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Otorhinolaryngology and Diving—Part 1: Otorhinolaryngological Hazards Related to Compressed Gas Scuba Diving
A Review

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IMPORTANCE Scuba diving is becoming increasingly popular. However, scuba diving is associated with specific risks; 80% of adults and 85% of juvenile divers (aged 6-17 years) have been reputed to have an ear, nose, or throat complaint related to diving at some point during their diving career. Divers frequently seek advice from primary care physicians, diving physicians, and otorhinolaryngologists, not only in the acute setting, but also related to the long-term effects of diving.

OBSERVATIONS The principles underpinning diving-related injuries that may present to the otorhinolaryngologist rely on gas volume and gas saturation laws, and the prevention of these injuries requires both that the diver is skilled and that their anatomy allows for pressure equalization between the various anatomical compartments. The overlapping symptoms of middle ear barotrauma, inner ear barotrauma, and inner ear decompression sickness can cause a diagnostic conundrum, and a thorough history of both the diver's symptoms and the dive itself are required to elucidate the diagnosis. Correct diagnosis and appropriate treatment result in a more timely return to safe diving.

CONCLUSIONS AND RELEVANCE The aim of this review is to provide a comprehensive overview of otorhinolaryngological complications during diving. With the increasing popularity of diving and the frequency of ear, nose, or throat–related injuries, it could be expected that these injuries will become more common and this review provides a resource for otorhinolaryngologists to diagnose and treat these conditions.

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Diving is an increasingly popular leisure activity, with a total of 3.1 million Americans having dived at least once in 2013 according to the Scuba Diving Participation Report 2014.[1] Ear, nose, and throat disorders are the main issues that divers encounter, with previous reports of 80% of adults[2] and 85% of juvenile divers (aged 6-17 years) having had an ear, nose, or throat complaint related to diving.[3]

The principles underpinning these disorders relate to 2 well-known gas laws: Boyle's law (**Figure 1**) and Henry's law (**Figure 2**). Boyle's law states that at a given temperature the volume of a gas is inversely proportional to its pressure. During the first 10 m of descent, the volume of gas in gas-containing spaces in the body is reduced by 50%. To prevent pressure-induced trauma, this negative pressure needs to be equilibrated via the eustachian tube and sinus ostia. Henry's law states that at a given temperature the amount of gas that dissolves in a liquid with which it is in contact is proportional to the partial pressure of that gas. In diving, compressed air is used as a breathing gas under hyperbaric conditions with a percentage of nitrogen near 80%. During ascent, this inert gas is required to be transported from the tissues into the lungs for expulsion. If the rate of ascent is not controlled, the decrease in ambient pressure is too great to keep the gas in solution, resulting in microscopic bubble formation in tissues or blood. This can cause both mechanical damage and anoxia, leading to cell injury and subsequent decompression illness. Due to the metabolic activity of oxygen and carbon dioxide, their contribution to this gas phase is minimal.

The following sections provide an overview of otorhinolaryngological problems and complications during diving and are presented by anatomical location.

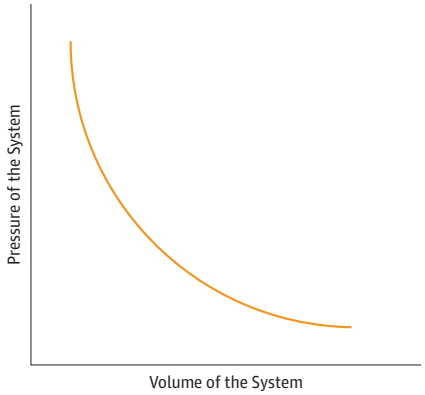
Otological Diseases of Diving and Related Conditions

Conditions of the External Ear

Otitis Externa

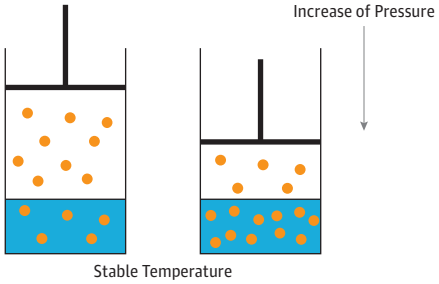
Otitis externa has been experienced by 44% of experienced divers during their diving history.[4] Retained moisture and introduction of microorganisms, as well as washing away of the protective acid mantle, have been implicated in diving-related otitis externa.[5]

Figure 1. Boyle's Law



Boyle's law states that at a given temperature the volume of a gas is inversely proportional to its pressure.

Figure 2. Henry's Law



Henry's law states that at a given temperature the amount of gas that dissolves in a liquid with which it is in contact is proportional to the partial pressure of that gas.

The best form of prevention is to avoid aquatic environments, but this is generally an impractical solution for divers. Those divers with untreated external auditory canal eczema should avoid diving until this is treated. Acetic acid, 2%, eardrops have been shown to reduce the growth of pathological gram-negative bacteria and promote normal ear flora.[6,7] A commercial preparation consisting of acetic acid and aluminum acetate is also effective.

Exostosis of the External Ear

Exostoses are benign bony growths, which occur in response to various irritants. It has been postulated that repeated and prolonged exposure to particularly cold water causes localized bone formation.[8] They can result in a narrowing of the ear canal, which predisposes the diver to debris and water trapping, resulting in transient hearing loss or otitis externa. Rarely, they can cause complete occlusion of the canal and as such rarely require treatment.

Cerumen

Cerumen can cause significant difficulty to divers. The cerumen in the ear canal can swell following immersion, causing ear pain. It can also result in water and debris trapping, predisposing to otitis externa or transient hearing loss. Cerumen occluding the canal or tight-fitting hoods can predispose to external ear barotrauma.[9] With regular aural care in affected individuals, this rarely causes problems in divers.

Conditions of the Middle Ear

Middle Ear Barotrauma (MEB)

Compression Barotrauma (MEB on Descent) | Compression barotrauma is the most common medical problem in diving.[10-12] Otherwise known as "middle ear squeeze," it occurs during descent as the ambient water pressure exceeds the middle ear air pressure (**Figure 3**). This causes the tympanic membrane (TM) to deform inward. During the first 10 m of descent, the volume of gas in the middle ear is reduced by 50%. To prevent pressure-induced trauma, this negative pressure needs to be equilibrated via eustachian tube opening to allow pressurized air from the scuba tank in the mouth access to the middle ear.[13] Inability to open the eustachian tube or too rapid a descent prevents pressure equalization and the TM bows into the middle ear with subsequent pain, vascular congestion, hemorrhage, and ultimately perforation.[14]

It is characterized by pain, usually on descent, conductive hearing loss, and otoscopic middle ear or TM barotrauma findings. Rupture of the TM results in cold water rushing into the middle ear, causing a cold caloric stimulus of the vestibular system. Symptoms of vertigo, nausea, and vomiting may occur, which can be fatal underwater. The degree of injury in MEB can be staged according to the Teed classification (**Figure 4**).[15]

The majority of cases will resolve on their own accord if the ear is not exposed to further barotrauma. Nasal decongestants such as oxymetazoline hydrochloride, a course of intranasal steroids, and nonsteroidal anti-inflammatory drugs may prove beneficial. The patient should not dive again until the hearing, eustachian tube function, and otoscopic findings have normalized.[16]

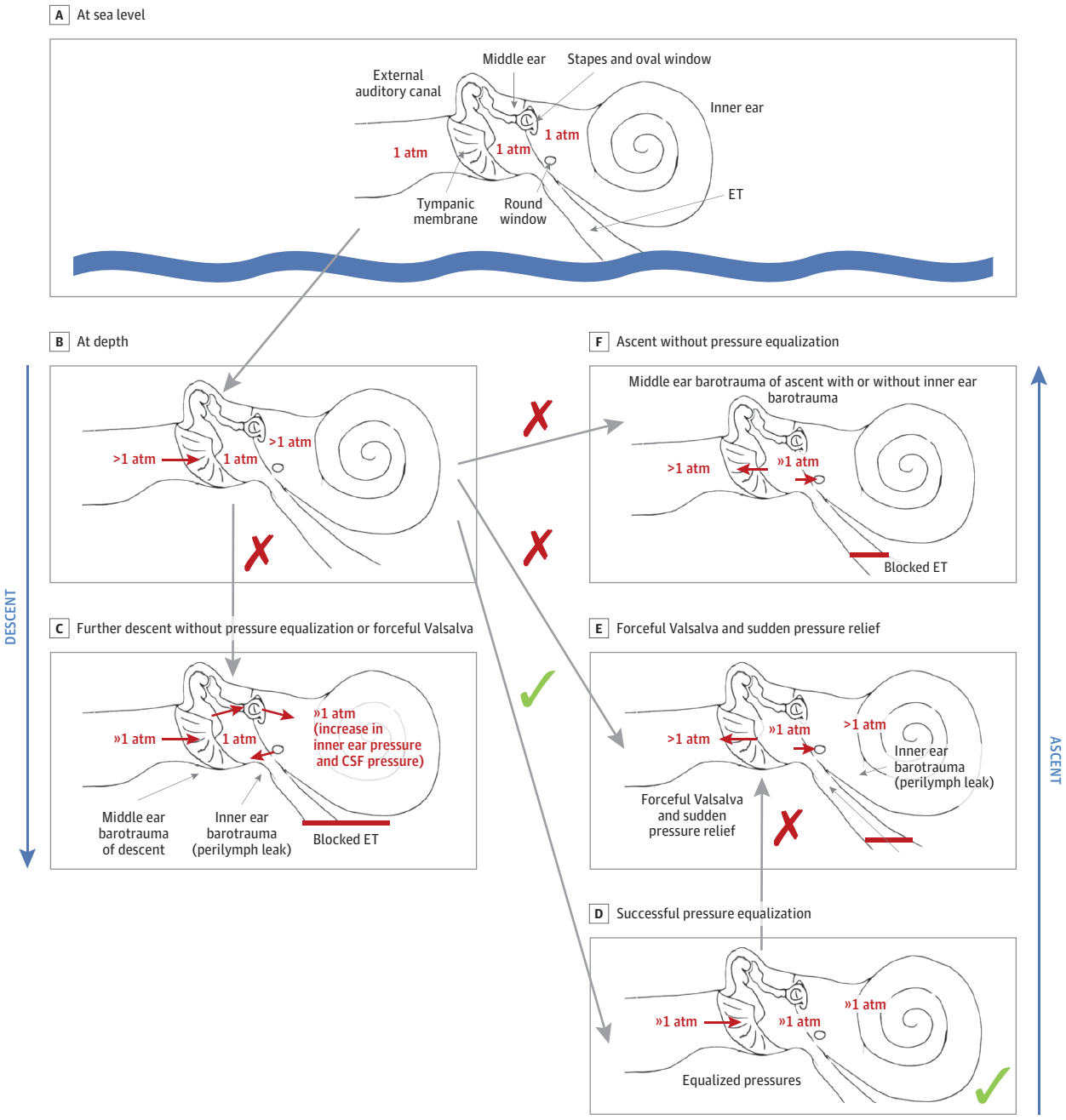
In grade 5 barotrauma, the TM has ruptured, allowing water into the middle ear, potentially resulting in infection. In the majority of cases, the TM does not require repair,[12] with most healing spontaneously.[17] Diving should not be resumed until the membrane has healed.

A variety of maneuvers for middle ear pressure equalization are used by divers, for example, the Valsalva maneuver, Toynbee maneuver, and Frenzel maneuver.[18] The position of the head when diving can aggravate equalization issues. A "head first" technique can induce higher nasopharyngeal pressures making equalization more difficult, whereas a "feet first" technique reduces this pressure. Middle ear ventilation should be started at the surface with maneuvers being performed 3 times per meter until a depth of 5 m is reached. A diver who has difficulty with pressure equalization should ascend, attempt an additional maneuver, and descend at a slower pace. If the repeated Valsalva maneuver on ascending is unsuccessful, the dive should be aborted.[12]

Goplen et al[14] observed a cohort of 53 patients for a period of 6 years. Their study found that MEB had no long-term effect on hearing loss. Additional studies have confirmed this good prognosis; however, repeated barotrauma may cause permanent hearing loss.[19]

Decompression Barotrauma (MEB on Ascent) | Decompression barotrauma occurs on ascent ("reverse squeeze") and is less com-

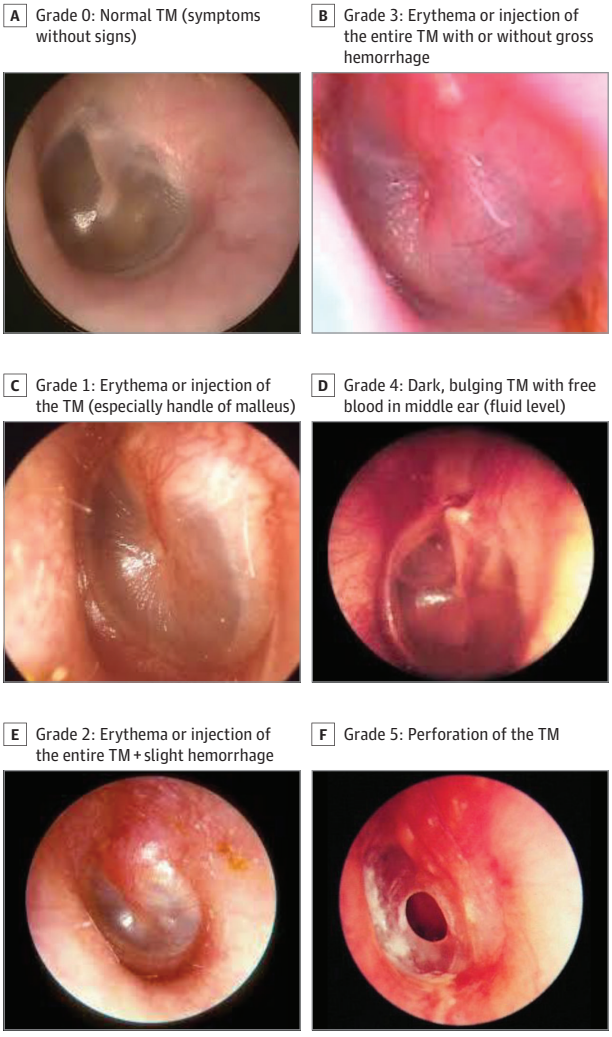
Figure 3. Middle Ear and Inner Ear Barotrauma: Schematic of Sequence of Events During Descent and Ascent



A, At sea level, pressures are equal. B- D, During descent the ambient water pressure increases. There is a need to equalize the middle ear pressure, for example, via a Valsalva maneuver. D, If the eustachian tube (ET) is patent and the diver successfully performs, for example, the Valsalva maneuver, both ambient pressure and middle ear pressure are equal. If the ET is blocked (in case of, eg, an upper respiratory tract infection) and descent is continued by the diver with inadequate equalization of pressures, the tympanic membrane and the oval window are bulging inward and the round window is bulging outward into the middle ear. This will eventually result in middle ear barotrauma of descent (C) and/or inner ear barotrauma with bulging and rupture of the round window into the middle ear ("exploding" from the inner ear perspective) with or without resulting leak of perilymph into the middle ear, especially if the cerebrospinal fluid (CSF) and/or inner ear pressure is high. E, If the ET is blocked and the diver performs a forceful Valsalva maneuver against the blocked ET, this forceful Valsalva maneuver can lead to inner ear barotrauma with inward bulging and a rupture of the round window ("imploding" from an inner ear perspective) with or without resulting leak of perilymph into the middle ear. F, If the ET is blocked on ascent and the middle ear pressure increases, this can cause middle ear barotrauma and inner ear barotrauma on ascent. Red x indicates barotrauma; green check, dive without barotrauma.

monly observed because airflow through the eustachian tube is less impeded during air egress. Pressure from expanding middle ear air then exceeds the surrounding water pressure, causing the TM to bulge outward. Causes for this include use of short-acting decongestants, which wear off while the diver is submerged.[9,13] Ear pain during ascent is the predominant feature of this disorder.

Figure 4. The Teed Classification of Middle Ear Barotrauma



TM indicates tympanic membrane.

Symptoms such as tinnitus, vertigo, or hearing loss may suggest rupture of the round window.[20] Individuals in whom the portion of the facial nerve within the middle ear is congenitally unsheathed or dehiscent can present with ipsilateral upper and lower motor neuron facial palsy ("facial baroparesis," discussed herein). This condition is treated in the same manner as compression barotrauma of the middle ear, and these conditions may coexist.

Alternobaric Vertigo

With the expansion of gas in the middle ear during ascent, the relative pressure in the middle ear increases. The eustachian tubes open and gas escapes into the nasopharynx. If there is unilateral obstruction of the eustachian tubes, the pressure discrepancy between the 2 ears results in stimulation of the vestibular system. This can cause symptoms of vertigo and is known as *alternobaric vertigo*. The disorientation associated and the vomiting can be catastrophic in an underwater setting. Correcting the disorder underwater requires halting ascent and performing a Valsalva maneuver or, if unsuccessful, descending to perform the Valsalva.[21] Diving should be avoided whenever middle ear pressure equalization is impaired.[19]

Conditions of the Inner Ear

Inner Ear Barotrauma (IEB)

This occurs when middle ear pressures do not equalize, secondary to forceful Valsalva maneuvers and to increased cerebrospinal fluid (CSF) pressure. This usually occurs on descent. The pathomechanism and the theoretical sequence of events is outlined in Figure 3.

There are 2 proposed explanations regarding the etiology of IEB. The first, more popular explanation suggests that on descent the TM bulges inward, resulting in the ossicles and eventually the stapes footplate being pushed into the oval window. This then causes redistribution of the perilymph within the cochlea and causes the round window to bulge outward. This increase in inner ear pressure and the increase in spinal fluid pressure contributing to this can be sufficient for the gradient between the middle ear and inner ear perilymph to cause rupture of the oval or round window, resulting in a perilymph leak from the inner ear.[22] The second explanation postulates that the increase in CSF pressure is transferred to the cochlea through a patent cochlear aqueduct during a forceful Valsalva maneuver, causing rupture or dislocation of the cochlear membranes and rupture of the round window membrane.[23] This has been demonstrated in animal experiments, with an increase in CSF pressure. The cochlear aqueduct decreases in size with age, and this may explain why children are more susceptible to IEB.[16]

This entity can mimic inner ear decompression illness, and a thorough history of both the patient's symptoms and the dive are necessary to establish a diagnosis. Symptoms of IEB can vary from mild sensorineural hearing loss to complete unilateral deafness, tinnitus and severe vertigo, nausea, and vomiting.[10,24] The patient may describe the unilateral sensation of water stuck in 1 ear.[25] The treatment depends on the severity of the symptoms. Conservative management with systemic steroids, vasodilators, local decongestants, and measures to prevent substantial increases in CSF pressure (eg, stool softeners) has been described. Surgery involves tympanotomy with a fat graft to repair the round window membrane for cases that do not resolve within the several-week observation period.[12]

To prevent inner ear barotrauma, middle ear ventilation using the Valsalva, Toynbee, or Frenzel maneuvers should be performed, as described herein. If difficulty with middle ear ventilation persists, the diver should abort the dive to prevent both IEB and MEB.[12]

Inner Ear Decompression Sickness (IEDCS)

Decompression illness or sickness (DCS) can be classified as mild, with skin, muscle, and joint symptoms (DCS type I), or severe, a diffuse, multifocal central nervous system disease (DCS type II). Inner ear decompression injury is a subtype of DCS type II and often occurs in isolation with no other organ or tissue involvement.[26]

Decompression sickness occurs when rapid ascent causes a sharp reduction in ambient pressure allowing dissolved inert gas within the inner ear to become supersaturated and form bubbles. It can be facilitated by isobaric counterdiffusion, when breathing gas

during decompression from a helium dive is switched to air. Residual helium within the middle ear then diffuses into the perilymph, into which nitrogen enters from the blood, thus increasing the total inert gas partial pressure. However, IEDCS does occur even with air diving. Postulated theories include native microbubble formation within the otic fluids and microvasculature,[18] and there is increasing evidence that arterial gas emboli transferred from venous blood via a right-to-left shunt and lodging in the labyrinthine artery arrests blood flow and causes ischemia.[18,27,28]

Inner ear DCS can present with vertigo, tinnitus, hearing loss, nausea, or vomiting, all of which overlap with inner ear barotrauma. Vestibular symptoms tend to predominate, with the suggested explanation being that the vestibular organ has lower perfusion than that of the cochlea.[29]

Differentiating between IEB and IEDCS can be difficult. However, the patient's history can provide important clues. If the symptoms occur on descent or follow a forceful Valsalva maneuver, it is likely IEB. The dive should have been aborted at that point. However, if occurring on ascent, IEDCS must be considered. If the patient had a previous history of eustachian tube dysfunction or MEB or if there is evidence of MEB on physical examination, IEB is more likely. Also, the depth of the dive is an important differentiating factor, with IEB being more prevalent in dives less than 30 m deep and IEDCS more common in dives greater than 30 m. Inner ear DCS is rare, with an incidence in recreational diving ranging from between 0.2% and 0.3% per dive[30] and although dizziness/vertigo occurs in approximately 20% of recreational DCS cases, not all of these are necessarily due to inner ear disease.[31]

Inner ear DCS manifestations often start up to 20 to 120 minutes after decompression. Evidence of other DCS manifestations such as sensory or motor signs also supports IEDCS. If IEDCS is suspected, prompt treatment is required, including first aid supplemental oxygen; definitive treatment for IEDCS consists of recompression therapy. Time to initiation of treatment has been shown to affect prognosis.[31,32] A concern is that by mistakenly treating a patient with IEB with recompression, Valsalva maneuvers during the compression phase could exacerbate the injury. To prevent this, some physicians perform myringotomy prior to recompression. Most patients with IEDCS eventually become asymptomatic because the brain can compensate for any residual injury to the vestibular apparatus, but mild hearing deficits may persist. Detailed testing, which is recommended, using audiograms and rotary and caloric testing has revealed a high rate of residual abnormalities (25%-90%).[25,33]

Facial Nerve Palsy Following Diving

If the facial nerve is dehiscent along its course through the temporal bone, it can be affected by pressure changes in the middle ear. Interestingly, there are case reports that pressure changes and associated changes in pressure and microcirculation can cause facial nerve palsy even during flying or a nose-blow.[34,35] Apart from symptoms and signs of facial nerve palsy, patients often report otalgia or other symptoms of otitic barotrauma. This can be differentiated from DCS by involvement of both the upper and lower facial muscles. In accordance with recommendations for MEB, use of nasal decongestants, a course of intranasal steroids, and nonsteroidal anti-inflammatory drugs may prove beneficial. There is not enough evidence to recommend a myringotomy, hyperbaric oxygen therapy, or a course of oral steroids routinely in these circumstances. Divers with a known dehiscence of the facial nerve should be advised that they are at increased risk of nerve injury during diving.[36]

Rhinological Hazards of Diving

Epistaxis

Approximately 3% of divers complain of recurrent epistaxis that usually resolves spontaneously. It is commonly associated with ascent due to gas expansion and may be triggered by irritation of the mucosa or by minor trauma to the Little area or other areas during the dive.[37]

Barotrauma of the Paranasal Sinuses

More than 10% of experienced divers have a history of paranasal sinus barotrauma in their career.[11] During descent, air in the paranasal sinuses is compressed according to Boyle's law (Figure 1). In the event of any blockage of the drainage pathways secondary to an acute upper respiratory tract infection or chronic rhinosinusitis, pressures cannot be equalized with air passing into the sinuses. The diver complains of pain over the affected sinus during descent, and this may be preceded by a sensation of tightness and pressure. The relatively low pressures cause mucosal edema and effusion with rupture of small blood vessels, causing bleeding into the sinuses, thereby reducing the pressure and the degree of pain and discomfort.

During ascent, this air then expands in the paranasal sinuses with drainage of mucus and blood from the sinuses (often visible in the diver's mask). Rarely, the drainage pathways are blocked to an extent that the pressure inside the sinuses cannot overcome the blockage and the expanding air may fracture the walls of the sinuses, tracking along the soft tissues. This may cause surgical emphysema, bruising, and hemorrhage in adjoining skin and damage to orbital contents and vision.[38] If air tracks intracranially, this leads to pneumocephalus.[39] Damage to the maxillary division of the trigeminal nerve has also been described. Depending on whether the infraorbital nerve or the superior alveolar nerve is affected, this can cause numbness and paraesthesia over the cheek or the upper teeth, gums, and mucosa of the same side, respectively.[40,41] The optic nerve runs adjacent to the sphenoid sinus; thus, if the bony canal is dehiscent, sphenoid sinus barotrauma of ascent can cause visual impairment.[42-44]

Usually the condition is self-limiting. In accordance with the recommendations for MEB, use of nasal decongestants, a course of intranasal steroids, and nonsteroidal anti-inflammatory drugs may prove beneficial. Antibiotics are not recommended routinely but are indicated if there is evidence of surgical emphysema. In cases of orbital or intracranial involvement, surgical decompression may rarely be indicated. If recurrent paranasal barotrauma occurs, functional endoscopic sinus surgery is highly effective to prevent sinus barotrauma in the future.[45]

Laryngeal Hazards of Diving

Laryngospasm

Laryngospasm consists of rapid and forceful contraction of the laryngeal adductors in response to a noxious stimulus,

causing stridor and airway obstruction. Laryngospasm can be difficult to predict in a diver, and to our knowledge, no study, thus far, has investigated its incidence in diving. Although laryngospasm at depth appears to be a very rare event,[46] it can have potentially devastating consequences. It may cause the diver to panic and to initiate an uncontrolled ascent. The inability to exhale the expanding air in the lungs during ascent can cause lung overexpansion injury, including subcutaneous or mediastinal emphysema, pneumothorax, and arterial gas embolism.[47]

Conclusions

Otorhinolaryngological disorders related to diving are largely caused by the inability to equalize pressures between anatomical compartments. This can be due to either anatomical obstruction or poor diving technique. While all efforts may be tried to prevent these injuries, because diving has become an increasingly popular sport, with millions of people diving each year, these disorders will likely become even more common presentations to the otorhinolaryngologist.

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REFERENCES

1. Sports and Fitness Industry Association. Scuba Diving Participation Report 2014. https://www.sfia.org/reports/84_Scuba-Diving-Participation-Report-2014. Accessed December 5, 2017.

2. Strutz J. Otorhinolaryngologic aspects of diving sports [in German]. *HNO*. 1993;41(8):401-411.

3. Vanderhoven G, Collard F, Schamp E. Children and diving: medical aspects. eight years' sport medical follow-up of the first scuba diving children in Belgium. *SPUMS J*. 2003;33:70-73.

4. Gonnermann A, Dreyhaupt J, Praetorius M, Baumann I, Plinkert PK, Klingmann C. Otorhinolaryngologic disorders in association with scuba diving [in German]. *HNO*. 2008;56(5):519-523.

5. Strauss MB, Dierker RL. Otitis externa associated with aquatic activities (swimmer's ear). *Clin Dermatol*. 1987;5(3):103-111.

6. Brook I, Coolbaugh JC, Williscroft RG. Effect of diving and diving hoods on the bacterial flora of the external ear canal and skin. *J Clin Microbiol*. 1982;15(5):855-859.

7. Jones EH. Prevention of "swimming pool ear". *Laryngoscope*. 1971;81(5):731-733.

8. Kennedy GE. The relationship between auditory exostoses and cold water: a latitudinal analysis. *Am J Phys Anthropol*. 1986;71(4):401-415.

9. Edmonds C, Lowry C, Pennefather J, Walker R. *Diving and Sub-aquatic Medicine*. 5th ed. London, England: Hodder Headline Group; 2015.

10. Taylor DM, O'Toole KS, Ryan CM. Experienced scuba divers in Australia and the United States suffer considerable injury and morbidity. *Wilderness Environ Med*. 2003;14(2):83-88.

11. Becker GD, Parell GJ. Barotrauma of the ears and sinuses after scuba diving. *Eur Arch Otorhinolaryngol*. 2001;258(4):159-163.

12. Klingmann C, Praetorius M, Baumann I, Plinkert PK. Otorhinolaryngologic disorders and diving accidents: an analysis of 306 divers. *Eur Arch Otorhinolaryngol*. 2007;264(10):1243-1251.

13. Spira A. Diving and marine medicine review part II: diving diseases. *J Travel Med*. 1999;6(3):180-198.

14. Goplen FK, Aasen T, Grønning M, Molvær OI, Nordahl SHG. Hearing loss in divers: a 6-year prospective study. *Eur Arch Otorhinolaryngol*. 2011;268(7):979-985.

15. O'Neill OJ, Weitzner ED. The O'Neill grading system for evaluation of the tympanic membrane: a practical approach for clinical hyperbaric patients. *Undersea Hyperb Med*. 2015;42(3):265-271.

16. Neblett LM. Otolaryngology and sport scuba diving: update and guidelines. *Ann Otol Rhinol Laryngol Suppl*. 1985;115:1-12.

17. Juul A, Kristensen S, Gammelgaard NP, Rasmussen OR. Traumatic rupture of the tympanic membrane: a retrospective study of 37 patients [in Norwegian]. *Tidsskr Nor Laegeforen*. 1987;107(17-18):1544-1547.

18. 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(1):298-301.

19. Edmonds C. Hearing loss with frequent diving (deaf divers). *Undersea Biomed Res*. 1985;12(3):315-319.

20. Lynch JH, Deaton TG. Barotrauma with extreme pressures in sport: from scuba to skydiving. *Curr Sports Med Rep*. 2014;13(2):107-112.

21. Becker GD, Parell GJ. Otolaryngologic aspects of scuba diving. *Otolaryngol Head Neck Surg (1979)*. 1979;87(5):569-572.

22. Bove AA. Diving medicine. *Am J Respir Crit Care Med*. 2014;189(12):1479-1486.

23. Freeman P, Edmonds C. Inner ear barotrauma. *Arch Otolaryngol*. 1972;95(6):556-563.

24. Shupak A. Recurrent diving-related inner ear barotrauma. *Otol Neurotol*. 2006;27(8):1193-1196.

25. 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):447-454.

26. Sarnaik AP, Vohra MP, Sturman SW, Belenky WM. Medical problems of the swimmer. *Clin Sports Med*. 1986;5(1):47-64.

27. Klingmann C, Benton PJ, Ringleb PA, Knauth M. Embolic inner ear decompression illness: correlation with a right-to-left shunt. *Laryngoscope*. 2003;113(8):1356-1361.

28. Ignatescu M, Bryson P, Klingmann C. Susceptibility of the inner ear structure to shunt-related decompression sickness. *Aviat Space Environ Med*. 2012;83(12):1145-1151.

29. Klingmann C. Inner ear decompression sickness in compressed-air diving. *Undersea Hyperb Med*. 2012;39(1):589-594.

30. Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. *Lancet*. 2011;377(9760):153-164.

31. Shupak A, Gil A, Nachum Z, Miller S, Gordon CR, Tal D. Inner ear decompression sickness and inner ear barotrauma in recreational divers: a long-term follow-up. *Laryngoscope*. 2003;113(12):2141-2147.

32. Moon RE, Sheffield PJ. Guidelines for treatment of decompression illness. *Aviat Space Environ Med*. 1997;68(3):234-243.

33. Gempp E, Louge P, de Maistre S, Morvan J-B, Vallée N, Blatteau J-E. Initial severity scoring and residual deficit in scuba divers with inner ear decompression sickness. *Aerosp Med Hum Perform*. 2016;87(8):735-739.

34. Motamed M, Pau H, Daudia A, Narula A. Recurrent facial nerve palsy on flying. *J Laryngol Otol*. 2000;114(9):704-705.

35. Onundarson PT. Acute nose-blow palsy: a pneumatic variant of sudden facial paralysis. *N Engl J Med*. 1987;317(19):1227.

36. Molvaer OI, Eidsvik S. Facial baroparesis: a review. *Undersea Biomed Res*. 1987;14(3):277-295.

37. Roydhouse N. 1001 Disorders of the ear, nose and sinuses in scuba divers. *Can J Appl Sport Sci*. 1985;10(2):99-103.

38. Bellini MJ. Blindness of a diver following sinus barotrauma. *J Laryngol Otol*. 1987;101:386-389.

39. Goldmann RW. Pneumocephalus as a consequence of barotrauma. *JAMA*. 1986;255(22):3154-3156.

40. Murrison AW, Smith DJ, Francis TJ, Counter RT. Maxillary sinus barotrauma with fifth cranial nerve involvement. *J Laryngol Otol*. 1991;105(3):217-219.

41. Edmonds C. Dysbaric peripheral nerve involvement. *SPUMS J*. 1991;21(4):190-197.

42. Gunn DJ, O'Hagan S. Unilateral optic neuropathy from possible sphenoidal sinus barotrauma after recreational scuba diving: a case report. *Undersea Hyperb Med*. 2013;40(1):81-86.

43. Hexdall EJ, Butler FK. Transient vision loss at depth due to presumed barotraumatic optic neuropathy. *Undersea Hyperb Med*. 2012;39(5):911-914.

44. Schipke JD, Cleveland S, Drees M. Sphenoid sinus barotrauma in diving: case series and review of the literature. *Res Sports Med*. 2017;10:1-14.

45. Skevas T, Baumann I, Bruckner T, Clifton N, Plinkert PK, Klingmann C. Medical and surgical treatment in divers with chronic rhinosinusitis and paranasal sinus barotrauma. *Eur Arch Otorhinolaryngol*. 2012;269(3):853-860.

46. Papadodima SA, Athanaselis SA, Skliros E, Spiliopoulou CA. Forensic investigation of submersion deaths. *Int J Clin Pract*. 2010;64(1):75-83.

47. US Navy. *US Navy Diving Manual, 6th Revision*. Washington, DC: US Government Printing Office; 2006.