Review articles

Scuba diving and otology: a systematic review with recommendations on diagnosis, treatment and post-operative care

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Abstract

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Scuba diving is a popular recreational and professional activity with inherent risks. Complications related to barotrauma and decompression illness can pose significant morbidity to a diver's hearing and balance systems. The majority of dive-related injuries affect the head and neck, particularly the outer, middle and inner ear. Given the high incidence of otologic complications from diving, an evidence-based approach to the diagnosis and treatment of otic pathology is a necessity. We performed a systematic and comprehensive literature review including the pathophysiology, diagnosis, and treatment of otologic pathology related to diving. This included inner, middle, and outer ear anatomic subsites, as well as facial nerve complications, *mal de debarquement* syndrome, sea sickness and fitness to dive recommendations following otologic surgery. Sixty-two papers on diving and otologic pathology were included in the final analysis. We created a set of succinct evidence-based recommendations on each topic that should inform clinical decisions by otolaryngologists, dive medicine specialists and primary care providers when faced with diving-related patient pathology.

Key words
Head and neck; ENT; Injuries; Medical conditions and problems; Barotrauma; Decompression sickness; Review article

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Introduction

Over the past half century there has been a dramatic increase in the number of recreational scuba divers, with over nine million certified in the United States in 2015 and approximately 100,000 new divers per year.[1] While scuba diving is commonly viewed as a safe recreational activity, it exposes the participant to real risks of injury or even death. More than 80% of all diving complications occur in the head and neck.[2] Of these, approximately 65% are outer, middle or inner ear disorders.[3] It is essential that clinicians understand the physiology and physics of scuba diving as well as the diagnosis, treatment and prevention of diving-related complications. However, there are currently few evidence-based recommendations or systematic reviews on this topic. The purpose of this report is to systematically review the current literature evaluating scuba diving physiology and complications pertinent to otology, and to provide a comprehensive resource with evidence-based recommendations where possible.

Two important scuba diving injuries are barotrauma and decompression sickness (DCS). To understand their pathophysiology, clinicians must understand the physics laws governing these injuries: Boyle's Law and Henry's Law.

BAROTRAUMA AND BOYLE'S LAW

Barotrauma is a pressure-mediated injury to tissue governed by Boyle's law. As a diver descends and the pressure increases, the volume of the gas compresses. This can result in a relative negative pressure in rigid- or semi-rigid-walled air-containing spaces in the body such as the middle ear or paranasal sinuses. Negative pressure can cause mucosal oedema, haemorrhage and even perforation if the space cannot equalize. On ascent, the volume of gas increases as the ambient pressure decreases. If an air-containing space cannot equalize with the surrounding pressure, the expanding volume of the gas may result in a variety of head and neck pathologies, including middle ear perforation or pneumocephalus from sinus barotrauma.[4]

DECOMPRESSION SICKNESS AND HENRY'S LAW

As a diver descends and ambient pressure increases, progressively higher pressure gas is delivered to the lung and more inert gas dissolves in the blood stream. The amount of inert gas that dissolves in a given tissue is proportional to the maximum depth and bottom time, as well as the perfusion and diffusion characteristics of that tissue. As a diver ascends, the additional inert gas load comes out of

solution at the level of the alveolus and is exhaled. If the rate of ascent exceeds the rate of alveolar gas exchange, inert gas will dissolute inside the diver, forming bubbles within the circulation and in tissues. The severity and nature of the DCS injury vary from mild systemic, musculoskeletal and cutaneous manifestations to severe, life-threatening central nervous and cardiorespiratory symptoms.

Literature search

A systematic search of the literature was performed using the following databases: Ovid/Medline, PubMed, EMBASE, UpToDate, Rubicon Repository and Cochrane Review Database up to January 2017. A screening literature search was used to identify all literature discussing scuba diving and any otolaryngology topics. Search terms included: "*scuba*" and/or "*diving*", and "*head and neck*", "*otolaryngolog**", "*otolog**" "*rhinolog**", "*sinus surgery*" or "*laryngolog**". Reference lists of identified publications were reviewed to ensure no relevant studies in this field were missed. Grey literature, including the Diver's Alert Network online resources, was also queried for completeness. Inclusion criteria included any paper discussing scuba diving and otology at any level of evidence (LOE). Exclusion criteria included papers that were not available in English or in an English translation. Given the limited amount of literature available, all studies meeting these inclusion criteria were included for completeness.

The combined search resulted in identification of 398 abstracts to be reviewed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.[5] Screening resulted in 285 abstracts being excluded owing to duplications, leaving 113 abstracts to be reviewed. In assessing eligibility, 19 abstracts were excluded as they were not available in full text or in English, two because the topic did not include scuba diving, 20 as they did not discuss otology as it relates to diving and 23 as they solely discussed rhinology and oromaxillofacial topics. This left a total of 49 articles that met the criteria of including both scuba diving and otology topics. The works cited section of these articles were reviewed and 13 additional studies were also identified for review.

Included studies were evaluated and their LOE was noted based on a reported research methodology utilized by the Oxford Centre for Evidence Based Medicine (CEBM).[6] After quality evaluation for each study, a summary was produced that included the aggregate grade of evidence and relevant recommendations. When there was only a single study available, an aggregate grade of evidence was not provided as grades are derived from the findings of multiple studies. Two authors (DML and KAS) reviewed the literature and produced the initial manuscript. A subsequent author (BL) was asked to review and critically appraise the recommendations based on the literature. Recommendations incorporate both the quality of evidence and the balance of benefit versus harm.

After additional review and synthesis of all information, a total of 74 articles were included in the study. There were no systematic reviews or meta-analyses found. The highest levels of evidence came from randomized controlled trials (Level 1b). The Appendix contains a summary of the 44 relevant papers that constituted primary literature related to otology and scuba diving. Eighteen papers were relevant but were either reviews, grey literature or individual case reports, and thus not included in the Appendix. The references section also contains 12 papers that did not specifically mention diving, but were relevant to the method used or contextualizing the relevant literature.

External ear

OTITIS EXTERNA

Otitis externa (OE) is the most common otologic disorder among divers, afflicting nearly half of all active divers on at least one occasion.[7] *Pseudomonas aeruginosa* is the most common micro-organism responsible.[8] Treatment is no different among divers versus non-divers and should consist of dry ear precautions, topical antimicrobial therapy (e.g., Ciprodex: three drops to affected ear BID x 7 days) and serial debridement of the external auditory canal. Divers should be counselled on the prevalence of this condition and may consider bringing antimicrobial drops on remote diving trips. Ethanol or acetic acid otic drops may be useful to prevent OE by drying the canal post-dive, and can be considered in divers prone to this condition.[4]

Recommendations (LOE)

- Divers should be counselled on the high prevalence of OE (5);
- Suggest bringing topical therapy on remote trips (5);
- Treatment similar to non-diving related OE (5).

EXOSTOSES

Exostoses are bony outgrowths of the external auditory canal (EAC) that typically occur bilaterally in scuba divers.[9] They are very common, with a prevalence of up to 40% among professional divers,[8] in comparison to about 6% among the general population living in coastal regions.[10] Exostoses can lead to recurrent otitis externa, recurrent otorrhoea, cerumen impaction and conductive hearing loss from canal obstruction.[8–10] If the canal is completely occluded, an asymmetrical caloric stimulus with vertigo can occur upon submersion.[4] Divers with symptomatic exostoses should seek evaluation by an experienced otolaryngologist for consideration of canalplasty.[9] Surgical indications for removal are identical to non-divers, with the caveat that they are very likely to experience progression of their exostoses due to further water exposure, and are at higher risk of OE.[7,9] Exostoses should not affect fitness to dive unless they are occluding the canal or causing recurrent infections.[11]

Recommendations (LOE)

- Exostoses can lead to recurrent otitis externa, recurrent otorrhoea, cerumen impaction and conductive hearing loss (4);
- Divers with symptomatic exostoses should seek evaluation by an experienced otolaryngologist for consideration of canalplasty (5);
- Exostoses should not affect fitness to dive unless they are symptomatic (5).

EXTERNAL AUDITORY CANAL BAROTRAUMA

External auditory canal barotrauma can also occur in the setting of occlusion due to cerumen impaction, foreign body, or a tight fitting wetsuit or drysuit hood. Severe exostoses can also cause cerumen impaction and lead to an isolated air space within the external auditory canal. Regardless of the aetiology of the predisposing canal occlusion, a relative vacuum is created on descent causing oedema and haemorrhagic vesiculation of the canal.[12] Treatment of EAC barotrauma consists of a short course of topical analgesic or steroid ear drops, and is similar to the treatment of OE.[13] The aetiology of canal barotrauma should be understood by the clinician and diver to prevent the occurrence of subsequent episodes.

Recommendations (LOE)

- Treat with debridement and a short course of topical steroid/antibiotic (5);
- Refrain from diving until resolved (5);
- Treatment similar to OE (5);
- Identify and mitigate inciting aetiology of EAC barotrauma (5).

Middle ear

MIDDLE EAR BAROTRAUMA AND IMPAIRED EQUALIZATION

Middle ear barotrauma (MEBt) occurs when there is dysequilibrium between the middle ear and ambient pressure, and accounts for up to 46% of patient presentations for diving-related head and neck pathology.[14] During descent, middle ear pressure becomes progressively less than ambient pressure and the Eustachian tube (ET) must open to equilibrate these pressures. If the diver is unable to equalize and continues to descend beyond 1.4 metres depth, a pressure differential of greater than 90 mmHg is created, irreversibly blocking the ET.[12] The persistent negative pressure in the middle ear space can lead to extravasation of fluid and haemorrhage into the middle ear and tympanic membrane (TM) perforation. The resulting middle ear transudation and tubal oedema can impair middle ear ventilation on ascent, leading to a progression of barotrauma.[15] MEBt with TM rupture can also occur during uncontrolled ascent, and is associated with pulmonary barotrauma if a diver ascends with a closed glottis.[16]

MEBt is not always associated with inner ear sequelae. In a cohort of 67 professional divers, recurrent MEBt was not associated with sensorineural hearing loss.[17] Risk factors for MEBt include poor ET function and poor mastoid pneumatization.[18,19] In patients with tubal tonsillar hypertrophy and inability to equalize on descent, there may be a role for tuboplasty operations, including laser tuboplasty. In a series of nine divers, improved middle ear equalization was demonstrated postoperatively with seven being able to return to diving long-term.[20]

Equalization of the middle ear space through the ET is active on descent, and passive on ascent. Importantly, equalization manoeuvre effectiveness is impacted by the diver's head position relative to their body. Clearing capacity is maximized in head-up positioning.[21] Head-down position impairs passive equalization and should be avoided during descent.[22] Multiple manoeuvres exist to assist pressure equalization of the middle ear space, including Valsalva, Frenzel, Toynbee, Lowry, Edmonds and voluntary tubal opening (French: *béance tubaire volontaire* – BTV) techniques. A forced Valsalva can be injurious to the inner ear, and the BTV manoeuvre is considered the least traumatic method.[12] A patient who can perform BTV is able to voluntarily contract their tensor veli palatine muscle and can often maintain ET patency on command. The Fédération Française d'Études et de Sports Sous-Marins has created a series of exercises to facilitate learning BTV. Divers with difficulty with equalization should descend at a slow rate and attempt to equalize pressure with every breath.

Importantly, diving at shallow depth is not useful to prevent MEBt as the greatest change in volume occurs near the water's surface; TM implosion can occur at depths as shallow as 1.2 m.[12] Rupture of the TM can lead to caloric stimulation of the vestibular apparatus, vertigo and disorientation underwater. This can be a highly dangerous scenario if it leads to a diver panicking.

Recommendations (LOE)

- Avoid head down position on ascent/descent (3b);
- Do not descend if unable to equalize, do not utilise the forced Valsalva (5);
- Ideally, utilize the BTV technique for equalisation (5);
- Optimise ET function and equalization technique (2b);
- Formal testing of ET function can be considered (5);
- Tuboplasty techniques may be helpful in specific ET dysfunction cases (4).

ALTERNOBARIC VERTIGO

Alternobaric vertigo (AV) is thought to arise from asymmetrical equalization of middle ear pressure transmitted

Figure 1
Implosive mechanism of inner ear barotrauma and perilymphatic fistula: a successful forced Valsalva manoeuvre (a) communicates excessive pressure to the middle ear space, distending the tympanic membrane, causing implosion of the round window (b) and subluxation of the stapes footplate (c)

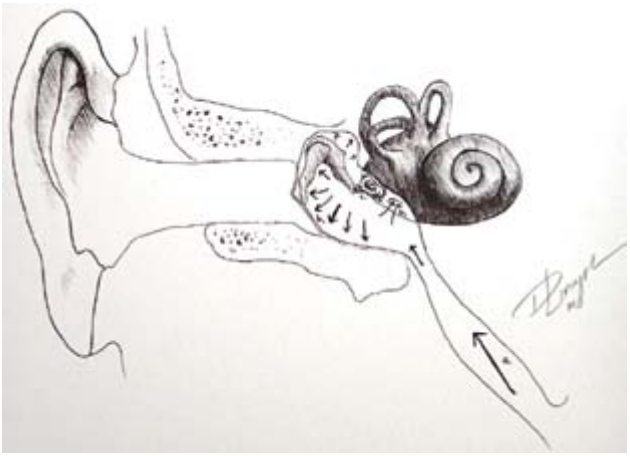
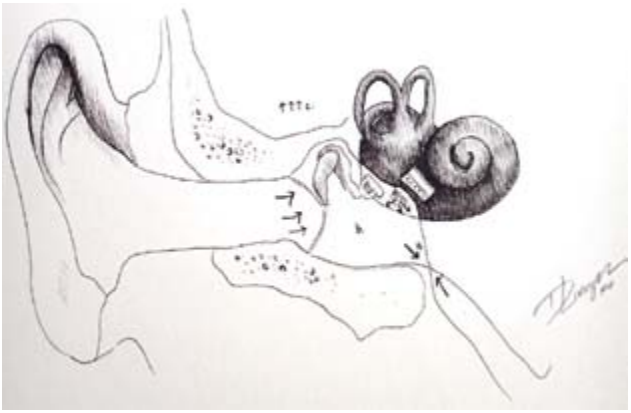


Figure 2
Explosive mechanism of inner ear barotrauma and perilymphatic fistula; a failed forced Valsalva manoeuvre due to a blocked Eustachian tube (a) in the setting of a relative vacuum within the middle ear space (b) causes elevation of CSF pressure (c); this increased pressure is communicated to the inner ear, resulting in round window rupture (d) and intracochlear hemorrhage.



via the oval and round window membranes. AV is the most common cause of transient vertigo while diving,[17] typically during ascent.[4] Vertigo can last for several minutes and is often accompanied by nausea, vomiting and disorientation.[23] Caloric effects on the vestibular system can also cause transient vertigo and are pathophysiologically distinct from the dysequilibrium associated with nitrogen narcosis. ET dysfunction is a significant risk factor for the development of AV, and is considered an independent risk factor for experiencing an adverse event or accident while diving.[24] However, divers with AV were not at increased risk of experiencing a life-threatening or critical event while diving among a cohort of 63 recreational divers or 64 professional divers.[23,24]

Risk factors for AV other than ET dysfunction include previous barotrauma, noise exposure during diving, cold water diving, a history of otitis media, a history of previous episodes of AV and female gender.[23,25,26] AV is approximately four times more prevalent in females than in males.[23] Patients should be counselled on the aetiology and nature of AV as well as the potential risks of this condition, including aspiration and death. If a diver can tolerate the transient vertigo and pause their controlled ascent or descent until their vertigo resolves, the risk to the diver is minimized. Transient vertigo of any aetiology during technical or commercial diving portends a significantly higher risk of disorientation, accidental regulator dislodgement, scuba apparatus damage and asphyxiation.

Recommendations (LOE)

- Divers must understand the risks of disorientation, accidental regulator dislodgment, scuba apparatus damage and asphyxiation due to attacks of AV (2b);
- Recommend ET function optimization to minimize AV symptoms (5).

Inner ear

INNER EAR BAROTRAUMA

Inner ear barotrauma (IEBt) occurs when pressure changes within the middle ear are transmitted to the cochlea through the round and oval windows, and can occur through either implosive or explosive mechanisms (Figures 1 and 2). The oval window is somewhat more protected from pressure changes compared to the round window due to the added stability of the stapes footplate and associated ligamentous attachments. The stapes footplate can, however, be forcefully displaced by divers that employ forced Valsalva methods for middle ear equalization during descent. High pressure air is forced into the middle ear space, causing rapid outward displacement of the stapes and inward displacement and implosion of the delicate round window membrane (Figure 1). The pressure wave generated by this manoeuvre can cause cochlear haemorrhage with disruption of Reissner's membrane and the basilar membrane. Perilymphatic fistula (PLF) can also occur due to tearing of the round window membrane via an explosive mechanism of IEBt. Rapid fluctuations in the CSF pressure can be communicated to the perilymphatic space via the cochlear aqueduct or the lamina cribosa. A failed Valsalva attempt in the setting of ET dysfunction may elevate CSF pressure causing round window membrane rupture (Figure 2). Early studies and grey literature on diving noted an association between hearing loss and diving, even in the absence of IEBt. Importantly, high frequency sensorineural hearing loss is common among commercial divers, but is likely a noise-induced phenomenon from the use of underwater machinery.[27] High activity sport

divers do not experience any increased risk of SNHL.[28,29]

Certain anatomic risk factors may predispose divers to IEBt. Patients may have an enlarged cochlear aqueduct orifice that can more readily transmit elevated CSF pressures to the delicate structures of the membranous labyrinth.[30] This may also be a consideration among paediatric divers who have a shorter cochlear aqueduct that can more easily transmit pressure fluctuations within the subarachnoid space.[31] The approximate length of the cochlear aqueduct in a newborn is 3.5 mm and undergoes postnatal lengthening to 10 mm in an adult.[32] The additional pressure may also cause rupture of Reissner's or the basilar membrane, leading to admixture of perilymph and endolymph.[33] Other anatomic risk factors for PLF are the potential communication between the inner and middle ear through the *fissula ante fenestrum* and enlarged vestibular aqueducts.[30,34]

Diagnosis of IEBt can be difficult, with vague and variable symptom onset. Vestibular dysfunction and/or hearing impairment as a consequence of IEBt can occur on descent owing to impaired equalization, on ascent from expanding pneumolabyrinth, or even on non-diving days while straining or lifting.[35] The symptomatology and severity are dependent on the specific anatomical subsite affected; isolated basilar membrane tears can present with sensineural hearing loss (SNHL) as the only presenting complaint.[36] Typically, patients present with a combination of vestibular and hearing deficits, and may complain of aural fullness, tinnitus and hyperacusis.[33] IEBt must be distinguished from inner-ear DCS, though these diagnoses are not mutually exclusive. A dangerous or atypical dive profile, diving using mixed gases or other symptoms of DCS elevates the likelihood of inner-ear DCS. Importantly, IEBt can occur in the absence of otoscopic findings of barotrauma, with a normal tympanic membrane and EAC.[37,38] Clinical suspicion must remain high in these circumstances; delay between dive and symptom onset does not exclude the diagnosis of IEBt.[37] The dive profile must be thoughtfully interpreted and can be used to inform clinical suspicion of IEBt and other otologic pathologies.[37, 39]

Treatment recommendations for IEBt include high-dose steroids (250 mg prednisolone for three days) with a taper for a total duration of therapy of 18 days, a recommendation based on expert opinion.[2] Surgical exploration is recommended when clinical suspicion of perilymphatic fistula is high, or the patient's hearing deteriorates despite appropriate conservative therapy. If a tympanotomy is undertaken, both the oval and round windows should be patched with fascia and absorbable surgical packing such as Surgifoam, even in the absence of active intraoperative perilymphatic leakage.[33] Intraoperative identification of a PLF can be facilitated using Trendelenburg positioning, or elevation of intrathoracic pressure in an intubated patient.[35] Conservative therapy for PLF should include bed rest, elevation of the head of the bed to 45^{o}, the use of stool softeners and avoidance of straining manoeuvres.[13] A high-resolution CT scan of the temporal bone should also be performed in all patients that experience IEBt to rule out any anatomic factors that may predispose the patient to future episodes.[30]

Patients should be counselled on the presence of anatomic risk factors that may influence their fitness to dive. Divers who suffer IEBt are often counselled to avoid diving. This recommendation may, however, be unnecessarily restrictive. A cohort of 21 patients who suffered IEBt and continued to dive against medical advice were counselled on middle-ear equalization techniques and methods to improve ET function. No further deterioration of inner-ear function was noted among these divers over a 1–12 year follow-up.[40] A recent comprehensive review identified five criteria for returning to diving following IEBt including stable hearing loss in a narrow frequency band, absence of vertigo/dysequilibrium, mitigation of risk factors for MEBt, no further anatomical risk factors present and no further surgical intervention required.[35]

Recommendations (LOE)

- Patients with IEBt should undergo initial observation and medical therapy (3b) including corticosteroids (5);
- Exploratory tympanotomy and round/oval window patching should be performed if there is clinical deterioration, or high suspicion for a PLF (3b);
- A high-resolution CT scan of the temporal bones should be done to rule out anatomic risk factors (4);
- ET dysfunction and risk factors for MEBt should be mitigated (5);
- A fitness-to-dive assessment and consultation should be performed by an otolaryngologist or dive medicine specialist before more diving is undertaken (5).

INNER EAR DECOMPRESSION SICKNESS

Inner-ear DCS occurs according to the principles outlined earlier with regard to Henry's Law. The vestibular portion of the labyrinth appears to be more prone to damage than the cochlea, a phenomenon that is likely related to tissue perfusion and washout of inert gas. The vestibular apparatus has a higher tissue volume to blood supply ratio in comparison to the cochlea, leading to higher rates of local supersaturation and arterial microbubble load.[41] Inner-ear DCS manifests with vertigo occurring within about two hours (h) of surfacing, with up to 40% of patients also experiencing some degree of hearing loss.[41] Haemorrhage and protein deposition in the membranous labyrinth can eventually develop into fibro-osseous labyrinthitis.[42] Inner-ear DCS can occur in isolation, or in combination with other CNS manifestations of DCS. Deep technical diving using He-O_2 and trimix breathing gases appears to confer a higher risk for inner-ear DCS.[33]

Treatment of inner-ear DCS includes recompression with hyperbaric oxygen treatment (HBOT) as soon as possible; if the latency to HBOT for inner-ear DCS exceeds 5 h, approximately 90% of divers can expect some degree of permanent inner ear damage.[43] An animal model study has shown that precipitated material within the perilymph can appear as early as 1.5 h in squirrel monkeys with experimentally induced inner-ear DCS.[42] Unfortunately the median time to treatment is often well in excess of these critical windows.[2] While waiting for HBOT, 100% oxygen should be administered during transport.[44] Adjunctive medical therapy can include steroids to reduce inflammatory oedema and low molecular weight dextran to improve microcirculation.[44] Doppler sonography should also be performed in these patients to rule out a cardiac or pulmonary right-to-left (R/L) shunt. Prevalence of a R/L shunt can be as high as 82% among inner-ear DCS patients compared with 25% in the normal population.[45] Patients with isolated R/L shunts may dive in accordance with 'low bubble diving' recommendations established by the Swiss Undersea Medical Society.[46] If the patient has suffered concurrent IEBt, emergent bilateral middle ear paracentesis or tympanostomy tube insertion followed by HBOT is recommended to prevent a worsening of symptoms.[2,47]

Recommendations (LOE)

- Patients with inner-ear DCS should receive emergent HBOT as soon as possible (3b);
- Medical adjuncts including corticosteroids can be considered (5);
- If there is concern regarding concurrent IEBt, bilateral myringotomies with equalization tubes should be performed (4);
- Once the inner-ear DCS is adequately treated, Doppler ultrasonography to rule out a R/L shunt should be performed (3b);
- The aetiology of the DCS should be considered and the patient must be counselled on preventative measures (5).

Facial nerve pathology

Facial nerve paralysis as a complication of scuba diving is a rare condition that is hypothesized to be a consequence of reduced axonal capillary blood flow in the setting of a defect in the bony covering of the tympanic segment of the facial nerve. Initially this process is caused by negative middle ear pressure due to the relative vacuum created by inadequate equalization.[48] Secondary post-ischaemic intraneural swelling from transudate or blood may perpetuate axonal compression and ischaemia.[15] Direct compression of the nerve can also occur due to trapped haemotympanum.[48] A bony defect of the tegmen, the bony separation between the cranial cavity and middle ear, can also result in pneumocephalus and associated intracranial complications.[49] Compressive neuropraxia may occur during ascent from expanding trapped gas within the middle ear space. Air can also enter a normal facial nerve canal and cause compression by tracking along the chorda tympani nerve.[50] The degree of facial nerve injury should be documented and followed closely for recovery. Oral corticosteroid therapy may be useful to minimize further neuropraxia secondary to intraneural swelling. Facial nerve decompression is generally not indicated although, similar to surgical decompression following temporal bone trauma, there is great institutional variability whether or not this procedure is performed. A simple myringotomy can be performed to evacuate any haemotympanum and relieve the middle ear over-pressure and consequent facial nerve compression.[48]

Recommendations (LOE)

- Corticosteroid therapy is recommended to minimize neuropraxia secondary to direct nerve compression (5);
- There is no consensus on the role of facial nerve decompression (5);
- Perform myringotomy to relieve middle ear over pressurization (4).

Sea sickness

Sea sickness, a type of motion sickness, occurs when there is a mismatch between vestibular, proprioceptive and visual inputs.[4] In diving, this typically occurs on a boat heading to a dive site, while a diver is attached to a shot line during decompression, or during a prolonged stay on a liveaboard diving vessel.[4,51] Divers are typically less susceptible to sea sickness once underwater, which can promote hasty and poorly executed water entries among those affected.[4] Almost everyone is susceptible to sea sickness, but tolerance can develop from episodic exposure and after two to three days of continuous open-ocean exposure.[4] Symptoms include a non-vertiginous sense of dysequilibrium, nausea, increased salivation, flushing, diaphoresis and general malaise.[52] Risk factors include female gender,[53] altered vestibular or visual sensory cues,[54] history of migraine[55] and hormonal effects of pregnancy and oral contraception.[52,56] Psychosocial factors also play a role; naval cadets were at lower risk of developing sea sickness when told they were unlikely to experience it.[57] Specific environmental modifications are useful for treatment and prevention of sea sickness. Lying supine while inside a ship may decrease susceptibility to motion sickness.[51] Standing on deck and staring at the horizon will lessen the degree of vestibular-visual mismatch and improves symptoms of sea sickness. Medications for treatment and prevention of sea sickness are typically antihistamines or anticholinergics. Ondansetron, a 5-HT antagonist, and droperidol, a dopamine antagonist, are typically less effective for motion sickness.[4] Scopolamine, an anticholinergic medication given trans-dermally every 72 h, has been the subject of three randomized, prospective double blind studies on motion sickness. It has been shown to be more effective in preventing motion sickness than

promethazine, meclizine, and lorazepam and has shown superiority over cinnarizine for sea sickness prevention in naval crew.[58,59] Caffeine may also be of benefit when combined with other anti-nauseants for motion sickness; caffeine plus promethazine was shown to be superior to scopolamine in preventing air sickness among helicopter passengers.[60] Importantly, scopolamine has not been shown to affect cognitive performance or manual dexterity in hyperbaric chamber dives.[61] Dimenhydrinate, however, has been shown to have significant cognitive side effects at therapeutic doses necessary to treat experimental motion sickness, and thus is likely unsuitable for diving.[62] In animal studies neither scopolamine nor cinnarizine increased the risk of CNS oxygen toxicity.[63,64]

Recommendations (LOE)

- Environmental modifications such as lying supine or visual fixation on the horizon should improve symptoms of sea sickness (4);
- Transdermal scopolamine q.72 h is superior to cinnarizine for prevention of sea sickness (1b), and superior to other anti-nauseant medications for motion sickness prevention (1b);
- Caffeine may be a useful adjunct for motion sickness medications; promethazine plus caffeine is more effective than scopolamine alone (1b);
- Caution should be exercised when diving while under the influence of any of these medications, and sedating medications should be avoided (5);
- Divers should not dive while nauseated due to the risk of vomiting underwater and subsequent asphyxiation (5).

Mal de debarquement

Mal de debarquement (MdDS) is the sensation of swaying or rocking movement with dysequilibrium that occurs in individuals upon returning to land after an extended period of time in a boat. Most patients have resolution of symptoms within minutes to days. Of 236 sailors, 73% experienced MdDS symptoms for up to 24 h (mean 2.6 h).[65] In some people, symptoms are persistent for months to years and are associated with significant fatigue, anxiety and cognitive impairment. Risk factors include female gender and previous history of seasickness.[47] Human and primate studies have implicated central maladaptation of the vestibulo-ocular reflex as a primary cause of MdDS.[66,67] Treatment for MdDS is generally considered to be ineffective, though recent work focusing on readaptation of the vestibulo-ocular reflex (VOR) has shown some promise. In one study, 24 subjects with persistent MdDS (mean duration 19.1 months) were treated by rolling the head side-to-side while being exposed to a full-field optokinetic stimulus in an attempt to readapt the VOR. Head roll frequency was matched with the subjective oscillations felt by the subjects, with direction of optic kinetic rotatory stimulus based on the Fukada stepping test or a patient's subjective sense of rotation. Seventeen subjects showed cure or substantial improvement at one-year follow-up. Other proposed therapies include vestibular rehabilitation, and use of long-acting benzodiazepines and tricyclic antidepressants.[68]

Recommendations (LOE)

- Novel techniques to alter a maladapted VOR in MdDS may prove to be useful (2b);
- Recommend trial of medical therapy, and vestibular physiotherapy as adjuncts to refractory cases (5).

Post-operative recommendations

TYMPANOSTOMY TUBES

When diving with tympanostomy tubes at sufficient depth or pressure, the surface tension of water at the tube orifice will be exceeded and water will enter the middle ear space. This can generate an asymmetrical caloric effect, leading to transient vertigo and dysequilibrium. There is also an increased risk of otitis media from water freely entering the middle ear leading to middle-ear sequelae and early tube extrusion. Patients should be counselled on these serious risks if they are considering diving with tympanostomy tubes, and may be considered unfit to dive. Some authors advocate use of one-way membrane (Castelli-type) tympanostomy tubes to prevent these sequelae.[12] A scuba diving mask that keeps water out of both ears, such as the ProEar 2000, may also be useful in this patient population.[12] The ProEar mask is built with a tube connecting the ear covers to the facial portion of the mask. This ensures that the air space within the EAC remains equal to ambient pressure, preventing EAC barotrauma.

TYMPANOPLASTY

The integrity of the TM while diving is closely related to its compliance. Sclerotic, immobile membranes may impair middle ear equalization, while a monomeric, hypermobile TM may be at higher risk for rupture during a forced Valsalva. Patients who have previously undergone tympanoplasty may also have impaired ET function at baseline and be at higher risk for associated barotrauma. Safety to return to diving should be considered on a case-by-case basis, and a trial of diving at depth in a controlled environment such as a swimming pool should be considered.[69]

STAPEDECTOMY

Stapes surgery was thought to increase the risk of IEBt, owing to an iatrogenic predisposition for perilymphatic fistula formation. However, no increased risk of otologic insult was found among large cohorts of divers who had undergone stapedectomy.[70,71] Postoperative recommendations include dry-ear precautions for the first three weeks, and allow diving one month postoperatively, assuming normal ET function.

Appendix

Primary literature on otology and scuba diving; AV – alternobaric vertigo; CRS – Chronic rhinous sinusitis; ENG – electronystagmography; ETD – ET dysfunction; ET – Eustacian tube; HBOT – hyperbaric oxygen treatment; IEBt – inner ear barotrauma; IEDCS – inner-ear decompression sickness; MEBt – middle ear barotrauma; NIHL – noise-induced hearing loss; OM – otitis media; PLF – perilymphatic fistula; PTA – pure tone audiometry; RCT – randomised controlled trial; SCC – semicircular canal; SHA – sinusoidal harmonic acceleration testing; SNHL – sensorneural hearing loss; TMJ – temporo-madibular joint; VOR – vestibulo-ocular reflex

First Author	LOE	*n*	Study design	Methods	Results	Conclusions/comment
External ear						
DiBartolomeo[10]	4	70	Retrospective cohort review	Chart review for exostoses	Incidence 6.36/1,000	Epidemiology and risk factors for exostoses identified
Middle ear						
Antonelli[72]	5	11	Retrospective post-mortem	18 temporal bones from 11 divers: 8 rapid ascent; 3 drowning, no rapid ascent	Middle ear haemorrhage in all rapid ascent bones; 5/11 rapid ascent TMs perforated	General commentary on histology of temporal bone injury due to rapid ascent
Ivarsson[21]	4	3	Case series	ET clearance in various head positions	Supine positioning impaired equalization on ascent and descent	Assuming an upright body posture during exposures to pressure changes is ideal
Jumah[20]	4	9	Prospective, non-consecutive case series	Divers with ET laser tuboplasty; pre- and post-op ET function/opening pressure	All subjects restored ability for pressure equalization; 7/9 able to resume diving	Divers with ETD may benefit from minimally invasive laser tuboplasty
Money[16]	4	3	Case series	Postmortem histology of temporal bones from recently deceased divers	MEBt found in pulmonary barotrauma patient; IEDCS case had neo-ossification in SCCs	Breath holding causes MEBt; maladaptive neo-ossification occurs following IEDCS
Ornhagen[22]	3b	7	Case control series	Dry and wet pressure chambers; measured passive ET opening vs head position	Horizontal position and prone position equivalent; head-down position worse	Avoid head down position on ascent
Roydhouse[3]	3b	650	Retrospective cohort study	Review of patients with diving-related ENT pathology	64.6% ear, 23.9% teeth/TMJ, 3.1% nose, 6.6% paranasal sinuses	Most common preventable cause of diving pathology is reversible nasal congestion
Uzun[18]	2b	24	Prospective cohort study	Survey, CT scan temporal bones of mastoid pneumatization	MEBt occurred in 15 ears of 11 divers; pneumatization related to risk of MEBt	Inverse relationship between degree of pneumatization and risk of barotrauma
Uzun[19]	3b	31	Retrospective cohort study	History, mastoid pneumatization, ET function	MEBt in 19 ears of 14 divers; ETD higher in divers	ETD measured by 9-step test and small mastoid are risk factors for MEBt
Vertigo						
Caruso[73]	4	14	Retrospective, consecutive case series	Audiometry, vestibular testing and DCS questionnaire	11/14 abnormal otologic, neuro-otologic findings	Expanded classification for vertigo related to diving
Kitajima[74]	3b	64	Non-consecutive cohort study	Sonotubometry and impedance tests	Diving incident group had significantly worse ETD	Divers with ETD prone to AV; check ET function pre dive
Klingmann[23]	2b	63	Retrospective cohort	Questionnaire, otoscopy, audiometry, caloric testing, ABR and MRI, etc	Higher prevalence of AV and ETD in females	Female divers 4x more likely to suffer AV; no increased risk for death if you experience AV
Molvaer[25]	3b	194	Retrospective cohort	Audiometry, interview of professional divers	39% had vertigo; 33% from AV; risk factors: previous barotrauma, noise exposure, cold water diving	Manage risk factors for AV while diving; AV did not cause any critical situations; theoretical risk remains

Appendix (cont.)

First Author	LOE	*n*	Study design	Methods	Results	Conclusions/comment
Uzun[26]	4	29	Retrospective case series	Survey, otoscopy, 9-step inflation/deflation tympanometry, ET function	Previous OM present in ¾ of AV divers; 14% of divers experienced AV	Risk factors for AV include previous history of OM and impaired ET function
Inner ear						
Cantais[45]	4	101	Case control, DCS/ non-DCS divers	Transcranial Doppler	R/L shunt associated with increased incidence of cerebral DCI	Paradoxical emboli may be potential mechanism of cochleovestibular symptoms/ cerebral DCI
Edmonds[37]	4	50	Consecutive case series IEBt	Clinical manifestations, audiometry and treatment	17/50 Teed grade 0	Absence of TM haemorrhage does not exclude diagnosis of IEBt
Harill[71]	5	231	Survey	Survey of otologists re: activity restrictions post stapes surgery	>50% recommend no diving; 32% had experience with postop Bt	No correlation between frequency of barotrauma reported and activity restriction
House[70]	5	22	Survey	22 divers post stapedectomy	3 otalgia on descent; 1 tinnitus; 1 transient vertigo; 1 PLF; no complaints related to labyrinthine injury	Stapedectomy does not increase the risk of IEBt in divers; dry ear precautions for 3/52, diving after 1/12 postop; ensure no ETD
Klingmann[2]	4	46	Retrospective case series	Review of divers treated for inner ear DCS/ barotrauma	Median latency to HBOT 10 hrs; 83% DCS patients had a R/L shunt	Divers with inner ear DCS associated with R/L shunting must undergo sonography
Klingmann[41]	3b	34	Retrospective non-consecutive cohort	Divers with IEDCS were analysed re: symptomatology	All had vertigo; 40% had hearing loss; symptoms within 120 min of ascent; 73% had a R/L shunt	Vestibular symptoms predominate in IEDCS, likely due to the decreased tissue washout relative to the cochlea
Parell[40]	3b	20	Retrospective cohort, previous IEBt	Serial audiometry	No divers suffered a secondary bout of IEBt despite regular diving	Divers who suffer IEBt may continue to dive
Shupak[33]	3b	9	Retrospective case series	Cases of IEDCS/IEBt reviewed over 2 years	IEBt 5/9 divers; exploratory tympanotomy in 2/5; IEDCS 4/9, 7 underwent recompression	Consider IEDCS even when diving within no-stop limits; initial observation in IEBt, surgery if deterioration
Shupak[30]	4	2	Retrospective case series	Cases of IEBt were reviewed	PLF repair was successful; patients had anatomic risk factors on CT scan	CT temporal bone scans should be performed in IEBt to rule out anatomic risk factors
Shupak[33]	3b	25	Case-control series	PTA, ENG and VOR and SHA	PTA hearing threshold higher and decreased VOR phase values in divers	Slightly lower VOR values may represent adaptive underwater opticokinetic changes
Shupak[43]	4	11	Retrospective consecutive case series	Audiometry, ENG, posturography, rotatory chair	10 IEDCS and 4 IEBt had cochleovestibular deficits	IEDCS has high risk for inner-ear sequelae even with HBOT; deficits common even if asymptomatic
Tal[47]	4	3	Retrospective case series	Review of IEDCS cases	Patients with IEDCS require HBOT	If doubt over diagnosis of IEDCS, recompression should still be undertaken
Wong[39]	4	8	Non-consecutive case series	Report on complaints and management of inner-ear symptoms in divers	See individual aetiologies highlighted in case series	Careful history and physical examination will help distinguish between IEBt and IEDCS

Appendix (cont.)

First Author	LOE	*n*	Study design	Methods	Results	Conclusions/comment
General articles						
Klingmann[14]	2b	306	Retrospective cohort analysis	Chart review of anatomical sub-site/symptoms	8% external ear; 46% middle ear; 18% inner ear; 17% nose, paranasal sinuses; 8% DCS	Female divers significantly more affected by ETD; CRS associated with higher number of dives
Beckett[7]	5	770	Internet survey of divers	Risk behaviours and safety practices	DCS symptoms in 53%; OE in approx. 50% divers	Injury more common in non-certified divers
Hearing						
Goplen[17]	4	67	Prospective cohort study	Audiometry, ENG, posturography; 3/6 year follow up	Occupational SNHL in commercial divers; no change in ENG or posturography	No contribution of diving frequency to high frequency hearing loss; no evidence of permanent vestibular loss
Klingmann[29]	3b	123	Cross-sectional control comparison	PTA, tympanometry and otoscopy of sport. vs professional divers	No differences detected	No hearing loss among rec divers; professional divers risk occupational NIHL
Molvaer[27]	2b	116	Prospective cohort	Audiometry of professional divers over 6 years	Diver's hearing deteriorated faster than non-divers at comparable age	Noise exposure during commercial diving likely cause of hearing loss
Taylor[28]	3b	16	Retrospective cohort	Audiometry	No significant differences in audiological testing	Recreational divers not at increased risk of hearing loss
Sea sickness						
Cooper[53]	2b	1350	Prospective cohort on ocean liner	Epidemiologic study of sea sickness	Odds ratio for seasickness: female sex 2.95; young age 0.99; non crew 19.87	Sea sickness more common in women, may share common pathophysiology with migraine
Eden[57]	1b	25	RCT of naval cadets	Experimental group given positive self-efficacy/'verbal placebo' during 5-day cruise	Experimental cadets reported less seasickness; rated as better performers	Seasickness may be a self-fulfilling prophecy, and may be reduced with 'verbal placebo'
Gahlinger[51]	2b	260	Prospective cohort on cruise	Epidemiologic data and cabin location in ship recorded	Risk of motion sickness associated with age and sex	Location of cabin not associated with likelihood of motion sickness
Gil[59]	1b	76	RCT with crossover	Navy crew given transdermal scopolamine and cinnarizine, follow up questionnaires	Scopolamine more effective, less drowsiness, preferred agent, 41 vs 12%	Scopolamine patch should be considered drug of choice for treatment of seasickness
Williams[61]	1b	24	RCT	Dexterity, arithmetic, sentence comprehension tested, varying depth and scopolamine dose	Dexterity and comprehension impaired at depth; no effects from scopolamine	Transdermal scopolamine during dive operations may be suitable; perform field testing
MdDS						
Dai[66]	5	5	Prospective	Videonystagmography and roll chair	Adaptation of the angular VOR generated through velocity storage	Central maladaptation of the VOR implicated in MdDS
Gordon[65]	2b	234	Retrospective cohort	Survey of MdDS among healthy crew members	73% had MdDS symptoms; <6 h in 93%	Transient MdDS common and expected among crew members

Appendix (cont.)

First Author	LOE	*n*	Study design	Methods	Results	Conclusions/comment
Animal studies						
Antonelli[15]	5	16	Prospective case control animal model	Guinea pig stapedectomy model; consecutive hyperbaric dives, electrocochleography and hair cell counts	Middle ear barotrauma in 8 stapedectomy ears vs. 5 controls; no difference in hair cell counts	No predisposition to cochlear sequelae post stapedectomy in guinea pig model of barotrauma
Arieli[64]	5	26	Prospective case control animal model	Rats given cinnarizine/control O_2; exposure protocols to explore CNS toxicity	Latency to first electrical discharge on EEG increased at high O_2 pressures	Cinnarizine does not increase risk of CNS O^2 toxicity
Bitterman[63]	5	36	Prospective case control animal model	EEGs and HR measured to test interaction of scopolamine with HBO	No difference in latent period between control and scopolamine rats	Scopolamine does not alter hyperoxic seizures
Landolt[42]	5	100	Case control animal model	Histology of squirrel monkey temporal bones	Histologic vestibular apparatus deficits post decompression exposure	Neo-ossification within otic fluid spaces post DCS could cause permanent deficit to diver

Conclusions

Scuba diving has a significant potential for complications in the external, middle and inner ear. Otolaryngologists and clinicians with an interest in dive medicine should have a keen understanding of the pathophysiology, treatment and fitness-to-dive implications of diving-related disorders of the head and neck. The recommendations within this review are intended to supplement good clinical judgment, and should be applied within the context of each individual patient's circumstance. Overall, there is a need for more high-quality research on diving-related head and neck pathology. Research in this area can lend insight into the pathophysiology of barotrauma and DCS while improving patient care and decreasing dive-related morbidity.

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