

Review articles

Diving-related ear, nose, and throat disorders: a structured review using a phase-of-dive framework

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Abstract

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Introduction: The aim of this review was to synthesise evidence on ear, nose, and throat (ENT) and related head and neck complications in scuba divers, with emphasis on mechanisms of injury, dive phase-specific risk patterns, and implications for clinical management and fitness-to-dive decision-making.

Methods: A structured literature search of PubMed, Embase (Ovid), and the Cochrane Library identified systematic reviews, clinical trials, cohort studies, and case series reporting clinically relevant ENT outcomes in scuba divers. Animal studies, freediving studies, and papers focused solely on non-ENT complications were excluded. Forty analytic studies met inclusion criteria, including large cross-sectional surveys and multiple case series of inner ear decompression sickness. Study characteristics and levels of evidence were summarised, and findings were integrated into a phase-of-dive framework (descent, bottom time, ascent).

Results: Across included studies, middle-ear barotrauma was the most prevalent diving-related ENT injury, particularly during descent. Inner ear barotrauma and inner ear decompression sickness were less common but carried a substantially higher risk of persistent auditory or vestibular deficit. Additional reported conditions included sinus barotrauma, otitis externa and external auditory canal exostoses, temporomandibular joint symptoms, odontobarotrauma, and marine-exposure injuries. Evidence concerning postoperative states, following operations such as stapedectomy, tympanoplasty, mastoidectomy, and cochlear implantation, was limited, heterogeneous, and often contradictory, with few data to guide return-to-dive clearance.

Conclusion: Scuba diving-related ENT complications appear to be diverse, influenced by dive phase, and shaped by individual anatomical and physiological factors. A phase-aware framework offers clinicians a useful way to contextualise symptom onset and guide prevention and counselling, particularly for divers with prior ear or nasal surgery or implanted devices. Prospective studies are needed to refine screening practices and establish evidence-based return-to-dive recommendations.

Introduction

Scuba diving is a globally popular recreational and professional activity, with an estimated six million active divers; millions more certified but not diving regularly;¹ and (as estimated in 2017) more than 100,000 new divers qualifying annually.² Diving exposes participants to unique environmental stressors, particularly pressure differentials across air-containing anatomical spaces.^{2,3} As a result, the head and neck region accounts for over 80% of all diving-related injuries, with up to 65% involving the ear alone.^{2,4,5} Diving-related pathology includes a wide range of ear, nose and throat (ENT) and adjacent complications, ranging from otic barotrauma, alternobaric vertigo, sinus barotrauma,^{3,4,6} and inner ear decompression illness.^{2,7} Even simple otitis externa is highly prevalent among divers and can compromise diving fitness,^{2,8} as even seemingly minor

complications can significantly impair a diver's situational awareness and may indirectly increase the risk of other serious dive-related injuries.^{8,9}

Despite this burden, awareness of available clinical guidance remains limited. A UK survey of 790 scuba divers found that 79% reported ear disorders since learning to dive and 40% met validated criteria suggesting Eustachian tube dysfunction, yet only 27% were aware of published ear health guidance for divers.¹⁰ This gap between the prevalence of diving-related ENT complaints and diver awareness of evidence-based preventive strategies underscores the clinical relevance of a comprehensive, accessible overview of this topic.

A growing number of reviews have addressed ENT pathologies in isolation or in subsets. These include

studies focused on otologic injury,² rhinologic and oromaxillofacial complications,⁶ and differentiation of inner ear decompression illness from barotrauma.³ However, they are often narrowly scoped, reflecting anatomical or physiological silos rather than a comprehensive clinical picture. Therefore, this review seeks to deliver a clinically oriented overview of ENT disorders organised by dive phase. This structure mirrors how divers commonly report the onset of their symptoms, thereby improving clinical relevance and practical applicability.

Methods

A structured literature search was conducted in PubMed, Embase (Ovid), and the Cochrane Library, using MeSH headings and free-text keywords related to scuba diving, with terms covering otologic, rhinologic, sinus, pharyngeal, laryngeal, and orofacial conditions relevant to diving-related injury. Forty studies met all inclusion criteria and were included in this structured review. The database search was supplemented by hand-searching and the documented search string is presented in Figure 1. Additional mechanistic and physiological references are cited in the text where relevant to contextualise findings but were not included in the evidence synthesis. Study methodological quality was assessed based on study design, sample size, potential for bias, and reporting completeness; these assessments are summarised in *[Supplementary Table 1](#). No validated risk-of-bias instrument (e.g., ROBINS-I, GRADE) was formally applied given the heterogeneity of study designs.

AI-assisted writing tools were used for language refinement only. Literature identification, selection, interpretation, and synthesis, as well as figure illustration and data representation, were performed by the author.

Findings are organised by dive phase (descent, bottom time, ascent) to highlight mechanisms of injury, contextualise associated risk factors and outcomes, and offer practical insights for prevention and management.

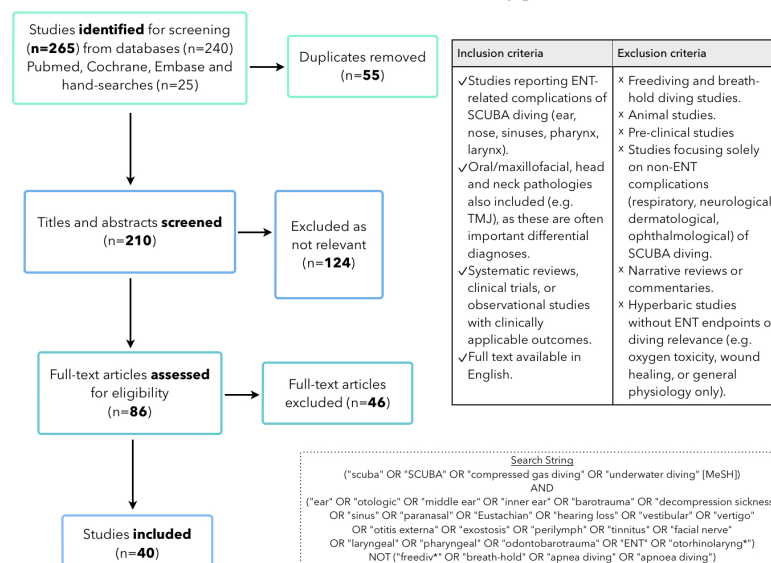
Results

EVIDENCE BASE AND STUDY CHARACTERISTICS

The available evidence on diving-related ENT disorders is unevenly distributed across condition types: barotrauma (particularly middle ear barotrauma) is extensively studied, while inner ear decompression sickness, fitness-to-dive after otological surgery, and long-term effects of repetitive diving on the head and neck are comparatively under-researched. Much of the published literature comprises small observational cohorts and case reports (see *[Supplementary Table 1](#)) and focuses on barotrauma, reflecting both the rarity of some of the other presentations and the logistical difficulty of studying divers prospectively. This heterogeneity highlights the need for larger, standardised prospective studies that can clarify the natural history and optimal management of inner ear decompression sickness; establishing evidence-based return-to-dive guidance following inner ear barotrauma, perilymphatic fistula repair,

***Note:** Supplementary Table 1 is available online on our website: <https://www.dhmjournal.com/index.php/journals?id=437>

Figure 1
Literature search and screening process



and otological implant surgery; and characterising the long-term audiological effects of cumulative diving exposure with appropriate control groups.

MECHANISMS OF INJURY

Several pathophysiological processes contribute to the spectrum of ENT complications encountered in divers. These are summarised in Figure 2.

Barotrauma and Boyle's law

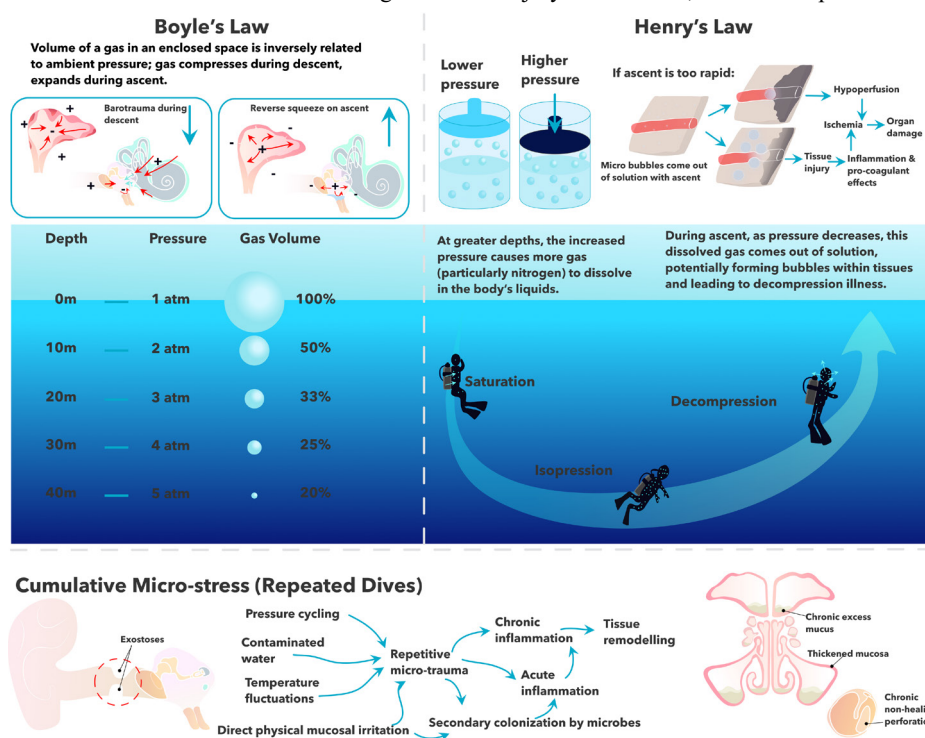
Barotrauma arises due to pressure differentials across anatomical compartments, including both gas-filled and fluid-filled spaces, during changes in ambient pressure. According to Boyle's law, the volume of gas within a closed cavity is inversely proportional to ambient pressure, leading to gas compression during descent and expansion during ascent. Failing to equalise these pressure changes adequately can result in significant mechanical stress: negative pressure on descent leads to mucosal oedema, haemorrhage, and tissue deformation, while positive pressure on ascent may cause outward distention and rupture of affected tissues.⁶ The severity and nature of barotrauma depend primarily on the compliance and ventilation of affected anatomical compartments and the degree and duration of pressure imbalance. At the tissue level, these pressure changes can lead to compromised vascular integrity, ischaemia, inflammatory responses, and direct mechanical injury.^{2,7}

Decompression and Henry's law

Decompression sickness (DCS) presents with a wide spectrum of manifestations, ranging from mild skin or musculoskeletal symptoms to serious neurological involvement. It can also specifically affect the inner ear (IEDCS). During descent, increased ambient pressure results in a higher inspired partial pressure of inert gases, typically nitrogen, which dissolve into tissues proportional to depth, bottom time, individual tissue perfusion and diffusion characteristics (Henry's Law).⁷ Upon ascent, the ambient pressure reduction allows these inert gases to come out of solution and be eliminated via alveolar gas exchange. If the ascent occurs too rapidly, exceeding the body's ability to adequately eliminate dissolved gas, inert gas can form bubbles within the venous circulation and tissues.² The vestibular portion of the ear is vulnerable due to relatively slow gas washout and thus heightened susceptibility to gas supersaturation and consequent bubble formation or amplification of any small bubbles arriving in the arterial circulation.¹¹ Inert gas bubbles may directly interact with endothelial cells in the labyrinthine blood vessels, initiating an inflammatory cascade, coagulation activation, and subsequent hypoxic tissue damage; or alternatively, direct mechanical damage caused by microbubble formation within the endolymphatic and perilymphatic spaces may injure local microvessels and membranous structures.^{5,12} Additionally, arterialised venous bubbles traveling through a right-to-left cardiac shunt, such as a patent foramen ovale may embolise the labyrinthine artery, contributing to inner ear ischaemia.¹³

Figure 2

An illustrated overview of diving-associated injury mechanisms; atm – atmospheres



Physiological modelling has further quantified this selective inner ear vulnerability: nitrogen washout half-times have been estimated at 8.8 minutes for the inner ear versus 1.2 minutes for the brain following a simulated air dive, implying that the inner ear remains supersaturated with inert gas for substantially longer after surfacing.¹⁴ This creates a post-surfacing temporal window during which venous gas emboli traversing a right-to-left shunt may preferentially grow within inner ear tissue due to inward diffusion of supersaturated gas, whereas equivalent bubbles entering the brain microcirculation would not grow and would redistribute to the venous side. This provides a mechanistic explanation for both the selective vulnerability of the inner ear and the characteristic latency of IEDCS symptoms. The differential gas kinetics between the vestibular organ and the cochlea within the inner ear itself may further explain why vestibular presentations predominate in IEDCS: the cochlea has greater blood flow and smaller tissue volume, implying faster inert gas washout and a shorter window of susceptibility than the vestibular apparatus.¹⁵

Cumulative exposure and inflammatory remodeling

Repetitive diving exposure, particularly in cold water, can induce low-grade, chronic tissue stress that culminates in structural changes over time. Persistent moisture, temperature fluctuations, and pressure variations can predispose to otitis externa and, over time, to the formation of external auditory canal exostoses.¹⁶ Similarly, the tympanic membrane may thicken, atrophy, or develop non-healing perforations in seasoned divers, which manifests as hearing loss. Long-term auditory effects due to diving *per se* are controversial: although often implied. For example, in a 12-year prospective cohort of professional divers, high-frequency hearing thresholds deteriorated significantly at 4 and 8 kHz, with greater loss in divers performing more than ~650 lifetime dives, suggesting a cumulative effect.⁹ Chronic ET dysfunction, even in the absence of acute symptoms, can contribute to these sequelae over time.¹⁷

Similar inflammatory changes occur within the nasal and paranasal sinus mucosa. Acute sinusitis can arise following isolated episodes of sinus barotrauma or when ostial obstruction from transient mucosal edema traps pathogens, resulting in infection and inflammation. Chronic sinusitis (common among experienced divers, even in the absence of overt symptoms), the result of repeated mechanical stress, particularly when combined with environmental factors such as cold water or allergens, may gradually impair mucociliary clearance mechanisms and promote inflammatory remodeling and fibrosis of the mucosa.⁶

INJURIES BY PHASE OF DIVE

The physiological demands of scuba diving shift throughout every dive; understanding the phase-specific distribution of injuries aids in both diagnosis and targeted prevention strategies. However, it should be noted that many of the

pathologies described below can appear in multiple phases of a dive, given varying dive profiles. Important elements of the following narrative are depicted in Figure 3.

Descent phase injuries

The descent is often the most pressure-intensive phase of the dive and is thus characterised by various forms of barotrauma. As ambient pressure increases rapidly, air-filled cavities must be actively equalised to prevent negative pressure effects. Middle ear barotrauma (MEBt), as described earlier, is the most prevalent complication during this phase, consistent with cohort data demonstrating that ~11% of divers present with MEBt and 24% report Eustachian tube (ET) dysfunction.⁸ These figures, derived from a tertiary referral cohort, likely underestimate the true population burden. In the largest survey to date, 81% of 1,881 Finnish recreational divers reported having experienced MEBt at some point while diving, with 38% of those affected having used medications and 1% requiring ENT surgery.¹⁸ The strongest independent risk factor was poor subjective success with the Valsalva manoeuvre, followed by poor Toynbee success, highlighting equalisation technique as a primary modifiable target. A UK survey of 790 divers similarly found that 40% met validated criteria for ET dysfunction and 35% had aborted a dive due to ear problems.¹⁰

ET dysfunction is caused by inadequate ET opening, resulting (during descent) in tympanic membrane retraction, haemorrhage, or rupture (see Figure 4 for grading of MEBt).^{2,19} The risk is highest in novice divers or those diving with upper respiratory tract infections or allergic rhinitis, but it can and does affect divers of all experience levels.^{2,20} In a condensed four-day open-water certification course, 48 of 67 novice candidates (72%) developed MEBt at some point during training, with language of instruction showing no significant association with incidence, suggesting that comprehension of equalisation instructions alone is not the primary barrier.²¹ Repetitive diving compounds this risk: in a prospective study of 28 recreational divers performing 437 dives over six consecutive days, MEBt prevalence reached 36.5% by the final examination and increased with the number of dives per day, while greater experience was protective.²² Tympanometric data from the same cohort demonstrated a significant positive shift in middle ear pressure from day one to day three and a progressive decrease in compliance across the diving week, most pronounced in less experienced divers, thus indicating that the physiological burden of repetitive diving accumulates measurably even within a single trip. A consideration particularly relevant for liveaboard divers or those undertaking three to four dives daily.²³

To prevent MEBt, divers should descend slowly when possible and be trained in multiple gentle but effective equalisation techniques, such as the Toynbee or Frenzel maneuvers, rather than simply forcing Valsalva manoeuvres, which can increase the risk of inner ear damage as well (see Figure 5).^{2,3} Furthermore, pre-dive screening for Eustachian

Figure 3

Phase-of-dive distribution of ENT and related disorders in scuba divers. Disorders are mapped to the phases of descent, bottom, and ascent, illustrating when specific injuries most commonly occur

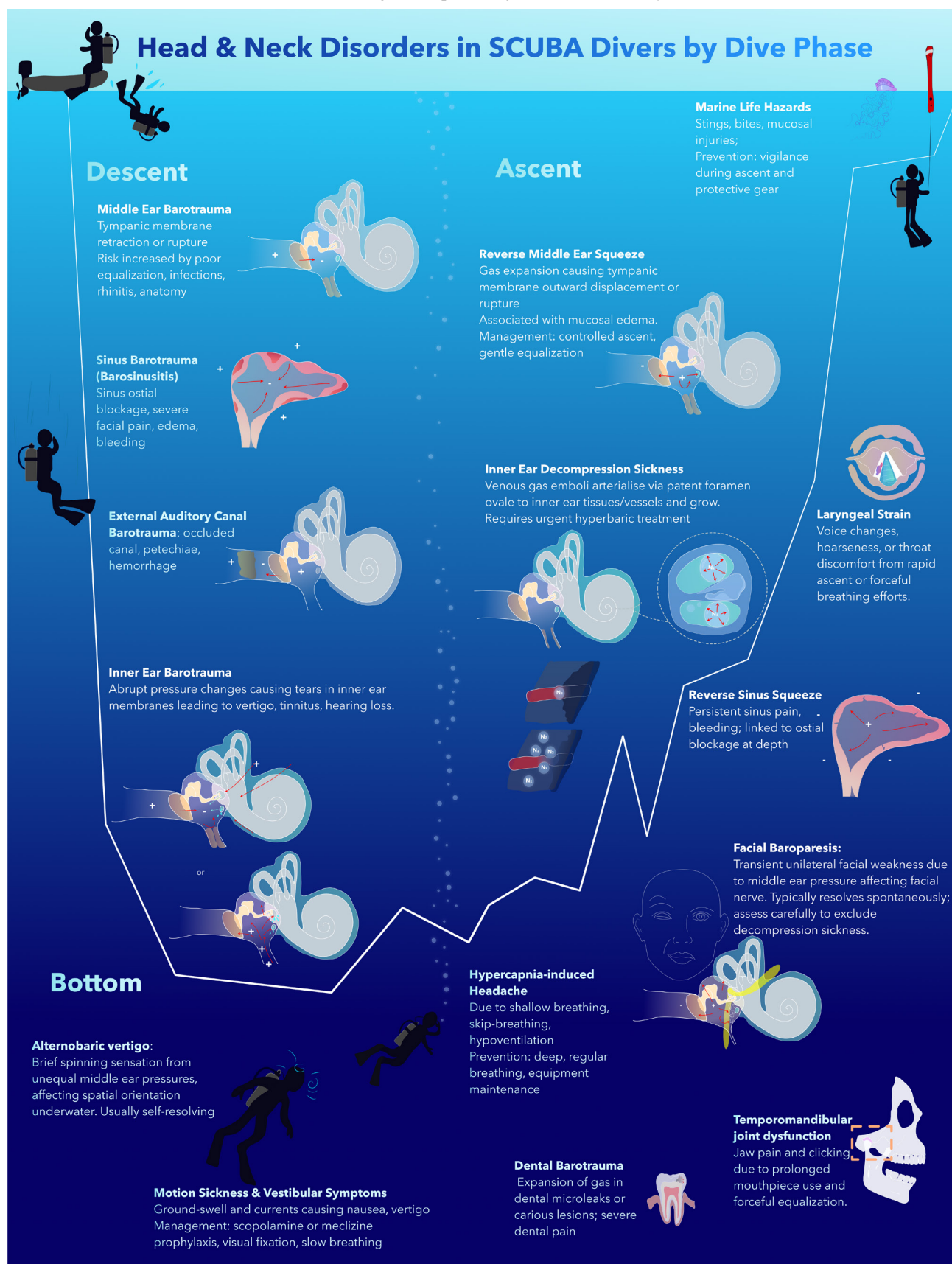
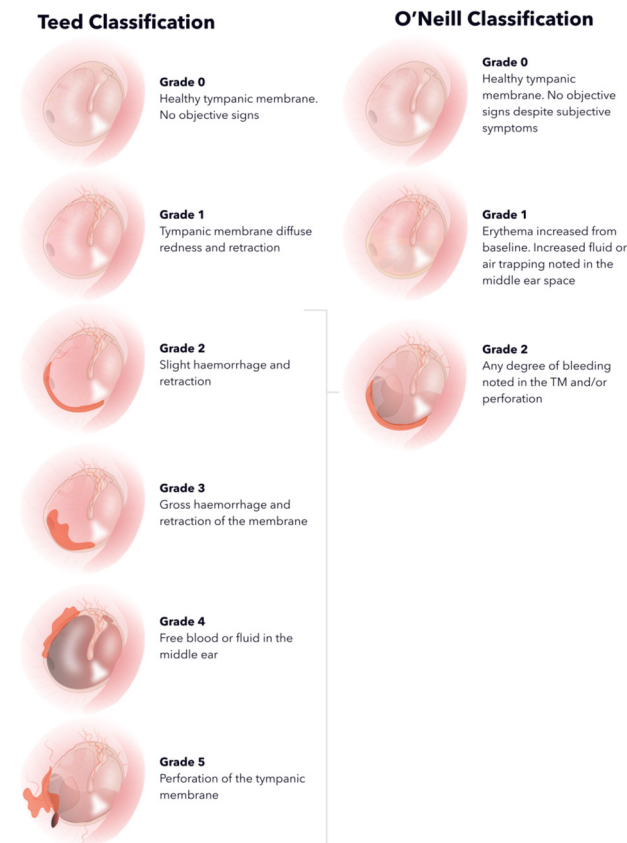


Figure 4

Clinical assessment of the tympanic membrane using Teed (grades 0–5) and O’Neill (grades 0–2) classifications. Management recommendations by grade include Teed 0–2 / O’Neill 1: nasal/oral decongestants, analgesia, and autoinflation techniques; advise delay of further diving until symptoms clear. Teed 3–4 / O’Neill 2: consider a short course of oral steroids, antibiotics if rupture or contamination present; avoid diving and obtain ENT follow-up. Teed 5 (tympanic membrane perforation): as above, plus possible tympanoplasty; heal time typically 4–12 weeks before resumption of diving



dysfunction and prophylactic nasal decongestants or antihistamines are advised for divers with known rhinitis, although evidence for benefit is limited.^{2,20,24} Objective pre-dive screening may complement subjective self-assessment: a nine-step tympanometric test battery combined with mastoid pneumatisation assessment achieved a positive predictive value of 86% for identifying military divers who subsequently developed symptomatic MEBt across 774 dives, suggesting a potential role for structured ET function testing before return to diving after injury.²⁵ However, immediate management of suspected barotrauma includes aborting the dive at the onset of significant pain or vertigo and seeking ENT evaluation if symptoms persist post-dive.²

Inner ear barotrauma (IEBt) can arise through abrupt pressure differentials between middle and inner ear spaces being transmitted through the oval or more commonly, round window, potentially resulting in tears of intra-labyrinthine structures such as Reissner’s membrane, or rarely,

rupture of the round window itself and thus resulting in a perilymphatic fistula. Clinical presentation typically involves sudden vertigo, acute tinnitus, hearing loss, or imbalance, usually noted during descent; however, it can occasionally occur during ascent as well. Cochlear symptoms (tinnitus and hearing loss) are significantly more common than vestibular symptoms in IEBt, a pattern that helps distinguish it from IEDCS, where vestibular presentations tend to predominate.^{3,5} Notably, divers attempting forceful Valsalva maneuvers, especially in the setting of delayed middle ear equalisation may inadvertently increase the risk of IEBt by creating an acute and intense pressure differential across labyrinthine membranes.¹² Perilymphatic fistula can also result from forceful equalisation on descent, and symptomatic audio-vestibular loss may appear hours after surfacing, thus making it difficult to identify precisely when injury occurred. Classic features are acute vertigo ‘blocked-water’ aural fullness, sensorineural hearing loss, and a nystagmus that beats away from the affected ear.

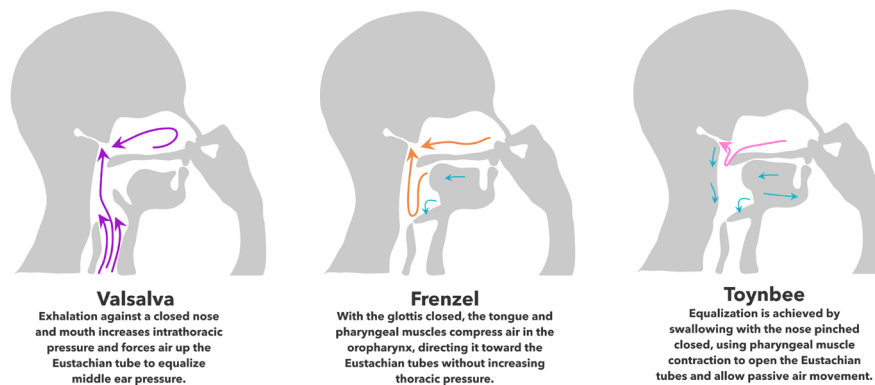
Management involves immediate cessation of diving, head elevation, stool softeners, and high-dose oral corticosteroids (prednisolone 1 mg·kg⁻¹ for five to seven days before tapering). Persisting or progressive symptoms beyond 48 h may warrant exploratory tympanotomy with fascia patching. Outcomes have not been intensively studied, but a 26-diver series showed residual deficits in 78%, suggesting IEBt can frequently result in permanent cochlear injury.¹³ Longer-term surgical outcome data are available from a series of 11 perilymphatic fistula cases following ear barotrauma, of which eight occurred after scuba diving. Of those explored surgically via an endaural approach, a cochlear fenestral fistula was visualised in seven with active perilymph leakage in six. After temporal fascia grafting, dizziness resolved promptly and early stable subtotal hearing recovery was achieved in seven patients at a median follow-up of 7.4 years, with tinnitus improving in four of six affected cases.²⁶ These findings support early surgical exploration when perilymph fistula is suspected and symptoms progress beyond 48 hours. Differentiating IEBt from IEDCS in the acute setting remains clinically challenging given overlapping cochleovestibular presentations. A systematic review of 302 manuscripts reported tinnitus in 73% and symptomatic vertigo in 46% of IEBt patients.²⁷

Prevention hinges on coaching divers in gentle equalisation and excluding return to diving until audiovestibular function and Eustachian-tube testing are completely normal.^{2,28} Long-term follow-up suggests that, with restored ET function and gentle equalisation, recurrent IEBt is uncommon in returning divers,²⁹ unless they have subtle anatomical vulnerabilities such as widened cochlear aqueducts or internal acoustic canals, underscoring the role of individual predisposition in injury risk.³⁰

Less frequently, external ear canal barotrauma can develop when an unvented air pocket between cerumen, exostoses, or a tight hood and the tympanic membrane creates a

Figure 5

Common middle-ear equalisation techniques used in scuba diving. The Valsalva maneuver is the most widely practiced but carries a higher risk of inner-ear barotrauma if performed too forcefully. The Frenzel and Toynbee maneuvers offer gentler alternatives, using controlled muscle contractions and swallowing actions to safely ventilate the middle ear without significantly elevating intrathoracic or intracranial pressure. Other techniques also exist and divers should ideally master multiple techniques to ensure reliable and safe equalisation across different diving scenarios



suction force, producing canal petechiae, serous exudate, or frank haemorrhage. Acute treatment involves removing predisposing blockages (cerumen clearance, canalplasty for exostoses); prophylactic drops may also be recommended. Preventive emphasis is on slow, head-up descents in susceptible divers.^{2,7,28}

Barosinusitis (sinus squeeze) similarly arises during descent when sinus ostia fail to ventilate adequately. The resulting vacuum effect within the sinus may cause mucosal oedema, capillary rupture, or pain localised to the frontal or maxillary regions.^{6,31} Pre-existing anatomical obstructions, such as septal deviation or nasal polyposis, may further predispose divers to sinus barotrauma.^{8,31} Survey data quantify the population burden: in the same Finnish cohort of 1,881 recreational divers, 49% reported having experienced sinus barotrauma while diving, with 32% of those affected having used medications. Independent risk factors included pollen allergy and regular smoking (both modifiable), as well as frequent (three or more per year) upper respiratory tract infections.³² These factors complement the anatomical predispositions already described and offer practical targets for pre-dive counselling. To minimise the risk, divers should abstain from diving during episodes of illness or significant nasal congestion. Management strategies should emphasise preventive measures, such as gentle equalisation techniques, optimal pre-dive nasal hygiene, and judicious use of prophylactic topical nasal corticosteroids or short-acting decongestants (e.g., oxymetazoline) to manage mucosal oedema and inflammation.⁶ Divers with recurrent symptoms should undergo thorough ENT evaluation to assess for underlying pathology or anatomical factors that may necessitate surgical intervention.^{6,33}

Rarely, divers who attempt forceful equalisation techniques during descent may place excessive mechanical strain on the larynx or pharyngeal soft tissues. This can result in minor mucosal injuries, localised edema, or even vocal fold

haemorrhage, presenting as throat discomfort, odynophagia, transient voice changes, or mild stridor during or shortly after the dive.^{5,34} Divers experiencing these symptoms should use gentler, controlled equalisation techniques or seek formal ENT evaluation if symptoms persist post-dive.

Any injuries thus sustained during descent can cause pain and discomfort throughout the whole dive and the indirect effects of this, not just on coloring the experience overall, but also on situational awareness and thus safety by extension, should not be minimised.

Bottom phase challenges and injuries

Once at depth, ambient pressure stabilises, and active equalisation demands generally decrease. Nonetheless, divers may still experience sustained mechanical stress, particularly from equipment or posture.^{4,6,12} Some divers report jaw discomfort during this phase, potentially linked to the application of pressure from ill-fitting mouthpieces and stress on the temporomandibular joint (TMJ) but this can also be referred pain from equalisation techniques, although this remains under-characterised.¹² Pressure-related microtrauma during descent and equalisation efforts may exacerbate pre-existing TMJ disorders. Though underreported, such complaints are not uncommon among seasoned divers and may influence diving tolerance and equipment selection (need for custom-fit mouthpieces or physical therapy to reduce strain).²⁰

Divers may be prone to headaches during the bottom phase related to hypercapnia, often secondary to hypoventilation, shallow breathing, or 'skip-breathing' techniques.³⁵ Increased partial pressure of carbon dioxide results in cerebral vasodilation, causing headaches, which can significantly impair situational awareness and diver safety. To mitigate this risk, divers should be advised to practice regular and deep tidal breathing, avoid excessive exertion

underwater, and ensure their equipment is properly maintained to facilitate efficient breathing.³⁵

On most dives, divers are safe from motion sickness once underwater. However, ground swell at depth can generate semicircular canal stimulation out of phase with visual cues, triggering nausea and retching into the regulator. Persistence of seasickness initiated from the surface can also last well into the dive while the vestibular system attempts to adapt. Currents can exacerbate this. Pre-exposure prophylaxis includes transdermal scopolamine 1.5 mg or meclizine 50 mg orally one hour before diving, alongside alcohol avoidance and hydration. Underwater, divers should fixate on a stable reference and adopt slow tidal breathing to mitigate hypercapnia-driven nausea. If vomiting occurs, the general advice is to assume vertical trim, maintain regulator purge, and end the dive.²⁸

Extended bottom times, particularly in cold water or enriched oxygen environments, can negatively impact mucociliary clearance, promote nasal mucosal oedema, and impair ET ventilatory function. These conditions may predispose divers to middle ear dysfunction, delayed middle ear barotrauma, or subtle ET injury due to hyperoxia-induced inflammation. Although free radical scavenger vitamins (C and E) have been studied, no clear protective effect has yet been demonstrated.^{12,36} As Meyer et al. point out, we have no standardised procedure for measuring successful equalisation during a dive. However, the cumulative physiological burden of repetitive diving is postulated to extend beyond the immediate descent phase and may compound susceptibility to barotrauma throughout multi-day itineraries.²³

Similarly, at depth, sinus barotrauma may become symptomatic or worsen if initial equalisation was incomplete, or if mucosal inflammation occurs while submerged. Persistent frontal or maxillary pain, often associated with epistaxis, may develop as a result of sustained pressure differentials within the paranasal sinuses.^{6,31} Divers frequently experiencing this should refer to the preventive strategies and management described previously.

Although less common at stable depth, prolonged bottom times involving continuous use of breathing apparatus against increased inspiratory resistance or poorly maintained regulator equipment may occasionally strain laryngeal musculature, resulting in mild dysphonia or throat discomfort.⁵ Such symptoms typically resolve spontaneously upon surfacing but can indicate the need for equipment evaluation or breathing technique adjustment.

Ascent phase injuries

During ascent, the reduction in ambient pressure causes expansion of gas trapped within anatomical compartments, increasing the risk of barotrauma from positive-pressure phenomena.

Reverse middle-ear block occurs when expanding air cannot adequately escape through the ET, causing outward displacement or even rupture of the tympanic membrane, intense otalgia, and conductive hearing loss. Though common during rapid or poorly controlled ascents, experienced divers can still encounter this phenomenon if mucosal oedema or inflammation develop during the dive, obstructing ET patency.^{12,19} Immediate management includes halting ascent, attempting gentle equalisation, or slightly descending until symptoms alleviate, followed by slow and controlled ascent. Post-dive evaluation is advised if symptoms persist.²

Paranasal sinuses may similarly retain trapped air that expands upon ascent, leading to acute sinus pain ('reverse sinus squeeze') as mucosal surfaces distend, sometimes accompanied by mucosal haemorrhage.^{6,31} This is more likely if mucus plugs or mucosal swelling or congestion developed at depth, restricting sinus ostial ventilation.⁶ Divers experiencing persistent sinus discomfort on ascent should be managed with topical or systemic decongestants and corticosteroids and evaluated for underlying anatomical issues that could be resolved surgically for the future.^{6,31}

Barosinusitis of ascent is variable in its presentation, and sometimes the only manifestation is bleeding from the same side as the sinus disorder. A differential diagnosis in these cases is anterior epistaxis, experienced by an estimated 3% of divers due to positive-pressure expansion across the congested Kiesselbach's plexus, producing brief unilateral nasal bleeding during ascent.⁷ Divers experiencing recurrent post-dive epistaxis should undergo ENT review. A small number of cases of sinus barotrauma can present rather dramatically, with nausea and vomiting, a sensation of impending syncope, and disorientation.⁶

Another critical concern specific to ascent is IEDCS. IEDCS arises from inert gas bubble formation within labyrinthine vessels or tissues due to supersaturation during ascent, or from right-to-left arterialisation of venous inert gas bubbles which subsequently reach the labyrinthine artery. Symptoms commonly include acute onset vertigo, nystagmus, hearing loss, and tinnitus, typically occurring within the first hour after surfacing.^{3,5,15} The largest published IEDCS case series, comprising 115 recreational scuba divers over a 13-year period, found that IEDCS represented 24% of all treated decompression sickness cases.³⁷ Median symptom onset was 20 minutes after surfacing, with pure vestibular disorders predominating (76.5%), cochlear deficit in 6%, and combined presentations in 17.5%. A large right-to-left shunt was detected in 77% of patients, with a striking right-sided lateralisation of injury (80%, $P < 0.001$), and incomplete recovery was observed in 68% of followed patients. A 30-year retrospective analysis of 437 DCS cases treated in Malta further demonstrated that vestibulo-cochlear decompression illness has been increasing in prevalence while musculoskeletal presentations have declined,

suggesting that evolving diving practices may be shifting the pattern of injury.³⁸

A key differentiator between IEBt and IEDCS is the timing: symptoms presenting primarily during descent with associated difficulty equalising strongly suggest IEBt, whereas symptoms emerging during or shortly after ascent more likely indicate IEDCS. But the distinction may be tricky even with a good clinical history, dive profile analysis, and audiovestibular assessment, as both may present with similar clinical findings, and the two may even coexist.³⁹ Unlike barotrauma, however, IEDCS often occurs without significant middle ear findings and may have a delayed onset after surfacing.³ Systematic review evidence has quantified several differentiating variables: mean depth at the causative dive was 13 metres for IEBt versus 43 metres for IEDCS, with additional discriminating factors including dive gas type, symptom onset timing relative to depth change, symptom distribution (vestibular versus cochlear predominance), and presence of associated DCS features.³ Notably, a history of difficult middle ear equalisation could not be reliably assessed as a differentiator because it was insufficiently reported in the IEDCS literature — a significant evidence gap. Structured diagnostic aids such as the HOOYAH tool may assist clinicians in navigating this differential when remote from specialist audiovestibular assessment.²⁷

Prompt recognition is critical, as IEDCS typically requires hyperbaric oxygen therapy and carries a risk of permanent auditory or vestibular dysfunction,⁷ whereas IEBt is primarily managed conservatively. Outcomes data from case series underscore the severity: in a retrospective series of 23 IEDCS patients treated at a single Australian hyperbaric unit between 2014 and 2020, 88% had vestibular involvement and 38% cochlear involvement, yet median maximum depth was only 24.5 metres and median dive time 40 minutes, demonstrating that IEDCS can occur on dives unremarkable by recreational standards.⁴⁰ Median time from surfacing to hyperbaric treatment was 22 hours, and complete symptom recovery was achieved in only 58%, underscoring the importance of early recognition and rapid referral. Among 126 recreational trimix divers, 7.1% reported at least one episode of IEDCS, of whom all reported vestibular symptoms, five of nine tested had a patent foramen ovale, and three reported persistent balance problems long after the event.⁴¹ IEBt has traditionally been managed conservatively, with recompression considered relatively contraindicated.¹⁶ However, a recent case series of five consecutive divers with sensorineural hearing loss attributed to IEBt reported substantial audiometric improvement following hyperbaric oxygen therapy administered 24 hours to 12 days after diving, with a median improvement of 28 dB across all frequencies and 38 dB for PTA4.⁴² While this represents a preliminary series requiring prospective confirmation, it suggests that the role of HBOT in IEBt-associated sensorineural hearing loss may warrant further investigation rather than blanket avoidance.

The functional consequences of IEDCS frequently persist beyond the acute phase. In a case series of 13 divers with acute IEDCS who received customised vestibular rehabilitation, vestibular and balance measures improved by discharge and at three-month follow-up; however, 67% still demonstrated objective vestibular deficits, including a positive head impulse test or nystagmus in the dark on videonystagmography, despite subjective symptom resolution.⁴³ This finding has important implications for return-to-dive counselling: symptom resolution alone should not be used as the sole criterion for fitness-to-dive clearance following IEDCS, and objective vestibular assessment coupled with vestibular rehabilitation should be incorporated into post-treatment evaluation.^{13,43}

Transient and typically benign alternobaric vertigo may also occur from asymmetrical middle-ear venting on ascent that creates a transient labyrinthine stimulation mismatch. Usually self-limiting, it presents as a brief spinning sensation without hearing impairment, lasting only seconds to minutes (< 90 s).^{5,12,44} ET dysfunction appears highly prevalent in divers with alternobaric vertigo, and allergic rhinitis was present in most affected divers in one study.⁴⁴ Severe episodes risk disorientation and uncontrolled ascent and even mild spells may still compromise a diver's spatial orientation temporarily. Furthermore, divers should be taught the 'ascend-pause-Toynbee' manoeuvre (nasal pinch + swallow) rather than forceful Valsalva and must abort the dive if vertigo persists beyond one minute. To minimise risk, underlying rhinitis or ET dysfunction should be treated before diving and tight hoods or occlusive earplugs should be discouraged.^{2,7,45}

A rare complication is facial baroparesis in which rapid expansion of middle ear gas compresses the facial nerve within its canal, particularly in the presence of anatomical canal dehiscence, resulting in transient unilateral facial weakness.⁴⁶ It is also possible for fifth nerve neuropraxia to produce facial numbness in maxillary sinus barotrauma, albeit in freediving in the most recently reported case.⁴⁷ These typically resolve spontaneously, but accurate differentiation from DCS is essential to ensure appropriate and timely hyperbaric therapy when indicated.⁴⁶ Balloon dilation of the ET prevented further episodes and enabled a full return to diving, suggesting a potential therapeutic role in selected cases.⁴⁸ No prospective series exists specifically in scuba divers; a series of freedivers treated with ET balloon dilation for refractory equalisation failure reported improvement in 15 of 20 patients (10 with complete resolution), suggesting potential applicability of this technique, though direct extrapolation to scuba divers requires caution.⁴⁹ Further prospective studies in scuba divers are needed.

Although ascent-phase complications are predominantly otological or sinogenic, maxillofacial symptoms may occasionally present diagnostic challenges. Maxillary pain, for example, could reflect dental or sinus barotrauma. Odontobarotrauma may be triggered during descent if pockets

of trapped air beneath dental restorations are compressed, manifesting as acute pain or crown displacement;⁶ however, odontocrexia (restoration fracture) is much more common with ascent, as rapid ambient-pressure shifts can propagate micro-bubbles beneath composite or amalgam restorations, leading to crown fracture or cusp blow-out. Incidence in frequent divers approaches 5%. Thus, routine dental reviews, sealing defective restorations, and completing endodontic work pre-dive, are recommended. Sharp fragments in situ require aborting the dive, waxing the defect, and urgent dental follow-up.⁶

Rare reports of laryngeal barotrauma have been associated specifically with rapid ascent or forceful respiratory efforts. Sudden pressure changes or excessive strain against respiratory resistance may cause transient vocal fold immobility, mucosal swelling, or soft tissue edema, manifesting as immediate post-dive hoarseness, throat tightness, or mild dyspnea.^{5,33} Divers experiencing these symptoms post-ascent should seek timely ENT evaluation, given potential airway implications and the need to distinguish from more serious pulmonary barotrauma (beyond the scope of ENT manifestations but relevant to clinical safety assessments). Divers with persistent cough, chest discomfort, or neurological symptoms should receive immediate medical evaluation for DCS or arterial gas embolism, as these may accompany ascent-phase ENT symptoms.¹⁶

During the final metres of ascent and at the surface interval, divers are particularly exposed to risks from marine life, although such incidents can occur at any point during the dive. Facial stings from jellyfish and other cnidarians may cause severe pain, peri-lesional oedema, and oropharyngeal dysesthesia. Immediate management involves terminating the dive, performing vinegar irrigation to neutralise unfired nematocysts, removing visible tentacles, and immersing in hot water ($\leq 45^{\circ}\text{C}$) for analgesia and toxin denaturation. Severe facial or neck stings require airway monitoring and ocular irrigation. Bites from territorial marine fauna, though rare, require thorough wound care, exploration for foreign materials, and empiric antibiotic therapy. Preventive measures include neutral buoyancy, visual vigilance during ascent, protective gear, and appropriate situational awareness.⁵⁰

Understanding these phase-specific mechanisms also informs clinical assessment of divers with pre-existing ENT conditions, where the same physiological stressors interact with altered anatomy or prior surgery.

FITNESS TO DIVE: CONSIDERATIONS IN PRE-EXISTING ENT CONDITIONS

ENT disorders frequently impact recreational divers, with approximately 80% of adult divers experiencing at least one related complaint,³³ and many of the aforementioned

disorders are avoidable. But what about ENT patients seeking advice on whether or not they can dive? Clinicians often advise blanket water precautions, but evidence regarding diving safety is mixed.

Otological conditions

Otological issues represent significant considerations for divers. Surgical history, such as stapedectomy, poses a controversial risk. While regulatory bodies typically advise against diving post-stapedectomy due to potential inner ear barotrauma, clinical studies suggest that divers with adequate ET function may safely dive post-procedure.^{51,52} Similarly, mastoidectomy poses variable risks depending on the procedure type.⁵² Simple mastoidectomy may not preclude diving, but more extensive atticotomy usually does due to increased risks of direct pressure transfer and deeper infections.^{33,52} Patients with ossicular prostheses in particular may be at increased risk of pressure transmission to the vestibule, particularly if equalisation is incomplete. For such individuals, careful dive clearance, counseling on conservative depth limits, and early symptom recognition are critical to minimising irreversible sequelae.² Pre-dive screening may not be limited to ENT assessment in these cases, as for example given the high prevalence of patent foramen ovale across IEDCS case series (reported in 60 to 83% of patients across multiple cohorts^{13,37,40}) raises the question of whether cardiac shunt screening should be considered for divers presenting with unexplained inner ear symptoms or recurrent vestibular episodes. A recent consensus of experts from two international diving medicine societies recommends consideration of PFO screening for divers who have suffered IEDCS.⁵³

Chronic ear disorders, such as cholesteatoma or active Ménière's disease, are generally contraindications for diving due to risks of vertigo exacerbation.³³ However, chronic external auditory canal or middle ear conditions with stable postoperative states, including tympanoplasty with successful ET function, may permit diving after comprehensive evaluation.^{33,52,54} Specifically, type I-IIIb tympanoplasties with stable membranes are often compatible with diving if a negative provocative test (such as tympanometry without induced nystagmus or vertigo) is documented.⁵⁴

Similarly, limited longitudinal evidence indicates that, when audiovestibular function and ET ventilation are normalised, divers with prior ear injury may return to diving without increased risk of recurrence.²⁹ ET dysfunction is problematic due to risks of middle ear barotrauma. Balloon tuboplasty has shown promise in restoring diving fitness by effectively managing persistent dysfunction.³³ Acute infections, especially otitis externa, represent temporary diving contraindications until fully resolved.⁵⁴

Hearing loss itself is not an absolute contraindication for recreational diving, but unilateral deaf divers should be

cautioned about potential total deafness from contralateral injury.^{33,54} Active middle ear implants and cochlear implants have specific pressure resistance limitations and require individual assessment of vestibular function and device integrity before diving.^{52,54} Emerging evidence suggests that cochlear implant recipients can safely dive within certain depth limits, but repetitive hyperbaric exposure may still carry theoretical risks, including electrode dysfunction, pressure-related vertigo, or altered impedance settings.¹⁷ Divers with cochlear implants or ossicular prostheses may encounter device-specific issues related to sustained hyperbaric exposure. Although contemporary cochlear implants are engineered to withstand pressures encountered during typical recreational dives (approximately 25–42 meters of seawater, depending on the manufacturer), repetitive dives have rarely resulted in electrode displacement, extrusion through the tympanic membrane, or device malfunction, manifesting as vertigo or tinnitus.^{16,17}

Rhinological conditions

Septal deviation significantly affecting sinus drainage predisposes divers to sinus barotrauma and should be surgically corrected before diving if severe. Chronic inflammatory nasal conditions such as allergic rhinitis and chronic rhinosinusitis require symptom control with topical treatments; acute exacerbations must be resolved before diving resumes. Divers with vascular nasal diseases, like granulomatosis with polyangiitis, face absolute contraindications due to systemic involvement risks.³³

Previous frontal or lateral skull base fractures necessitate rigorous evaluation due to pneumocephalus risk upon ascent. A negative β -2-transferrin test and computed tomography confirming sinus patency are required before diving can be considered safe.^{33,54}

Laryngological and upper airway conditions

Acute laryngitis and other upper airway infections contraindicate diving until fully resolved due to risks of laryngospasm and impaired middle-ear pressure equalisation.^{33,54} Anatomical abnormalities (e.g., laryngoceles, stenosis) preclude diving unless surgically corrected due to pulmonary barotrauma risks. Inflammatory conditions affecting the larynx, such as rheumatoid arthritis or relapsing polychondritis, constitute absolute contraindications due to the risk of respiratory compromise.³³ Contrastingly, unilateral vocal cord paralysis with adequate airway compensation and normal pulmonary function does not necessarily preclude diving.³³

Diving post-treatment for head and neck cancer requires thorough evaluation due to anatomical distortions, friable vasculature, and risks from radiotherapy-induced fibrosis and oedema. Late otological effects from radiation, such as ET dysfunction, may impede middle ear pressure equalisation, thus warranting a specialist review.³³ Many patients will not

have the performance status or desire to take on the risk, but those who do wish to should be given thorough counselling.

Conclusions

The phase-of-dive framework proposed here offers a clinically useful organising principle for ENT presentations in divers: descent injuries predominantly reflect failure of equalisation under negative pressure, bottom-phase complications arise from sustained exposure and equipment factors, and ascent-phase pathology encompasses a mix of reverse barotrauma and decompression sickness whose diagnostic ambiguity carries direct management consequences. IEDCS in particular is associated with incomplete recovery even after hyperbaric treatment, with persistent objective vestibular deficits documented in the majority of patients across multiple series.

The same framework may inform fitness-to-dive assessment by anticipating where altered anatomy or prior surgery creates vulnerability. What the available evidence cannot yet support is firm, quantitative guidance: the two controlled trials included here both showed null results for pharmacological prophylaxis, and the broader literature is dominated by small observational cohorts with moderate-to-high risk of bias. Prospective studies with standardised outcome measures, validated pre-dive screening protocols, and device-specific depth guidelines for implant recipients remain a genuine priority. Until then, the most useful tools available to the clinician are a mechanistically informed history anchored to dive phase, individualised risk assessment that accounts for dive profile, cumulative exposure, and anatomical predisposition, and realistic outcome counselling, particularly following IEDCS, where subjective recovery consistently outpaces objective vestibular function in the available data. For the diver, the practical takeaway is that most serious ENT complications are not random: they reflect identifiable physiological vulnerabilities that a well-informed clinician, and a well-informed diver, can anticipate and mitigate.

References

- 1 Darcy K. How to accurately measure the market size of the scuba diving industry. Scubonomics. 2024. [cited 2025 Jun 26]. Available from: <https://medium.com/scubonomics/how-to-accurately-measure-the-market-size-of-the-scuba-diving-industry-ceb4ac0631d3>.
- 2 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;47:97–109. doi: 10.28920/dhm47.2.97-109. PMID: 28641322. PMCID: PMC6147252.
- 3 Lindfors OH, Räisänen-Sokolowski AK, Hirvonen TP, Sinkkonen ST. Inner ear barotrauma and inner ear decompression sickness: a systematic review on differential diagnostics. Diving Hyperb Med. 2021;51:328–37. doi: 10.28920/dhm51.4.328-337. PMID: 34897597. PMCID: PMC8923696.

- 4 Alolaiwi LA, Alzahrani FA, Al Shammari SA. Diving into discomfort: orofacial pain dynamic – A systematic review. *Front Public Health*. 2025;13:1553541. doi: [10.3389/fpubh.2025.1553541](https://doi.org/10.3389/fpubh.2025.1553541). PMID: 40416680. PMCID: PMC12098605.
- 5 Scarpa A, Ralli M, De Luca P, Gioacchini FM, Cavaliere M, Re M, et al. Inner ear disorders in SCUBA divers: A review. *J Int Adv Otol*. 2021;17:260–4. doi: [10.5152/iao.2021.8892](https://doi.org/10.5152/iao.2021.8892). PMID: 34100753. PMCID: PMC9450052.
- 6 Livingstone DM, Lange B. Rhinologic and oral-maxillofacial complications from scuba diving: a systematic review with recommendations. *Diving Hyperb Med*. 2018;48:79–83. doi: [10.28920/dhm48.2.79-83](https://doi.org/10.28920/dhm48.2.79-83). PMID: 29888379. PMCID: PMC6156823.
- 7 Lechner M, Sutton L, Fishman JM, Kaylie DM, Moon RE, Masterson L, et al. Otorhinolaryngology and diving – Part I: Otorhinolaryngological hazards related to compressed gas scuba diving: a review. *JAMA Otolaryngol Head Neck Surg*. 2018;144(3):252–8. doi: [10.1001/jamaoto.2017.2617](https://doi.org/10.1001/jamaoto.2017.2617). PMID: 29450472.
- 8 Klingmann C, Praetorius M, Baumann I, Plinkert PK. Otorhinolaryngologic disorders and diving accidents: an analysis of 306 divers. *Eur Arch Otorhinolaryngol*. 2007;264:1243–51. doi: [10.1007/s00405-007-0353-6](https://doi.org/10.1007/s00405-007-0353-6). PMID: 17639445.
- 9 Skogstad M, Eriksen T, Skare Ø. A twelve-year longitudinal study of hearing thresholds among professional divers. *Undersea Hyperb Med*. 2009;36:25–31. PMID: 19341125.
- 10 St Leger Dowse M, Waterman MK, Jones R, Smerdon GR. Aural health awareness and incident prevention in UK scuba divers. *Diving Hyperb Med*. 2022;52:22–6. doi: [10.28920/dhm52.1.22-26](https://doi.org/10.28920/dhm52.1.22-26). PMID: 35313369. PMCID: PMC9177430.
- 11 Doolette DJ, Mitchell SJ. Biophysical basis for inner ear decompression sickness. *J Appl Physiol*. 2003;94:2145–50. doi: [10.1152/japplphysiol.01090.2002](https://doi.org/10.1152/japplphysiol.01090.2002). PMID: 12562679.
- 12 Mallen JR, Roberts DS. SCUBA medicine for otolaryngologists: Part I. Diving into SCUBA physiology and injury prevention. *Laryngoscope*. 2020;130:52–8. doi: [10.1002/lary.27867](https://doi.org/10.1002/lary.27867). PMID: 30776099.
- 13 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;28:447–54. doi: [10.1097/MAO.0b013e318030d356](https://doi.org/10.1097/MAO.0b013e318030d356). PMID: 17417111.
- 14 Mitchell SJ, Doolette DJ. Selective vulnerability of the inner ear to decompression sickness in divers with right-to-left shunt: the role of tissue gas supersaturation. *J Appl Physiol* (1985). 2009;106:298–301. doi: [10.1152/japplphysiol.90915.2008](https://doi.org/10.1152/japplphysiol.90915.2008). PMID: 18801958.
- 15 Mitchell SJ, Doolette DJ. Pathophysiology of inner ear decompression sickness: potential role of the persistent foramen ovale. *Diving Hyperb Med*. 2015;45:105–10. PMID: 26165533. [cited 2026 Jan 18]. Available from: https://dhmjournal.com/images/IndividArticles/45June/Mitchell_dhm.45.2.105-110.pdf.
- 16 Mallen JR, Roberts DS. SCUBA medicine for otolaryngologists: Part II. Diagnostic, treatment, and dive fitness recommendations. *Laryngoscope*. 2020;130:59–64. doi: [10.1002/lary.27874](https://doi.org/10.1002/lary.27874). PMID: 30776095.
- 17 Hintze JM, Geyer L, Fitzgerald CW, Simoes Franklin C, Glynn F, Viani L, et al. The impact of repetitive hyperbaric exposure during SCUBA diving on cochlear implants. *Laryngoscope*. 2019;129:2760–4. doi: [10.1002/lary.27880](https://doi.org/10.1002/lary.27880). PMID: 30810235.
- 18 Lindfors OH, Räisänen-Sokolowski AK, Suvilehto J, Sinkkonen ST. Middle ear barotrauma in diving. *Diving Hyperb Med*. 2021;51:44–52. doi: [10.28920/dhm51.1.44-52](https://doi.org/10.28920/dhm51.1.44-52). PMID: 33761540. PMCID: PMC8313771.
- 19 Uzun C, Adali MK, Korten M, Yagiz R, Aydin S, Cakir B, et al. Relationship between mastoid pneumatization and middle ear barotrauma in divers. *Laryngoscope*. 2002;112:287–91. doi: [10.1097/00005537-200202000-00016](https://doi.org/10.1097/00005537-200202000-00016). PMID: 11889385.
- 20 Duplessis C, Fothergill D, Gertner J, Hughes L, Schwaller D. A pilot study evaluating surfactant on eustachian tube function in divers. *Mil Med*. 2008;173:1225–32. doi: [10.7205/milmed.173.12.1225](https://doi.org/10.7205/milmed.173.12.1225). PMID: 19149344.
- 21 Blake DF, Gibbs CR, Commons KH, Brown LH. Middle ear barotrauma in a tourist-oriented, condensed open-water diver certification course: incidence and effect of language of instruction. *Diving Hyperb Med*. 2015;45:176–80. PMID: 26415068. [cited 2026 January 18]. Available from: https://www.dhmjournal.com/images/IndividArticles/45Sept/Blake_dhm.45.3.176-180.pdf.
- 22 Jansen S, Meyer MF, Boor M, Felsch M, Klünter H-D, Pracht ED, et al. Prevalence of barotrauma in recreational scuba divers after repetitive saltwater dives. *Otol Neurotol*. 2016;37:1325–31. doi: [10.1097/MAO.0000000000001158](https://doi.org/10.1097/MAO.0000000000001158). PMID: 27636390.
- 23 Meyer MF, Boor M, Jansen S, Pracht ED, Felsch M, Klünter M, et al. Influence of repetitive diving in saltwater on pressure equalization and Eustachian tube function in recreational scuba divers. *Diving Hyperb Med*. 2017;47:216–22. doi: [10.28920/dhm47.4.216-222](https://doi.org/10.28920/dhm47.4.216-222). PMID: 29241230. PMCID: PMC6706334.
- 24 Moayed S, Gizaw A, Sweet S, Sethuraman K, Witting M. Pseudoepithelium prophylaxis does not prevent middle ear barotrauma in hyperbaric oxygen therapy. *Undersea Hyperb Med*. 2025;52:101–7. PMID: 40819351.
- 25 Uzun C. Evaluation of pre-dive parameters related to eustachian tube dysfunction for symptomatic middle ear barotrauma in divers. *Otol Neurotol*. 2005;26:59–64. PMID: 15699720.
- 26 Morvan J-B, Gemppe E, Rivière D, Louge P, Vallee N, Verdalle P. Perilymphatic fistula after underwater diving: a series of 11 cases. *Diving Hyperb Med*. 2016;46:72–5. PMID: 27334993. [cited 2026 Jan 10]. Available from: https://www.dhmjournal.com/images/IndividArticles/46June/Morvan_dhm.46.2.72-75.pdf.
- 27 Rozycki SW, Brown MJ, Camacho M. Inner ear barotrauma in divers: an evidence-based tool for evaluation and treatment. *Diving Hyperb Med*. 2018;48:186–93. doi: [10.28920/dhm48.3.186-193](https://doi.org/10.28920/dhm48.3.186-193). PMID: 30199891. PMCID: PMC6205852.
- 28 Ears & Diving. Divers Alert Network. n.d. [cited 2025 Jun 27]. Available from: <https://dan.org/health-medicine/health-resource/dive-medical-reference-books/ears-diving/>.
- 29 Parell GJ, Becker GD. Inner ear barotrauma in scuba divers: A long-term follow-up after continued diving. *Arch Otolaryngol Head Neck Surg*. 1993;119:455–7. doi: [10.1001/archotol.1993.01880160103016](https://doi.org/10.1001/archotol.1993.01880160103016). PMID: 8457309.
- 30 Shupak A. Recurrent diving-related inner ear barotrauma. *Otol Neurotol*. 2006;27:1193–6. doi: [10.1097/O1.mao.0000231499.69404.22](https://doi.org/10.1097/O1.mao.0000231499.69404.22). PMID: 16983314.
- 31 Chen T, Pathak S, Hong EM, Benson B, Johnson AP, Svider PF. Diagnosis and management of barosinusitis: A systematic review. *Ann Otol Rhinol Laryngol*. 2023;132:50–62. doi: [10.1177/00034894211072353](https://doi.org/10.1177/00034894211072353). PMID: 35130739.
- 32 Lindfors OH, Räisänen-Sokolowski AK, Suvilehto J, Sinkkonen ST. Sinus barotrauma in diving. *Diving Hyperb Med*. 2021;51:182–9. doi: [10.28920/dhm51.2.182-189](https://doi.org/10.28920/dhm51.2.182-189). PMID: 34157734. PMCID: PMC8426117.

- 33 Lechner M, Sutton L, Fishman JM, Kaylie DM, Moon RE, Masterson L, et al. Otorhinolaryngology and diving—Part 2: Otorhinolaryngological fitness for compressed gas scuba diving: A review. *JAMA Otolaryngol Head Neck Surg.* 2018;144:259–63. doi: [10.1001/jamaoto.2017.2616](https://doi.org/10.1001/jamaoto.2017.2616). PMID: 29450499.
- 34 Talmi YP, Finkelstein Y, Zohar Y. Decompression sickness induced hearing loss. A review. *Scand Audiol.* 1991;20:25–8. doi: [10.3109/01050399109070786](https://doi.org/10.3109/01050399109070786). PMID: 1842265.
- 35 Burkett JG, Nahas SJ. Diving headache. *Curr Pain Headache Rep.* 2019;23(7):46. doi: [10.1007/s11916-019-0787-8](https://doi.org/10.1007/s11916-019-0787-8). PMID: 31147799.
- 36 Mutzbauer TS, Neubauer B, Mueller PH, Sigg O, Tetzlaff K. Can eustachian tube ventilatory function impairment after oxygen diving be influenced by application of free radical scavenger vitamins C and E? *Laryngoscope.* 2001;111:861–6. doi: [10.1097/00005537-200105000-00020](https://doi.org/10.1097/00005537-200105000-00020). PMID: 11359167.
- 37 Gempp E, Louge P. Inner ear decompression sickness in scuba divers: a review of 115 cases. *Eur Arch Otorhinolaryngol.* 2013;270:1831–7. doi: [10.1007/s00405-012-2233-y](https://doi.org/10.1007/s00405-012-2233-y). PMID: 23100085.
- 38 Azzopardi CP, Caruana J, Matity L, Muscat S, Meintjes WAJ. Increasing prevalence of vestibulo-cochlear decompression illness in Malta – an analysis of hyperbaric treatment data from 1987–2017. *Diving Hyperb Med.* 2019;49:161–6. doi: [10.28920/dhm49.3.161-166](https://doi.org/10.28920/dhm49.3.161-166). PMID: 31523790. PMCID: PMC6881197.
- 39 Adkisson GH, Meredith AP. Inner ear decompression sickness combined with a fistula of the round window. Case report. *Ann Otol Rhinol Laryngol.* 1990;99(9 Pt 1):733–7. doi: [10.1177/000348949009900913](https://doi.org/10.1177/000348949009900913). PMID: 2396811.
- 40 Mason JS, Buzzacott P, Gawthrop IC, Banham ND. A retrospective review of divers treated for inner ear decompression sickness at Fiona Stanley Hospital hyperbaric medicine unit 2014–2020. *Diving Hyperb Med.* 2023;53:243–50. doi: [10.28920/dhm53.3.243-50](https://doi.org/10.28920/dhm53.3.243-50). PMID: 37718299. PMCID: PMC10735645.
- 41 Guenzani S, Mereu D, Messersmith M, Olivari D, Arena M, Spanò A. Inner-ear decompression sickness in nine trimix recreational divers. *Diving Hyperb Med.* 2016;46:111–6. PMID: 27334999. [cited 2026 Jan 10]. Available from: https://www.dhmjournal.com/images/IndividArticles/46June/Guenzani_dhm46.2.111-116.pdf.
- 42 Smart D. Five consecutive cases of sensorineural hearing loss associated with inner ear barotrauma due to diving, successfully treated with hyperbaric oxygen. *Diving Hyperb Med.* 2024;54:360–7. doi: [10.28920/dhm54.4.360-367](https://doi.org/10.28920/dhm54.4.360-367). PMID: 39675746. PMCID: PMC12018693.
- 43 Stokes RJ, Watts D, Smerdon G, Hall SD, Bunn L, Marsden J. Vestibular rehabilitation and recovery in divers with inner ear decompression sickness: a case series. *Diving Hyperb Med.* 2025;55:236–45. doi: [10.28920/dhm55.3.236-245](https://doi.org/10.28920/dhm55.3.236-245). PMID: 40986919. PMCID: PMC12790930.
- 44 Kitajima N, Sugita-Kitajima A, Kitajima S. Altered eustachian tube function in SCUBA divers with alternobaric vertigo. *Otol Neurotol.* 2014;35:850–6. doi: [10.1097/MAO.0000000000000329](https://doi.org/10.1097/MAO.0000000000000329). PMID: 24751737.
- 45 Klingmann C, Knauth M, Praetorius M, Plinkert PK. Alternobaric vertigo--really a hazard? *Otol Neurotol.* 2006;27:1120–5. doi: [10.1097/01.mao.0000235373.78116.a8](https://doi.org/10.1097/01.mao.0000235373.78116.a8). PMID: 17130801.
- 46 Hyams AF, Toynton SC, Jaramillo M, Stone LR, Bryson PJ. Facial baroparesis secondary to middle-ear over-pressure: a rare complication of scuba diving. *J Laryngol Otol.* 2004;118:721–3. doi: [10.1258/0022215042244813](https://doi.org/10.1258/0022215042244813). PMID: 15509373.
- 47 Canarslan Demir K, Yücel Z. Maxillary sinus barotrauma with infraorbital nerve paraesthesia after breath-hold diving. *Diving Hyperb Med.* 2024;54:230–2. doi: [10.28920/dhm54.3.230-232](https://doi.org/10.28920/dhm54.3.230-232). PMID: 39288929. PMCID: PMC11659071.
- 48 Utz ER, Wise SR. Navy diver with recurrent facial nerve baroparesis treated with eustachian tube balloon dilation. *Laryngoscope.* 2019;129:E412–E414. doi: [10.1002/lary.28221](https://doi.org/10.1002/lary.28221). PMID: 31400145.
- 49 Giunta AA, Liberati L, Pellegrino C, Ricci G, Rizzo S. Eustachian tube balloon dilation in treatment of equalization problems of freediving spearfishermen. *Diving Hyperb Med.* 2019;49:9–15. doi: [10.28920/dhm49.1.9-15](https://doi.org/10.28920/dhm49.1.9-15). PMID: 30856662. PMCID: PMC6526055.
- 50 Nochetto M, Denoble P. Hazardous marine life. Divers Alert Network. n.d. [cited 2025 Jul 3]. Available from: <https://dan.org/health-medicine/health-resource/dive-medical-reference-books/hazardous-marine-life/>.
- 51 House JW, Toh EH, Perez A. Diving after stapedectomy: clinical experience and recommendations. *Otolaryngol Head Neck Surg.* 2001;125:356–60. doi: [10.1067/mhn.2001.118183](https://doi.org/10.1067/mhn.2001.118183). PMID: 11593171.
- 52 Riestra-Ayora J, Fernández-Navarro C, Martín-Sanz E, Salvador-Marín MF. Evidence-informed decision aid for fitness-to-dive assessment after otologic surgery. *Diving Hyperb Med.* 2026;56:177–84. doi: [10.28920/dhm56.2.177-184](https://doi.org/10.28920/dhm56.2.177-184). PMID: 42290578.
- 53 Smart D, Wilmshurst P, Banham N, Turner M, Mitchell SJ. Joint position statement on atrial shunts (persistent [patent] foramen ovale and atrial septal defects) and diving: 2025 update. South Pacific Underwater Medicine Society (SPUMS) and the United Kingdom Diving Medical Committee (UKDMC). *Diving Hyperb Med.* 2025;55:51–5. doi: [10.28920/dhm55.1.51-55](https://doi.org/10.28920/dhm55.1.51-55). PMID: 40090026. PMCID: PMC12043516.
- 54 Klingmann C, Praetorius M, Böhm F, Tetzlaff K, Plinkert PK. [Fitness to dive in the otorhinolaryngological field]. *HNO.* 2008;56:509–18. doi: [10.1007/s00106-008-1743-9](https://doi.org/10.1007/s00106-008-1743-9). PMID: 18415065. German.

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