

[Primary Care]

Otologic Hazards Related to Scuba Diving

Tiffany A. Glazer, MD,*† and Steven A. Telian, MD†

Context: As of 2015, more than 23 million scuba diver certifications have been issued across the globe. Given the popularity of scuba diving, it is incumbent on every physician to know and understand the specific medical hazards and conditions associated with scuba diving.

Evidence Acquisition: Sources were obtained from PubMed, MEDLINE, and EBSCO databases from 1956 onward and ranged from diverse fields including otologic reviews and wilderness medicine book chapters.

Study Design: Clinical review.

Level of Evidence: Level 5.

Results: Otologic hazards can be categorized into barotrauma-related injuries or decompression sickness.

Conclusion: When combined with a high index of suspicion, the physician can recognize these disorders and promptly initiate proper treatment of the potentially hazardous and irreversible conditions related to scuba diving.

Keywords: scuba diving; barotrauma; decompression sickness

According to the Professional Association of Diving Instructors (PADI), as of February 2015, more than 23 million diver certifications have been issued across the globe, with a mean 900,000 certifications in more than 200 countries issued annually. Roughly two-thirds of these certified divers are men (mean age: men, 30 years; women, 27 years).¹⁶ Given the popularity of scuba diving, it is incumbent on every physician to know and understand the specific medical hazards and conditions associated with scuba diving.

Barometric pressure at sea level is 100 kPa and increases linearly underwater with every 10 meters of descent, adding 100 kPa. As depth increases, the diver breathes gas of increased density while the diving equipment provides oxygen and allows for elimination of carbon dioxide. Increased depth and pressure also leads to a decrease in gas volume and an increase in the amount of gas dissolved in body tissues. The otologic conditions of barotrauma and decompression sickness that can result from scuba diving are related to the behavior of gas changing under pressure as the diver descends. To understand this behavior more thoroughly, a brief overview of 2 gas laws is necessary. Boyle's law explains pressure-related diving conditions like barotrauma, stating that at a constant temperature, the volume of gas inversely varies with pressure applied. Henry's law explains decompression sickness and

states that the amount of a given gas that is dissolved in a liquid at a given temperature is directly proportional to the partial pressure of that gas. These 2 laws are essential to understanding and treating pressure-related diving disorders that develop because of problems caused by the mechanical effects of pressure on closed air spaces (barotrauma) or problems caused by breathing gases at elevated partial pressure (decompression sickness). Both of these disorders will be discussed in detail in this review.

BAROTRAUMA

Barotrauma is the most common medical problem in scuba diving and can involve any structure that leads to entrapment of gas in a closed space. If a diver does not equalize pressure in gas-filled body spaces during descent, these spaces will decrease in volume in accordance with Boyle's law. Gas-filled body spaces located within a bone space cannot collapse, so the space they take up is replaced by engorgement of the mucous membranes, often followed by hemorrhage.⁸ The risk of barotrauma is greater near the surface of the water as it is here that a small change in depth may lead to a large change in relative gas volume. Barotrauma can occur in any structure or combination of structures that leads to entrapment of gas in a

From the †Department of Otolaryngology–Head and Neck Surgery, University of Michigan Health System, Ann Arbor, Michigan

*Address correspondence to Tiffany A. Glazer, MD, Department of Otolaryngology–Head and Neck Surgery, University of Michigan Health System, 1500 East Medical Center Drive, 1904 TC, SPC 5312, Ann Arbor, MI 48109-5312 (email: tglazer@med.umich.edu).

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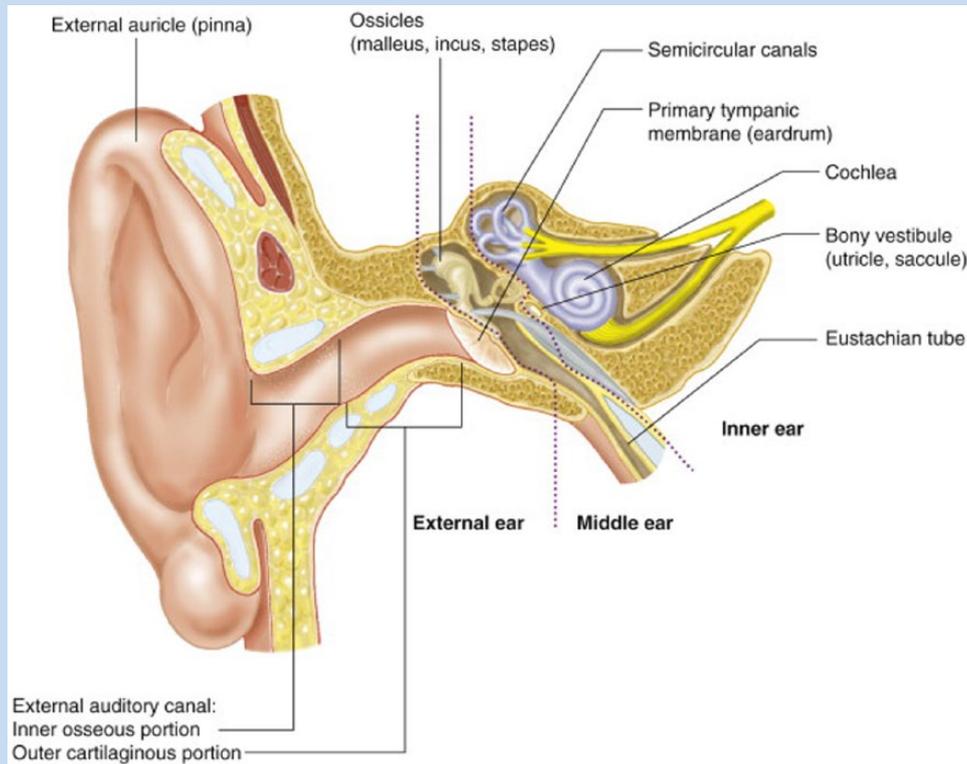


Figure 1. Basic anatomy of the external, middle, and inner ear. Reprinted with permission from Wenig.²²

closed space, including the ears (inner, middle, and/or outer ear spaces; Figure 1), paranasal sinuses, lungs, gastrointestinal tract, portion of the face under a face mask, and skin trapped under a fold in a drysuit.¹⁵

Middle Ear Barotrauma (Barotitis Media)

Middle ear barotrauma, also known as “middle ear squeeze,” is the most common diving-related medical disorder, affecting more than 40% of divers at some point.^{5,15} As the diver descends, hydrostatic water pressure forces the tympanic membrane inward and the volume within the middle ear space is reduced. The diver is taught to add air into the middle ear by ventilating through the eustachian tube. This is accomplished by performing a Valsalva maneuver, which equalizes the pressure in the middle ear cavity with the external ambient pressure. Middle ear barotrauma occurs when the eustachian tube cannot open to equilibrate pressure despite a Valsalva maneuver. A diver will begin to have otalgia with a 60 mm Hg pressure differential between the middle ear space and ambient water pressure. When a pressure differential of 90 mm Hg is reached between the middle ear space and the nasopharynx, the eustachian tube cannot be opened and Valsalva maneuvers will be unsuccessful.² The diver must ascend to equalize the middle ear pressure with ambient pressure. Middle ear edema, transudation, and hemotympanum may occur. The tympanic membrane becomes displaced medially (inward), and if the pressure is great enough and remains unaddressed by the diver,

rupture of the membrane may occur. The failure of expanding gas to vent through the eustachian tube may occasionally lead to middle ear barotrauma during ascent as well, referred to as “reverse squeeze.”²

If a diver does not equalize pressure in the ear with a Valsalva maneuver, symptoms of middle ear barotrauma may occur. The main symptom is otalgia that increases with depth. Relief of pain without proper equalization of middle ear pressure usually indicates that the tympanic membrane has ruptured. Once above water, divers will typically complain of a sensation of pressure, fullness, and conductive hearing loss. If cold water enters an ear with a ruptured tympanic membrane, one may develop severe vertigo due to unilateral vestibular stimulation by the caloric effect.¹⁵ Late complications include chronic tympanic membrane perforation and chronic otitis media. Rarely, the tympanic portion of the facial nerve is injured by increased middle ear pressure, and a temporary facial paralysis results. This occurs when middle ear pressure exceeds the capillary pressure of the facial nerve and causes an ischemic neuropraxia. The presence of middle ear barotrauma prohibits future diving until it has resolved.

Treatment of middle ear barotrauma includes topical nasal and systemic decongestants to decrease middle ear edema and transudate. If purulent otorrhea is observed and infection has developed, topical antibiotic drops should be prescribed. The majority of tympanic membrane perforations heal spontaneously within 1 to 3 months if otorrhea is controlled,

and normal eustachian tube function is restored.¹⁵ Prevention is key for barotitis media. Training must emphasize the importance of early and ideal pressure equalization of the middle ear space. Concomitant inner ear barotrauma is discussed next and should be ruled out in all cases of middle ear barotrauma.¹

Inner Ear Barotrauma

Inner ear barotrauma is the most serious form of aural barotrauma because of the potential for lasting injury to the cochleovestibular system, which may lead to permanent deafness and/or vestibular dysfunction. Inner ear barotrauma may develop if a diver performs a forceful Valsalva maneuver in an attempt to equalize middle ear pressure with the eustachian tube blocked or if the descent occurs too quickly. During descent, the tympanic membrane is pressed inward, pushing the stapes against the oval window. Because perilymph and endolymph are not compressible, the resulting increased pressure may cause the round window membrane to bulge outward. The diver may attempt a forceful Valsalva maneuver to equalize the middle ear. This will raise intracranial pressure, which may be propagated through the internal auditory canal or the perilymphatic duct to the inner ear, causing the round or oval window to rupture. Rupture of either window can also occur by a sudden increase in middle ear pressure by a forceful Valsalva. This sudden pressure may cause any of several types of injury to the cochleovestibular apparatus, including hemorrhage within the inner ear, rupture of the Reissner membrane leading to mixing of endolymph and perilymph, fistulization of the oval or round window with development of a perilymph leak, or a mixed injury involving any number of these conditions.¹⁷ During ascent, the expanding middle ear gas may be forced through the perilymph fistula and enter either into the scala tympani or scala vestibule, which may damage cochlear or vestibular structures leading to permanent hearing loss. Although they typically occur concomitantly, inner ear barotrauma may occur independently of middle ear barotrauma.^{14,17} Vigorous isometric exercise such as yoga after a dive may complete an incipient or partial membrane rupture.

Severity may vary but divers suffering from inner ear barotrauma will classically complain of sensorineural hearing loss, loud roaring tinnitus, and persistent vertigo after diving. Treatment varies from observation and symptomatic treatment of vertigo and nausea with anti-emetics and benzodiazepines to surgical exploration for repair of the round or oval window. Tinnitus and reduced hearing may become chronic, particularly if a fistula is present and no treatment is provided. Divers who exhibit clinical evidence of inner ear barotrauma with intact round and oval windows may have suffered a pressure injury to the organ of Corti and/or the membranous labyrinth.¹⁸

There is no clinical basis for definitively distinguishing a perilymphatic fistula from inner ear barotrauma due to an internal membrane break. Traditional initial treatment is to recommend complete bed rest with the diver's head elevated 30° and avoidance of any activities that can increase cerebrospinal fluid pressure as this may aggravate a

perilymphatic leak.¹⁷ Vertigo and nausea can be managed symptomatically with anti-emetics and benzodiazepines. If inner ear function persists or continues to deteriorate despite conservative management, the patient may require an exploratory tympanotomy with patching of the round or oval window. Although no consensus exists as to how long to wait before surgical intervention, it has been recommended to explore the ear if the diver shows no improvement after 24 hours.¹⁵

External Auditory Canal Barotrauma

External auditory canal (EAC) barotrauma is less common but can occur if the diver wears a tight-fitting wetsuit hood or drysuit hood that can trap air in the EAC and lead to a painful "external ear squeeze" during descent. It may also occur if the EAC is blocked by cerumen, exostoses (bony growth in the outer ear canal), or a foreign object. Signs and symptoms of EAC barotrauma include pain, swelling, erythema, petechiae, or hemorrhagic blebs of the EAC wall. Blebs should not be incised. Antibiotic otic drops can be used to prevent infection due to contamination with seawater. EAC barotrauma can be prevented by breaking the seal of the wetsuit hood to allow water to fill the EAC before descent. Ear plugs should never be worn when scuba diving.

Alternobaric Vertigo

Alternobaric vertigo is a specific type of middle ear barotrauma that occurs if the reduction of middle ear pressure is not equivalent in the bilateral middle ear spaces. If the pressure differential exceeds a threshold of 45 mm Hg, asymmetric stimulation of the labyrinths will occur, resulting in what is called alternobaric vertigo.⁹ Symptoms include nausea, vomiting, disorientation, and generalized malaise. The sensation of vertigo may persist for 1 to 2 hours after ascent but gradually disappears without therapy. Divers may be particularly susceptible to alternobaric vertigo if they have had previous injury or infection of either labyrinth. In susceptible divers, use of moderate doses of antihistamines or decongestants prior to diving may prevent symptoms, although these medications are not recommended for routine use before diving as they may potentiate reverse squeeze. Although symptoms are usually mild, they may occur underwater, which is a serious problem that may lead to aspiration and drowning.¹¹ The disorder must be differentiated from inner ear decompression sickness, which is described later and is usually associated with deeper, prolonged diving.

DECOMPRESSION SICKNESS

In accordance with Henry's law, as a diver breathes under increased pressure, the tissues are loaded with increased quantities of oxygen and nitrogen. Increased oxygen can be used in tissue metabolism but nitrogen cannot as it is physiologically inert. Therefore, as a diver descends, the nitrogen content of tissue increases proportionally to ambient pressure and in relation to fat tissue content as nitrogen is 5

times more soluble in fat than in water.⁶ As a diver ascends, the ambient pressure decreases and a state of supersaturation is created. The sum of the gas tensions in the tissue may exceed the absolute ambient pressure and free gas is liberated from the tissues, leading to the onset of decompression sickness (DCS). The liberated gas can disturb organ function by blocking arteries, veins, and lymphatic vessels. In addition, its expansion can rupture or compress tissues. When the gas is liberated into a space with rigid boundaries, it may lead to compartment syndrome. Gas may also form within cells causing the rupture of the cell membrane. Finally, reactions at the interface of blood and gas bubbles can activate the clotting cascade.^{3,6} DCS can be avoided if rates of ascent are controlled with planned stops during the ascent, as specified in decompression tables such as those provided by the US Navy.²⁰ However, severe DCS may occur despite adherence to decompression tables.

The chance of suffering DCS on any single dive is about 4 in 10,000 in warm water and 59 per 10,000 in cold water.²¹ Risk factors for DCS are those that enhance the accumulation of inert gases in tissues or slow their release. These include advanced age, female sex, obesity, elevated arterial carbon dioxide tension, low water temperature, poor physical fitness, and the level of physical activity during the dive. Repeated dives are also a major risk factor since a considerable quantity of nitrogen may remain in the tissues after a dive, increasing the risk of DCS during subsequent dives.^{7,20}

Decompression sickness is classified into type I and type II based on clinical manifestations. Type I involves a mild insult, with the most common manifestation being localized joint pain, also known as “the bends.” Typically, it will develop within 1 hour of ascent, and symptoms may gradually become more severe during the following 24 to 36 hours. The joints most commonly affected are the elbow and shoulder. Pain is usually not exacerbated by motion, and tenderness and inflammation are very uncommon. Other symptoms of type I DCS include pruritus and skin rashes due to the presence of gas bubbles in subcutaneous fat. Typically, this will resolve on its own but if skin marbling or localized edema occurs, the diver would require recompression treatment.¹

Type II is the more severe form of DCS and may lead to permanent neurologic injury and death. Symptoms start with a feeling of malaise and fatigue and occur 10 to 30 minutes after ascent. The mechanism of neurologic insult is related to paradoxical gas embolization. Gas bubbles may escape pulmonary filtration and enter the arterial circulation through a patent foramen ovale or an atrial-septal defect or they may be generated de novo as microemboli within the arterial circulation. The gas bubbles go on to occlude capillaries or damage the blood-brain barrier and result in a neurologic insult. Divers who have suffered from DCS have been reported to have a higher prevalence of patent foramen ovale.¹³ Poorly perfused areas of the nervous system or areas with high fat content are at a particularly high risk of DCS.⁶

When the spinal cord is involved, divers may complain of paresthesias that may progress to numbness, paraparesis,

referred abdominal pain, fecal incontinence, or bladder paralysis. When the cerebrum is involved, divers may complain of severe headache, blurred vision, diplopia, tunnel vision, dysarthria, dizziness, sensorineural hearing loss, tinnitus, and/or vertigo, as well as mental status and personality changes. Severe cerebral involvement may lead to convulsions and death.^{3,6}

Inner ear DCS is a type II manifestation and results from blockage of the microcirculation of the inner ear, hemorrhage, and transudation of protein within the cochlea. This irritates the endosteum within the bony semicircular canals, initiating osteoblastic differentiation and leading to fibroosseous labyrinthitis.⁴

Treatment for DCS involves urgent transfer to a recompression chamber for the administration of inhaled oxygen at the highest possible concentration. The time to administration of hyperbaric oxygen is one of the main determinants of outcome.¹²

Transportable single-person rescue chambers are commonly available and can be used as a diver is transferred to a recompression chamber.¹⁰ Hyperbaric oxygen is thought to work by eliminating gas bubbles and alleviating damage to hypoxic tissue. Treatment is given according to the US Navy treatment tables, and the recompression protocol is selected according to the severity of illness (type I or type II) and the patient's response during therapy.²⁰ Hydration with intravenous fluids is also of utmost importance as the hemodynamic changes that occur during severe DCS lead to hemoconcentration. High doses of steroids and mannitol can be given to reduce local edema in the nervous system and maintain an intact blood-brain barrier. Another therapeutic option first introduced by the US Navy in 1958 is administration of a mixture of helium and oxygen.¹⁹ Although the exact mechanism of action is unclear, it seems that when such mixtures are used in recompression, nitrogen is eliminated faster from the tissues as it is much more soluble than helium.

CONCLUSION

Given the millions of recreational, commercial, and military divers worldwide with nearly 1 million new divers trained each year, all physicians can benefit from a basic knowledge of barotrauma and decompression sickness. When combined with a high index of suspicion, the physician can recognize these disorders and promptly initiate proper treatment of the potentially hazardous and irreversible conditions related to scuba diving.

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