Diving-related otological injuries

Initial assessment and management

Lyndon Nofz, Jemma Porrett, Nathan Yii, Nadine De Alwis

Background
Scuba diving–related otological injuries comprise the majority of diving-related incidents that present to general practitioners (GPs). Correct diagnosis and management are key to prevent permanent hearing loss and vertigo.

Objective
The aim of this article is to increase awareness of the pathophysiology of otological diving injuries and provide an approach to initial assessment and treatment, as well as to highlight particular circumstances in which onward referral is required.

Discussion
Accurate diagnosis and treatment of diving-related otological injuries by GPs can have profound positive effects on a patient's long-term outcomes. Complete otolaryngological assessment in those who have previously had a dive-related injury is critical to ensure patient safety prior to recommencing scuba diving.

SCUBA DIVING, during which a diver uses a self-contained underwater breathing apparatus (scuba), is a popular recreational activity resulting in frequent presentations to the general practitioner (GP). Incorrect diagnosis and management of otological diving injuries can lead to significant morbidity including chronic vestibulopathy and hearing loss. As many patients present to the GP with diving-related concerns, it is pertinent that treating clinicians are aware of the common presentations, initial treatment and prevention of such injuries.

Epidemiology

Diving-related otological injuries account for 65–72% of all diving-related presentations to practitioners.[1,2] Middle ear barotrauma (MEB) is most common, accounting for nearly 50% of presentations.[2] Decompression sickness (DCS) is one of the most severe diving-related complications and has an Australian incidence rate of 10 per 100,000 dives.[3] Inner ear DCS (IEDCS) is rare, with an estimated recreational diving incidence rate of 0.01–0.03%.[4]

Pathophysiology – A lesson in physics

Otological diving injuries occur as a result of the effect of ambient pressure changes on the ear structures. At sea level the atmospheric pressure is 1 atmosphere (atm). For every 10 m a diver descends below this level, the ambient pressure increases by 1 atm. For most purposes, atmospheric gas is made up of 21% oxygen and 79% nitrogen, similar to compressed gases found in a scuba tank.

Two important laws of physics govern the physiology associated with pressure changes during scuba diving. Boyle's Law ($P_1V_1 = P_2V_2$) dictates that the pressure and volume of a gas at a constant temperature are inversely proportional. As the diver descends, ambient pressure increases. The middle ear, being an air-filled space, correspondingly reduces its volume by 50% in the first 10 m of descent (Figure 1). If equalisation does not occur, then a vacuum is created, placing pressure on the tympanic membrane and round and oval windows (the flexible walls of this space). This accounts for direct pressure–related ear injuries during diving.

As ambient pressure increases, so does the partial pressure of each of the gases within the environment. Henry's Law dictates that as the partial pressure of a gas increases, a greater amount becomes dissolved in surrounding liquids. During uncontrolled ascent, the reduction in pressure causes release of dissolved gas nitrogen bubbles within tissues or blood that can lead to cell injury and hypoxia, causing the symptoms of DCS.

External ear pathology

Otitis externa

Otitis externa is common among those who spend considerable amounts of time submerged underwater. Prolonged exposure of the ears to water results

in a change in canal pH and skin maceration, damaging the primary skin barrier and promoting infection.[5] *Pseudomonas aeruginosa* is the organism most responsible for acute otitis externa following diving.[6] The principles of treatment remain the same as for non-divers, with dry ear precautions, aural toilet and topical antimicrobial therapy.

Exostoses

Exostoses are bony outgrowths of the medial ear canal. They commonly occur in divers in response to chronic cold water or wind exposure that triggers refrigeration periosteitis.[7] Bony canal stenosis can result in water and wax retention, predisposing to otitis externa. When severe, complete occlusion of the canal results in a conductive hearing loss. Furthermore, unilateral occlusion can lead to an asymmetric caloric stimulus underwater, leading to vertigo.[8] Promoting the use of appropriately fitted wetsuit hoods can help to prevent this progression. Referral to an otolaryngologist for consideration of a canalplasty may be necessary in severe or symptomatic cases.

External canal barotrauma

External canal barotrauma can occur as a result of occlusion of the ear canal by exostoses, impacted cerumen and tight wetsuit hoods. An air-filled space between the occlusion and tympanic membrane is created, and negative pressure is generated on descent, leading to swelling and haemorrhagic blistering of the canal wall. Treatment consists of analgesia and topical steroid ear drops.[9] External canal barotrauma can be reduced by avoiding tightly fitted hoods and cotton bud use for wax removal. Aural toilet is recommended for persistent wax impaction.

Middle ear pathology

Middle ear barotrauma

MEB occurs when the diver cannot or does not regularly equalise on descent or ascent. This may be due to inexperience or eustachian tube dysfunction (ETD) secondary to rhinosinusitis, allergy or nasopharyngeal obstruction. As external pressures rise on descent, middle ear volumes decrease, creating a vacuum. Serous or haemorrhagic effusion and tympanic membrane rupture can result secondary to the negative pressure.[10] Conversely, during ascent, middle ear volume expands as pressure decreases. Normally this is passively released through a functional eustachian tube. However, with ETD, volume expansion can create bulging of the tympanic membrane and perforation.

Symptoms include dullness or pain in the ear and hearing loss. Tympanic membrane perforation during a dive can cause severe vertigo from caloric stimulation of cold water in the middle ear. If nausea and vomiting ensues, this can be fatal because of obligate mouth breathing during scuba diving.[11] MEB can be graded using the modified Teed classification,[12] which is useful in guiding flying and diving restrictions post-injury (Table 1).

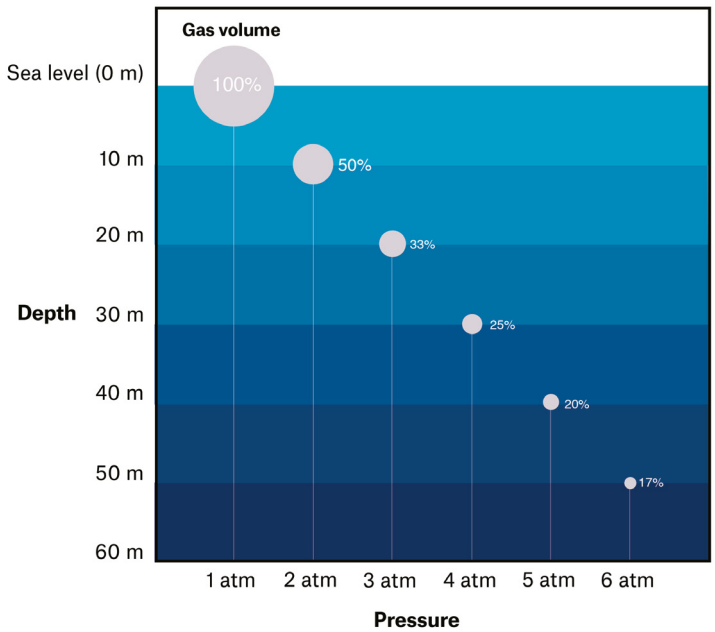

Figure 1. Boyle's law dictates that as pressure increases below the water surface, the volume of closed spaces decreases.

Table 1. Modified Teed classification[12] for middle ear barotrauma*

Grading	Otoscopic findings	Estimated return to dive
Grade 0	Normal tympanic membrane	Seven to 10 days (complete resolution)
Grade 1	Tympanic membrane erythematous/ inflamed	
Grade 3	Gross haemorrhage of the tympanic membrane	Six weeks (blood reabsorption)
Grade 4	Extensive free blood in middle ear with bubbles visible behind tympanic membrane (haemotympanum)	
Grade 5	Perforation of the tympanic membrane	Three months (healed perforation)

**Grade 2 has not been included in this modification*

Management involves exclusion of inner ear barotrauma, short-term use of nasal decongestants and/or intranasal steroid sprays and antibiotics for any secondary infection.[11] Diving should not recommence until all symptoms have resolved, tympanic membrane perforations have healed and equalisation is possible. In cases of persistent tympanic membrane perforation, surgical repair may be necessary.

Inner ear pathology

Inner ear barotrauma

Rupture of the round or oval windows creates a perilymphatic fistula (PLF), which can cause sensorineural hearing loss (SNHL), vertigo and tinnitus. Two mechanisms have been postulated (Figure 2).[13,14]

Patients may present with an unsteady gait and ear examination findings similar to MEB, though the ear can also appear completely normal. Tuning fork assessment can be unreliable, thus formal audiometry is required. Fistula testing by applying abrupt tragal pressure to the affected ear or air insufflation by pneumatic otoscopy will elicit nystagmus if positive. However, a negative fistula sign does not exclude PLF.[15]

Management for PLF is initially conservative with strict bed rest, head elevation, high-dose oral steroids, stool softeners, anti-emetic medications and avoidance of straining.[16] Audiometry should be performed daily to monitor for improvement. Progressive SNHL or worsening vestibular function should prompt urgent referral to an ear, nose and throat (ENT) surgeon for surgical repair, which should ideally be performed within 10 days of injury.[17] Divers should undergo a fitness to dive assessment by an otolaryngologist or dive medicine physician prior to recommencing diving.[8]

Inner ear decompression sickness

Two main theories exist for inner ear DCS. The first is formation of inert gas bubbles within the microvasculature or otic fluids of the vestibulocochlear apparatus on ascent. The second is arterial gas emboli shunted from the venous system (ie patent foramen ovale).[11,13,18] Patients commonly present with vertigo, nausea and vomiting, although hearing loss and other central nervous system symptoms frequently occur.[19] Examination usually reveals a normal external canal and tympanic membrane. Tuning forks may reveal SNHL, and nystagmus is usually present.

It is important for inner ear DCS to be differentiated from inner ear barotrauma as treatment is markedly different (Table 2).[20] Treatment of inner ear DCS includes early recompression with hyperbaric oxygen, preferably within five hours of symptom onset. In the general practice setting, it is recommended that administration of 100% oxygen and intravenous saline or oral fluids be provided. Urgent referral to the nearest centre with a hyperbaric chamber is critical to long-term prognosis.[4]

If there is doubt regarding whether the diagnosis is inner ear barotrauma or DCS, patients should be treated for DCS as it is more severe and can lead to persistent vertigo and ataxia.

Prevention and fitness to dive

Adequate ventilation of the middle ear is essential to the prevention of otological injury. Divers with conditions predisposing them to ETD should avoid diving, and those who are unable to autoinsufflate the ear underwater should immediately abort the dive. Controlling the rate of ascent and taking decompression stops will reduce the risk of DCS. To avoid nitrogen excess, flying is also not recommended within 24 hours following diving.[13] The use of oral or topical decongestants is discouraged to treat ETD or sinusitis prior to diving as their effects may wear off while underwater, leaving the diver in danger.[13]

Fitness to return to diving depends on residual symptoms and ongoing pathology. Persisting neurology, especially affecting the vestibular system, is associated with high risk. An estimated 90% of patients with previous diving-related vestibular dysfunction have ongoing long-term deficits necessitating thorough assessment prior to continuing scuba diving.[18] Some authors recommend full vestibulocochlear assessment and exclusion of a right-to-left vascular shunt in those who have previously had inner ear DCS.[6,18]

Return to diving is possible following otological surgery. Divers should wait three months following tympanoplasty, with diving permissible once the tympanic membrane has healed and function returned.[21] Limited evidence exists for return to diving following stapes surgery or canal wall down mastoidectomy and while some authors do advocate this,[22,23] in our opinion, the potential lethal risks of experiencing vertigo and vomiting underwater far outweigh any benefits. We recommend that each patient undergoes a full assessment by an otolaryngologist and return to diving is judged on a case-by-case basis and any recommendations are clearly documented.

Table 2. Characteristic features differentiating inner ear barotrauma and inner ear decompression sickness

Inner ear barotrauma	Inner ear decompression sickness
Conductive or mixed hearing loss	Sensorineural hearing loss
Descent or ascent	Ascent
Cochlear symptoms (ie hearing loss predominates)	Vestibular symptoms predominant; right sided
History of forced or difficult Valsalva manoeuvre	Not associated with a history of eustachian tube dysfunction
Low-risk dive profile	Dive profile >15 m, technical diving (helium mixtures), multiple dives over a short period
Isolated inner ear symptoms	Other neurological/dermatological manifestations

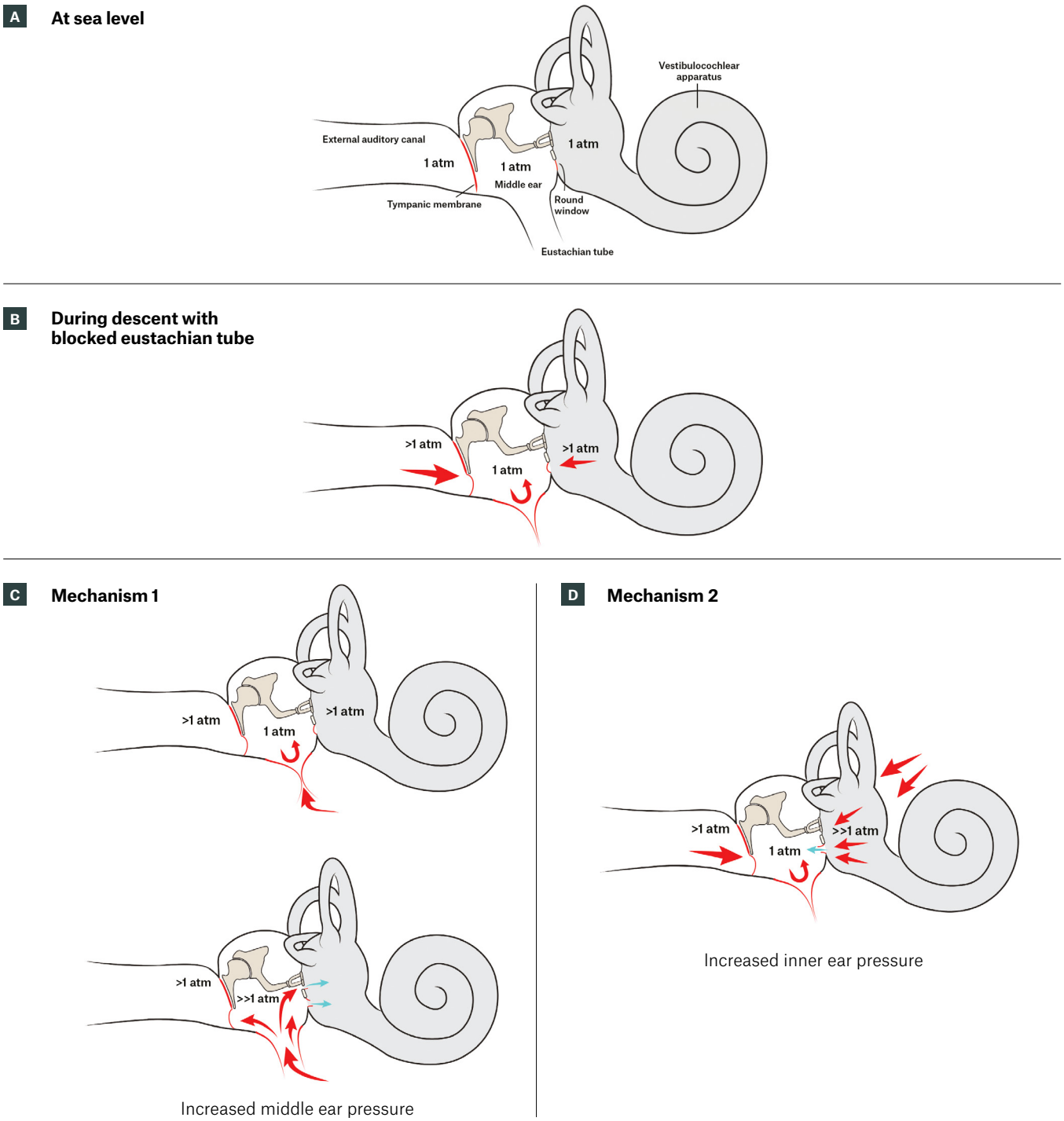

Figure 2. Inner ear barotrauma

A. The inner ear at sea level; **B.** The inner ear during descent with blocked eustachian tube; **C.** Mechanism 1 suggests a forced Valsalva manoeuvre against a blocked eustachian tube with sudden opening and rush of air into the middle ear. This pressure can cause stapes footplate dislocation and implosion of the oval or round window; **D.** Mechanism 2 suggests a forced Valsalva manoeuvre against a blocked eustachian tube with increased cerebrospinal fluid pressures transmitted through the cochlear aqueduct and otic fluids leading to round window explosion.

Conclusion

Patients may present to the GP with a number of injuries related to scuba diving. The ability of the GP to understand the physiology, recognise pathologies and initiate management can greatly influence the patient's long-term outcomes and recovery. Furthermore, fitness to dive certifications for people with a history of ENT conditions and those who have previously had a dive-related injury are mandatory in Australia.

Authors

Lyndon Nofz MBBS, MSc, Principal House Officer, Department of Otolaryngology Head and Neck Surgery, Cairns Hospital, Qld; Associate Lecturer, University of Queensland, Qld. lyndon.nofz@health.qld.gov.au

Jemma Porrett MBBS (Hons), Principal House Officer, Department of Otolaryngology Head and Neck Surgery, Cairns Hospital, Qld

Nathan Yii MBBS, Principal House Officer, Department of Otolaryngology Head and Neck Surgery, Cairns Hospital, Qld

Nadine De Alwis MBBS, FRACS (Otolaryngology Head and Neck Surgery), Visiting Medical Officer, Department of Otolaryngology Head and Neck Surgery, Cairns Hospital, Qld

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References

1. Roydhouse N. 1001 disorders of the ear, nose and sinuses in scuba divers. Can J Appl Sport Sci 1985;10(2):99–103.
2. Klingmann C, Praetorius M, Baumann I, Plinkert PK. Barotrauma and decompression illness of the inner ear: 46 cases during treatment and follow-up. Otol Neurotol 2007;28(4):447–54. doi: 10.1097/MAO.0b013e318030d356.
3. Lippmann J. Review of scuba diving fatalities and decompression illness in Australia. Diving Hyperb Med 2008;38(2):71–78.
4. Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. Lancet 2011;377(9760):153–64. doi: 10.1016/S0140-6736(10)61085-9.
5. Russell JD, Donnelly M, McShane DP, Alun-Jones T, Walsh M. What causes acute otitis externa? J Laryngol Otol 1993;107(10):898–901. doi: 10.1017/s0022215100124739.
6. Azizi MH. Ear disorders in scuba divers. Int J Occup Environ Med 2011;2(1):20–26.
7. Ito M, Ikeda M. Does cold water truly promote diver's ear? Undersea Hyperb Med 1998;25(1):59–62.
8. Livingstone DM, Smith KA, Lange B. Scuba diving and otology: A systematic review with recommendations on diagnosis, treatment and post-operative care. Diving Hyperb Med 2017;47(2):97–109. doi: 10.28920/dhm47.2.97-109.
9. Becker GD, Parell GJ. Barotrauma of the ears and sinuses after scuba diving. Eur Arch Otorhinolaryngol 2001;258(4):159–63. doi: 10.1007/s004050100334.
10. O'Neill OJ, Frank AJ. Ear barotrauma. Treasure Island, FL: StatPearls Publishing, 2020. Available at www.ncbi.nlm.nih.gov/books/NBK499851 [Accessed 23 March 2020].
11. Lechner M, Sutton L, Fishman JM, et al. Otorhinolaryngology and diving – Part 1: Otorhinolaryngological hazards related to compressed gas scuba diving: A review. JAMA Otolaryngol Head Neck Surg 2018;144(3):252–58. doi: 10.1001/jamaoto.2017.2617.
12. Beuerlein M, Nelson RN, Welling DB. Inner and middle ear hyperbaric oxygen-induced barotrauma. Laryngoscope 1997;107(10):1350–56. doi: 10.1097/00005537-199710000-00011.
13. Mallen JR, Roberts DS. SCUBA medicine for otolaryngologists: Part I. Diving into SCUBA physiology and injury prevention. Laryngoscope 2020;130(1):52–58. doi: 10.1002/lary.27867.
14. Bove AA. Diving medicine. Am J Respir Crit Care Med 2014;189(12):1479–86. doi: 10.1164/rccm.201309-1662CI.
15. Chu H, Chung WH. Images in clinical medicine. Perilymph fistula test. N Engl J Med 2012;366(4):e8. doi: 10.1056/NEJMicm1010568.
16. Osetinsky LM, Hamilton GS 3rd, Carlson ML. Sport injuries of the ear and temporal bone. Clin Sports Med 2017;36(2):315–35. doi: 10.1016/j.csm.2016.11.005.
17. Park GY, Byun H, Moon IJ, Hong SH, Cho YS, Chung WH. Effects of early surgical exploration in suspected barotraumatic perilymph fistulas. Clin Exp Otorhinolaryngol 2012;5(2):74–80. doi: 10.3342/ceo.2012.5.2.74.
18. Shupak A, Gil A, Nachum Z, et al. Inner ear decompression sickness and inner ear barotrauma in recreational divers: A long-term follow-up. Laryngoscope 2003;113(12):2141–47. doi: 10.1097/00005537-200312000-00017.
19. Klingmann C. Inner ear decompression sickness in compressed-air diving. Undersea Hyperb Med 2012;39(1):589–94.
20. Rozycki SW, Brown MJ, Camacho M. Inner ear barotrauma in divers: An evidence-based tool for evaluation and treatment. Diving Hyperb Med 2018;48(3):186–93. doi: 10.28920/dhm48.3.186-193.
21. Mallen JR, Roberts DS. SCUBA medicine for otolaryngologists: Part II. Diagnostic, treatment, and dive fitness recommendations. Laryngoscope 2019;130(1):49–64. doi: 10.1002/lary.27874.
22. House JW, Toh EH, Perez A. Diving after stapedectomy: Clinical experience and recommendations. Otolaryngol Head Neck Surg 2001;125(4):356–60. doi: 10.1067/mhn.2001.118183.
23. Sim RJ, Youngs RP. Otolaryngological requirements for recreational self-contained underwater breathing apparatus (SCUBA) diving. J Laryngol Otol 2007;121(4):306–11. doi: 10.1017/S0022215106001976.

correspondence **ajgp@racgp.org.au**