (requires a transducer).[29] Useful in excluding a CHL, particularly in excluding MEBt.[27] Comparison of pre- and post-incident PTA provides invaluable data supporting a confident diagnosis of PLF, as SNHL suggests IEBt. IEBt on a post-incident audiogram is defined as hearing loss ≥ 20 dB in two or more frequencies, compared to the initial PTA.[20] Positional PTA (PTA conducted when lying with affected ear up) is deemed positive if ≥ 2 frequencies improve by at least 10 dB.[47,66] Vertical PTA shows a greater hearing loss to a horizontal PTA after 30 minutes waiting period owing to displacement of air trapped in the cochlea affecting the conduction of sound.[9,20,47,66] Positional PTA is specific for PLF.[9,47] Higher frequency SNHL, i.e., > 4,000 Hz, may also be caused by industrial deafness (a confounder, but is usually bilateral).
High resolution computed tomography (HRCT) – temporal bones	Excludes inner ear and intracranial lesions. Excludes inner ear anomalies that may predispose the person to ongoing IEBt. In acute situations, may identify air in the cochlea or vestibular apparatus (pneumolabyrinth), thereby confirming the presence of a PLF peri-operatively.[8,44,47] Can also exclude the presence of superior canal dehiscence (SCD) which has implications for management (surgery is less successful in the presence of a SCD).[8]	HRCT (1.0–1.5 mm slices) allows the ability to differentiate otic cortical bone from air (which MRI fails to do).[44] Not available in all hospitals and not readily accessible in a timely manner. Requires radiation exposure. Negative test does not rule out IEBt.
Electronystagmography (ENG)	Detects and quantifies vestibular disturbance, as eye movements are electronically recorded in response to provocation.[13,28] Particularly useful if vestibular symptoms are not overtly subjective.[20] Differentiates peripheral from central (cerebral and cerebellar) causes.[20] Nystagmus can be spontaneous, or provoked by eye movements, positional changes, optokinetics, and caloric testing.[58]	In 50-case series, four cases who denied vestibular symptoms had positive ENG on testing.[20] Accurate in 35–91%.[10]

Table 3 (continued)

Caloric test	Tests vestibular function. Ear canal is exposed to water hotter or colder than body temperature, i.e., 30–44ºC, stimulating a compromised labyrinth and inducing nystagmus which can be directly observed or recorded via ENG.[58]	Cannot be conducted on perforated tympanic membrane.[9] Low specificity in identifying PLFs.[10]
Tullio phenomenon	The affected ear is exposed to a burst of noise (95 dB at 500 Hz) for a few seconds to induce dysequilibrium and/or vertigo = positive test, which may be a sign of PLF.[44,51] Initiates a vestibulospinal reflex from otolith organs.[48]	Low sensitivity in identifying PLFs.[44]
Otoacoustic emission (OAE) testing	OAEs are spontaneous or responsive sounds made by the cells within the inner ear, giving an appreciation of functionality of the inner and middle ear.[54] Helps differentiate cochlea injury from other forms of IEBt.[42] Can detect subclinical IEBt in divers, identifying 'transient emission shifts' as a more sensitive assessment of objective SHL.[54]	Only accessible in specialist laboratories.
Tympanotomy	A diagnostic procedure and treatment for PLF.	Direct vision of PLF possible. High correlation with resolution of vestibular symptoms if PLF confirmed at operation then repaired.[13,48,53]

authors.[6,14,20,23,39,42,46,51,52,65] Likewise, if regular PTA testing (daily in some studies) demonstrates deterioration, surgical treatment may be indicated.[47] A number of studies show significant improvement in vestibular symptoms post surgery (Table 2).[1,6,22,26,46,51,53,65]

Tympanotomy confirms the diagnosis of PLF when leaks are seen from either window at exploration. False positives may occur: an iatrogenic fistula, pooling of local anaesthetic, or misinterpretation of the tissue architecture (e.g., adhesions) for a window rupture.[47,60,68] A number of techniques provoke leakage of the perilymph during tympanotomy: Valsalva manoeuvre in a conscious patient; Trendelenberg positioning; Quekenstedt manoeuvre; internal jugular vein compression or increasing the intrathoracic pressure in an intubated patient.[6,45,48,51,60] PLF may not always be observed; however, a graft placed over both windows may improve symptom severity.[1,6,13,26,51] It may also indirectly promote healing of intracochlear structures. Grafting carries a potential risk of inducing further CHL in an ear with SNHL, hence it is usually undertaken in the setting of more severe symptoms.[45] Despite this caveat, tympanotomy and grafting appears a relatively safe intervention.[1,4,6,48,51,60]

According to studies of divers, ataxia and vertigo are significantly improved with surgical intervention, and tinnitus is mostly relieved; hearing recovery is less consistent.[3,6,39,44,47,65,67] However, the procedure has not been tested in a randomized controlled trial, and any comparative trials are limited by selection bias (Table 2). A theoretically increased risk of labyrinthitis in the setting of a PLF has not been proven; however, case studies have documented a link with chronic PLF.[39,45,65] Out of positive PLFs confirmed surgically, 96% had a resolution in vestibular symptoms with window closure.[4] Even those identified clinically at risk of PLF but not confirmed at surgery had a 68% recovery from vertigo, dizziness, nystagmus, and other symptoms after surgical repair.[4]

A key finding from this and other studies was that no individual had deterioration in their symptoms despite all cases receiving tympanotomy and repair irrespective of whether a PLF was confirmed. The associated symptoms of nausea, vomiting and motion intolerance also are improved, as is cognitive memory.[48] Surgical intervention for RWR was successful in treating vertigo for all patients in another series, but had variable effect on tinnitus and hearing loss.[53] Similar findings were found in a small series of nine cases.[1]

In the retrospective series of 50 divers with IEBt, two thirds were treated conservatively with improvement in two thirds of these, but complete recovery in only five divers.[21] A quarter underwent tympanotomy with mixed results. One patient reporting worse symptoms post-operatively.[21] In one diver; the delay to surgery was 10 weeks post injury, yet symptoms of vertigo and tinnitus improved markedly.[21] Small numbers and multiple other confounders prevented

Table 4
Other investigations of limited use in the diagnosis of inner ear barotrauma

Investigation	Indication	Features/limitations
Auditory brainstem response (ABR)	Distinguishing cochlear from retrocochlear lesions[30,46]	Aids with determining auditory and vestibular pathway pathology in the brainstem; useful in isolating inner ear versus central nervous system causes [46]
Smooth harmonic acceleration test (SHA)	Assessment of the vestibulo-ocular response[46]	Used alongside ABR to isolate central versus peripheral pathologies;[46] requires highly specialised laboratory
Magnetic resonance imaging (MRI)	Excluding differential causes	Sensitive in detecting soft tissue lesions, e.g., acoustic neuroma, vestibular schwannoma, cholesteatoma, or multiple sclerosis plaques;[8,14,30] not widely available
Biochemical markers e.g., β2-transferrin	Visualisation of β2-transferrin intraoperatively (when applied as an assay);[44] β2-transferrin only produced in CSF and perilymph[45]	PLF is otherwise too small to detect directly. Time consuming (hours) laboratory analysis, therefore, impractical for intra-operative diagnosis[45]
Craniocorpography (CCG)	An objective test in which the upper body is recorded with the patient marching on the spot while blindfolded[60]	Not sensitive or specific for IEBt[60]
Electrocochleography	Aids in delineating Meniere's disease from PLF[8]	Technically challenging; limited access to testing; difficult interpretation
Vestibular evoked myogenic potential VEMP)	Identifies Tullio phenomenon from SCD[8,14]	Operator dependent; high error risk = poor quality

formal statistical comparison between surgical and conservative groups. Normal hearing was also reported post-operatively in two of 13 cases; the two with resolution were either operated on shortly post injury or had only a very small fistula.[53] Exploratory tympanotomy may provide diagnostic and curative management of a PLF, although there is some debate as to the indication and timing of exploratory surgery.[47,60]

TIMING OF REPAIR OF PLF

Timing of surgical exploration and repair of PLF has received considerable attention, derived from studies of both diving-related and non-diving-related trauma. The general opinion is that urgent tympanotomy is unnecessary.[1,6,22,44,45,46,51,53,55,65] One to three weeks is recommended to allow for spontaneous healing or resolution of acute ear pathology and facilitation of access to the labyrinthine windows, depending on the severity of SNHL and vertigo.[6,22,23,44,46,53,67] For incapacitating vertigo, severe hearing loss or deteriorating hearing post injury, tympanotomy within 24–48 h of injury has been recommended.[3] It appears that little improvement in hearing is achieved if exploration occurs greater than two weeks post injury.[3]

With strict bed rest, spontaneous healing should occur within four days, with five to 10 days as the limit to conservative management for suspected MEBt and IEBt not including PLF before review and consideration for surgery.[23,39,53] Others recommend early (< 48 h) surgery if the tympanic membrane is also ruptured, because OWR was more likely in this setting.[65] Some recommend antibiotics, sometimes with steroids, prior to any repair undertaken later than 48 hours post injury.[65] In one series, hearing improved within 10 days of injury compared with those who waited four to six weeks.[1] Ten days post injury seems to be accepted as the best time to intervene, with no maximum time. The ability to improve hearing does depreciate with time.[1,26,53,67] The conservative approach allows time over weeks to months for central vestibular compensation to occur, correcting peripheral vestibular dysfunction.[19,57]

There is an up to 10% risk of recurrence after surgical repair.[4] Tympanostomy tubes have been used to aid resolution of PLF symptoms by reducing external pressure effects on the stapes.[2] However, this is not an appropriate management option for occupational divers. If symptoms persist or return following PLF repair, one author recommended repeating

the repair no more than twice.[2] The severity of symptoms correlates negatively with prognosis, irrespective of the treatment.[1]

Prognosis for the injured diver and returning to diving

It has been proposed that divers with IEBt are prone to further incidents that exacerbate their tinnitus and hearing loss,[3] but this is not supported by others.[14,24,39] One group stated; "*Although the older literature clearly suggests otherwise, we believe that scuba divers who completely recover from inner (or middle) ear barotrauma may return to diving as long as they exercise caution and care*".[26] However, this advice was not backed up by what these authors reported, as two of their three cases (both undergoing tympanotomy) were advised not to continue diving. Long-term follow up of IEBt cases (seven conservatively and two surgically managed) reported complete recovery of symptoms in six patients, and return to diving as early as one month post tympanotomy repair was permitted, provided that predisposing anatomical abnormalities were excluded, hearing was stable, and their balance was normal.[14] This study has some limitations as it included both IEDCS and IEBt cases and lacked a clear clinical algorithm for making the diagnosis. In addition, follow-up was reported for only nine of the 19 divers, creating significant risk of positive selection bias.[14]

Of 20 divers who returned to diving against medical advice, only one sustained a further episode of IEBt.[24] Seven of these were professional divers and four had undergone surgical repair of their PLF.[24] These findings suggest that divers may not be at increased risk of recurrence of IEBt when returning to diving. It was not reported whether these divers were part of a larger cohort of divers with IEBt who received the standard counselling post injury "*to discontinue diving*".[24] Once the manifestations of IEBt have settled with no disequilibrium, hearing is within normal range and other clinical parameters normalised, it has been suggested that the diver can return to light duties after 10 days, full duties at six weeks, and diving after three months.[3]

In the retrospective series of 50 divers with IEBt, return to diving was not supported, on the basis that a high proportion of them had predisposing risks, such as MEBt and ear clearing difficulties.[21] In contrast, the recommendation that "*after careful and probably radical repair of a round window membrane rupture, the diver can return to diving*" was on the proviso that the diver received counselling from an ENT surgeon knowledgeable about diving, and adhered to safe diving practices, including ear clearing.[25]

In 917 patients responding to a survey sent to 2,222 past stapedectomy patients, 208 had gone snorkelling, scuba diving or sky diving post procedure.[69] Twenty-eight provided detailed responses regarding symptoms in relation to diving activities and of 22 who had scuba dived, four had experienced otalgia, tinnitus or vertigo and one had SNHL and vertigo unrelated to diving. It was concluded that there were no significant diving-related long-term effects when diving after stapes surgery.[69] These results may be affected by selection bias.

Characteristics that would exclude divers and who should avoid further risk or aggravation of IEBt from returning to diving are:
- symptomatic non-compensated vestibular damage;
- anatomical risk factors identified on HRCT or tympanotomy;
- persistent difficulties with ear clearing and MEBt;
- persistent, significant global hearing loss.

The risk not only involves compressed gas diving, but also other activities such as free diving, skydiving, and flying, where pressure changes can be pronounced.[3]

From this literature review, the recommendation for returning to diving depends on satisfying five criteria:
- Hearing loss is in a narrow frequency band and stabilised.
- Vertigo or imbalance is not a feature.
- Risk factors for MEBt are mitigated.
- No anatomical risk factors have been identified.
- No further surgical intervention is required.

Residual hearing loss is not a contraindication to diving per se, although the deficit could worsen. Occupational hearing loss is a common work-related (compensable) injury in Australia. Diving is contra-indicated where there is active vestibular disturbance.

There are valid reasons to differentiate between occupational divers and recreational divers. The decision not to undertake further diving has less impact on recreational divers who do not rely on diving for their livelihood. Occupational divers are dependent on diving for income, and are usually keen to return to diving where possible. Occupational divers also have different risk profiles to recreational divers. They are task-focused, have less control over the diving conditions and frequency, and their equipment is less conducive to ear clearing (e.g., full face masks). This may place them at greater risk of recurrence of IEBt. The overlap in symptomatology between recurrence of IEBt and vestibular DCI may also cause future diagnostic dilemmas. Occupational divers may be under greater incentive to return to diving after an upper respiratory tract infection (URTI), adding to risk of IEBt recurrence. An additional recommendation to avoid diving for at least two weeks following an URTI has been proposed.[23]

Returning to diving for occupational divers requires negotiation and risk acceptance by all stakeholders. Despite medical clearance to return to diving, employers may not be prepared to accept the risk.

Recommendations from this literature review

- All divers require a baseline assessment of hearing prior to commencing diving (compulsory with diving medicals AS4005.1 and AS/NZS 2299).[61,62] Where IEBt is suspected, reference to the baseline audiometry can identify and monitor cochlea injury, thereby assisting diagnosis and management.
- Audiometry should be repeated at each subsequent health assessment because of the risk of subclinical IEBt and other aural pathology in divers, and to allow an up-to-date reference point for new injuries.
- All divers require education regarding the rationale and technique for equalising their middle ear effectively and regularly during their dive. The potential impact of ear clearing difficulties should be emphasised and, if persistent, dives terminated and medical advice sought.
- Investigations that will assist to identify IEBt include PTA (air conduction/bone conduction) with comparison to previous audiograms, the fistula test, an ENG, and combining PTA or ENG with pneumatic manoeuvres.
- A diagnosis of IEBt and particularly PLF is achieved by a thorough history assessing the risk of the dive profile, rapidity of onset of vestibular symptoms or hearing loss, problems with equalising, timing of symptoms in relation to ear equalisation manoeuvres or other manoeuvres, such as straining, coughing or lifting, and the use of the fistula test.
- If IEDCS cannot be excluded, a trial of hyperbaric oxygen treatment does not appear to worsen IEBt (case data) or its prognosis for recovery, provided the diver can equalise their ears. Careful and gentle ear clearing is advised.
- Conservative management is first-line care for almost all suspected IEBt, except with profound global hearing loss and/or major vestibular symptoms. This allows for healing of mild IEBt (inner ear haemorrhage, membrane tears) and MEBt and the development of some central compensatory mechanisms.
- Use of steroids has little supporting evidence in the management of IEBt, but is commonly used.
- Indications for explorative tympanotomy and closure of a confirmed or suspected PLF include severe or persisting vestibular symptoms or hearing loss, and/or deterioration of these symptoms, or lack of improvement in these symptoms over 10 days.
- HRCT of the temporal bones may identify predisposing anatomical abnormalities and exclude any congenital propensity to IEBt. This should be performed before clearing an occupational diver for return to diving.
- Returning to diving as early as one to three months post injury may be considered after stabilisation of hearing, absence of disequilibrium, normalisation of all vestibular symptoms, exclusion of anatomical predisposition and education to prevent MEBt.

Occupational divers may return to full diving capacity after PLF repair (provided the above criteria are satisfied). This would require extensive counselling as to the need and techniques for equalising, and future impacts of any concurrent URTI. It would also require risk acceptance by the employer.

Conclusion

IEBt constitutes pathology of the inner ear induced by failure to equalise pressure changes secondary to diving. IEBt produces tinnitus, vertigo and impaired hearing and balance. Diagnosis requires a combination of clinical suspicion, dive risk assessment, clinical assessment and investigation, including reference to baseline PTA. There is no definitive clinical algorithm or single diagnostic test that objectively confirms IEBt. Diagnosis of the PLF subset of IEBt is supported by positive fistula test and direct observation at tympanotomy. Conservative management is recommended first-line therapy for IEBt, with exploratory surgery indicated for severe or persisting vestibular symptoms or hearing loss, and/or deterioration of these symptoms, or lack of improvement in these symptoms over 10 days.

Provided that the SNHL is stable and not severe (measured by PTA), and there is no sign of uncompensated vestibular involvement (via ENG) and HRCT has excluded anatomical predilection to IEBt, it may be possible for divers to resume diving. This requires careful counselling of the diver regarding ear clearing, risk acceptance by all stakeholders, and detailed, informed consent before actioning. There is need to establish a data registry and long-term follow up of divers returning to their occupation post IEBt in order to gain a better understanding of their functional recovery, risk of recurrence or morbidity.

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