Chapter 214: Diving Disorders

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**FIGURE 214-1.**

**INTRODUCTION**

Millions of recreational, commercial, and scientific dives are logged annually, and the vast majority of dives are completed without incident. However, there are physiologic effects and injuries relatively unique to the underwater environment. Generally, these effects and injuries are secondary to pressure changes on the submerged human body and the breathing of compressed gas. This chapter outlines the most common diving injuries: barotrauma of descent (otic, sinus, and pulmonary), barotrauma of ascent (pulmonary overinflation syndromes and arterial gas embolism), decompression sickness, immersion pulmonary edema, oxygen toxicity, and nitrogen narcosis.

**THE GAS LAWS**

Understanding diving injuries requires familiarity with the three relevant gas laws most pertinent to diving: Boyle's law, Dalton's law, and Henry's law.

Boyle's law states that given a constant temperature, the pressure and volume of an ideal gas are inversely related. That is, if pressure is doubled, the volume of gas is halved. This law is stated as: $P_1V_1 = P_2V_2$.

Pressure can be measured in a variety of units. The International System of Units defines pressure using the pascal (Pa). Other commonly used units of pressure include millimeters of mercury (mm Hg), torr, pounds per square inch (psi), bar, or atmosphere (atm): $1 \text{ atm} = 760 \text{ mm Hg} = 760 \text{ torr} = 14.7 \text{ psi} = 1.013 \text{ bar} = 101,325 \text{ Pa} = 101.325 \text{ kPa}$. Additionally, pressure in diving settings is often described using feet of seawater (fsw) or meters of seawater (see below). In this chapter, we use atm, mm Hg, and fsw for pressure units.

Because of the high density of water, a relatively small change in depth causes a great change in pressure. The weight of seawater produces a change of 1 atm for each 33 ft of depth. For freshwater, pressure increases 1 atm for each 34 ft of depth. Therefore, the pressure exerted on a diver at a depth of 33 ft in seawater = 1 atm for the seawater + 1 atm for the atmosphere above the water = 2 atmospheres absolute (ATA). A diver at 165 ft of seawater would experience 6 ATA of pressure (1 atm for each 33 ft of seawater = 5 atm + 1 atm for atmospheric pressure at sea level).
Thus, **Boyle's law** dictates as a diver descends in the water column, the volume of air-containing structures will decrease. For example, if the lungs contain volume $V$ at the surface, a diver who descends to 33 ft of seawater holding his or her breath would have a lung volume of $\frac{1}{2}V$. If the diver then breathes compressed air at this depth (from scuba equipment or from a surface-supplied source of gas), lung volume would return to $V$. If the diver then ascends to the surface without exhaling, lung volume would be $2V$ at the surface. This pressure–volume relationship governed by Boyle's law is important in the etiology of injuries due to barotrauma and produces the volume changes of bubbles in the tissues and circulation that are associated with recompression (hyperbaric) therapy.

**Dalton's law** states that the total pressure exerted by a mixture of gases is the sum of the partial pressures of each gas. Therefore, the partial pressure of a given component of a gas mixture will increase as the ambient pressure increases, although the proportion of gas in the mixture remains constant. The partial pressure of nitrogen in air at sea level is approximately 600 mm Hg or 0.79 ATA (the fraction of nitrogen in air, $0.79 \times 760$ mm Hg or 1 ATA). At a depth of 99 fsw, the partial pressure of nitrogen in air would be $4 \times 600 = 2400$ mm Hg (or 3.16 ATA).

**Henry's law**, which states that at equilibrium the quantity of a gas in solution in a liquid is proportional to the partial pressure of the gas, along with Dalton's law, explains the uptake of inert gas into tissues when breathing compressed air at depth. It is the uptake of inert gas that is intrinsic to the development of decompression sickness.

**BAROTRAUMA OF DESCENT**

The clinical conditions resulting from barotrauma of descent are barotitis (ear squeeze), external ear squeeze, sinus barotrauma, inner ear barotrauma, and face, tooth, or dry-suit squeeze (**Table 214-1**).
TABLE 214-1

Summary of Barotrauma of Descent and Ascent

<table>
<thead>
<tr>
<th>Barotrauma</th>
<th>Clinical Features</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barotrauma of descent</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Otic barotrauma (“ear squeeze”)</td>
<td>Pain, fullness, vertigo, conductive hearing loss from inability to</td>
<td>Decongestants, consider antibiotics</td>
</tr>
<tr>
<td>Sinus barotrauma (“sinus squeeze”)</td>
<td>equalize middle ear pressure</td>
<td>Decongestants, consider antibiotics</td>
</tr>
<tr>
<td>Inner ear barotrauma</td>
<td>Pain over affected sinus, possible bleeding from nares</td>
<td>Head of bed up, no nose blowing, antivertigo medications, and urgent</td>
</tr>
<tr>
<td></td>
<td>Sudden onset of sensorineural hearing loss, tinnitus, severe vertigo after forced</td>
<td>otolaryngology consultation as some surgeons advocate early exploration</td>
</tr>
<tr>
<td></td>
<td>Valsalva</td>
<td></td>
</tr>
<tr>
<td>Barotrauma of ascent</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary overinflation syndromes (pulmonary</td>
<td>Dyspnea, chest pain, subcutaneous air, extra-alveolar air on radiograph;</td>
<td>Pneumomediastinum requires only symptomatic care and does not require</td>
</tr>
<tr>
<td>barotrauma)</td>
<td>usually occurring secondary to rapid or uncontrolled ascent</td>
<td>recompression</td>
</tr>
<tr>
<td>Arterial gas embolism</td>
<td>Neurologic symptoms occurring immediately after uncontrolled or rapid ascent or</td>
<td>Pneumothorax requires drainage and does not require recompression (if</td>
</tr>
<tr>
<td></td>
<td>neurologic symptoms in the setting of pulmonary barotrauma</td>
<td>recompression is instituted for treatment of arterial gas embolism, then</td>
</tr>
<tr>
<td></td>
<td></td>
<td>the pneumothorax must be drained before recompression)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Airway, breathing, circulation, high-flow oxygen, IV hydration, immediate</td>
</tr>
<tr>
<td></td>
<td></td>
<td>recompression (hyperbaric oxygen), consider adjunctive lidocaine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Any neurologic symptom in the setting of documented pulmonary barotrauma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>must be treated as an arterial gas embolism</td>
</tr>
</tbody>
</table>

**PATHOPHYSIOLOGY**

During descent, the volume of gas in all air-containing body cavities decreases. The air space in the middle ear makes the tympanic membrane the tissue most commonly affected by this phenomenon, if active measures such as "clearing the ears" with a Valsalva or other maneuvers are not successful. As the volume
of gas decreases, the tympanic membrane is bent inward, causing a feeling of fullness or pain in the ear. Forcing air through the Eustachian tube with a Valsalva maneuver will equalize the pressure between the middle ear and external ear canal by filling the middle ear with additional gas. Generally, divers who experience pain in an ear during descent will attempt to clear the ear and, if unsuccessful, will ascend to decrease the pressure differential and attempt equalizing again. If the diver is unsuccessful in equalizing and continues the descent, prolonged pain and injury to the tympanic membrane may result, known as barotitis or "ear squeeze."

**BAROTITIS (EAR SQUEEZE)**

Barotitis can range from symptoms of pain or fullness without otoscopic changes, to hemorrhage within the tympanic membrane or hemorrhage into the middle ear with hemotympanum. Ultimately, the tympanic membrane may rupture, resulting in relief of the pain but also possibly causing an influx of water into the middle ear. This, in turn, might cause calorically induced vertigo and potential panic, drowning, or other injury.

Barotitis is treated conservatively with analgesics and decongestants. If tympanic membrane rupture occurs, antibiotics can be prescribed, especially if the diving occurred in contaminated water. Divers with perforated tympanic membranes should refrain from diving until the perforation heals. Most such perforations heal without difficulty, but referral to an otolaryngologist is appropriate for individuals with larger perforations or when healing does not occur. Divers with barotitis without perforation should refrain from diving until the diver is again able to equalize the pressure in the affected middle ear.

**EXTERNAL EAR SQUEEZE**

If the external canal is occluded by cerumen or an ear plug, the inability to equalize pressure between the external canal and the tympanic membrane causes the bending of the tympanic membrane outward, producing an injury called "external ear squeeze" that produces pain and tympanic membrane hemorrhage.

**SINUS BAROTRAUMA**

If the ostia to the sinuses are occluded, air cannot enter the sinuses during descent to equalize the increasing pressure. This causes pain and mucosal edema and can lead to submucosal hemorrhage and stripping of the sinus mucosa from bone, hemorrhage (often causing bleeding from the nose into the mask), and, rarely, paresthesias in the infraorbital nerve distribution. A similar traumatic neuropathy can occur to the facial nerve with middle ear barotrauma. Sinus barotrauma is treated with conservative measures, including decongestants and, possibly, antibiotics.

**INNER EAR BAROTRAUMA**

The inner ear is also susceptible to barotrauma, occasionally causing significant, long-term damage. If a diver attempts a forceful Valsalva maneuver to equalize the middle ear against an occluded Eustachian tube, the pressure differential between the cerebrospinal fluid, transmitted through the vestibular and cochlear
structures and the middle ear air space, can cause rupture of the oval or round window, fistulization of the window, tearing of the vestibular membrane, or a combination of such injuries. Additionally, if the diver is able to open the Eustachian tube in this situation, a rapid increase in middle ear pressure may occur. This pressure wave is transmitted to the inner ear and can also cause a similar injury. Divers with inner ear barotrauma will generally present with unilateral roaring tinnitus, sensorineural hearing loss, and profound vertigo. A "fistula test" may be positive—that is, insufflation of the tympanic membrane on the affected side causes the eyes to deviate to the contralateral side. Because this injury usually occurs on descent and divers will provide a history of difficulty clearing the ears, this condition can usually be easily differentiated from other causes of vertigo, such as inner ear decompression sickness, cerebral arterial gas embolism, or alternobaric vertigo (discussed below).

Immediate complications of inner ear barotrauma are potential panic or disorientation, leading to possible drowning or a rapid ascent that predisposes the diver to pulmonary barotrauma. Divers with barotraumatic injuries to the inner ear require urgent otolaryngologic evaluation. Treatment is controversial, with some authors advocating immediate exploration and others suggesting a trial of bed rest (head upright), medications to control vertigo, and mechanical measures to reduce cerebrospinal fluid pressure spikes (e.g., stool softeners, no nose blowing). These authors reserve exploration for patients whose symptoms do not respond to conservative therapy or patients with severe hearing defects or significant abnormalities on an oculo-nystagmogram. Divers with potential inner ear barotrauma who will be treated with hyperbaric oxygen for decompression sickness or cerebral arterial gas embolism require emergent tympanostomy, because hyperbaric treatment will recreate the same pressure differentials that caused the injury, potentially causing more perilymph leakage and, possibly, worsening the injury.²

FACE SQUEEZE, TOOTH SQUEEZE, AND DRY-SUIT SQUEEZE

Other air-containing structures can be compressed during descent, producing "squeeze" symptoms. A face squeeze occurs when air is not added to the facemask during descent, causing the face and eyes to be forced into the collapsing mask. This can produce facial bruising, conjunctival injection or hemorrhage, changes in vision, and, rarely, retrobulbar hemorrhage. The latter could be a true ophthalmologic emergency. A tooth squeeze occurs when air spaces inside a tooth—due to decay, a filling, or an abscess—become compressed during descent. A dry-suit squeeze occurs when suit folds are compressed into the underlying skin, producing local trauma manifested by painful red streaks.

BAROTRAUMA OF ASCENT

The clinical conditions of barotrauma of ascent are alternobaric vertigo, pulmonary barotrauma, arterial gas embolism, and decompression sickness (Table 214-1).

ALTERNOBARIC VERTIGO

During ascent, the physics of gas in air-containing organs is, of course, opposite that of descent—that is, air will expand as the pressure decreases. Air will flow through the ostia of the sinuses, and the expanding air in
the middle ear will open the Eustachian tube (much like during takeoff in an airplane). Should air be trapped temporarily in one middle ear cavity, the pressure differential may cause unequal vestibular impulses to the brain, resulting in vertigo (alternobaric vertigo). This is usually transient and generally requires no specific treatment.

**PULMONARY BAROTRAUMA**

Air also expands within the lungs with ascent. If a diver breathing compressed air ascends with a closed glottis (holds breath, coughs, vomits), most frequently seen in a rapid, panicked, out-of-air ascent, the expanding air may cause parenchymal lung injury. This can occur even in shallow water (e.g., a swimming pool). Pulmonary barotrauma, also called *pulmonary overinflation* or *burst lung syndrome*, can lead to pneumomediastinum. This generally only requires symptomatic treatment and may be subtle on the chest radiograph.³ Mediastinal air can track superiorly into the neck, resulting in subcutaneous air on physical examination or air on a cervical spine radiograph. Pulmonary overinflation injury can cause pneumothorax, requiring aspiration or air or tube thoracostomy. If air enters the pulmonary venous circulation, embolization of the gas through the arterial system occurs. The most sensitive end-organ to such embolization is the brain, and *cerebral arterial gas embolism* is the term applied to this condition, although the air emboli distribute to other tissues and organs.⁴ Any neurologic symptom or sign referable to the circulation to the CNS in the setting of barotrauma associated with ascent should be considered to be secondary to cerebral arterial gas embolism. The symptoms, signs, and treatment are discussed below in the section "Arterial Gas Embolism."

Pulmonary barotrauma (Figure 214-1) can occur without a rapid ascent or closed glottis in divers with congenital cysts, obstructive pulmonary disease, or other processes that cause air trapping.

**FIGURE 214-1.**

Pulmonary barotrauma. Note the air in the mediastinum in this radiography *(arrow)*. There is also air in the soft tissues of the neck.
OTHER BAROTRAUMAS OF ASCENT

An air pocket underneath a tooth may equilibrate with ambient pressure while diving, only to expand during ascent. This produces severe pain and may dislodge a filling or fracture a tooth. Swallowed air during diving may expand during ascent, rarely producing gastric distention and abdominal cramps.

DECOMPRESSION SICKNESS

The pathophysiology of decompression sickness is related to the obstructive and inflammatory effects of inert gas bubbles in tissues and the vascular system. Decompression sickness may occur in divers breathing compressed air, caisson workers, high-altitude pilots, or astronauts. Bubbles may form when a body with additional inert gas in solution experiences a decrease in ambient pressure that causes liberation of the gas. Uptake of inert gas occurs at different rates in different tissues.
The U.S. Navy publishes dive tables to provide the limits to a dive (measured by bottom depth and time) that can be undertaken without a decompression stop ("no decompression" or "no stop" dives). Other Navy tables provide a variety of decompression schedules for longer dives. A multitude of dive computers, often using proprietary mathematical models, provide divers with relatively safe diving limits. Decompression sickness is unlikely to occur if the limits of the dive tables or dive computer are followed, but compliance with dive table limits or a dive computer does not completely eliminate risk.

Bubbles are necessary but not sufficient by themselves to cause decompression sickness; bubbling occurs after many dive profiles that do not lead to decompression sickness. Obviously, there must be a threshold at which the bubble load causes symptoms. The exact mechanism of bubble formation is not known, although preexisting gas micronuclei in the circulation likely form a nidus for gas accumulation. This is inferred, because the energy required to form bubbles de novo is much higher than the energy state caused by the saturation of inert gas in tissue. Bubbles may form directly in tissues or the circulation (usually the low-pressure venous circulation). Classically, it is thought that bubbles directly obstruct blood flow, leading to direct ischemia. Also, the air–blood and air–endothelial interfaces initiate a variety of inflammatory and thrombotic processes; activate the endothelium, leading to neutrophil adhesion and activation; and change the permeability of the endothelium, resulting in third spacing of fluid. In addition, decompression stress induces the production of microparticles, which are lipid bilayer–enclosed membranous vesicles extruded from vascular endothelial and other cells. Injection of these microparticles in animal models creates a clinical condition consistent with decompression sickness.

There are no current definitive diagnostic criteria for decompression sickness. The San Diego Diving and Hyperbaric Organizations criteria use a point system to identify dive injuries resulting in decompression sickness with a high degree of specificity. This is helpful to create databases of divers with decompression sickness to study outcomes and allow study of adjunctive therapies. Unfortunately, this system has relatively low sensitivity. Studies of therapies for decompression sickness often lack an acceptable case definition of decompression sickness.

**CLINICAL FEATURES**

The most commonly used classification divides decompression sickness into two (or sometimes three) main groups (Table 214-2). We focus on type I and II for clarity. Type I is also called "pain-only" decompression sickness and involves the joints, extremities, and skin ("cutis marmorata"). Lymphatic obstruction can occur in type I, causing lymphedema, which usually takes days to resolve despite recompression therapy. Type II involves the CNS (mainly the spinal cord in compressed air divers and the brain in high-altitude decompressions), vestibular symptoms ("staggers"), and cardiopulmonary symptoms ("chokes"). To further complicate the nomenclature and classification of decompression sickness, it can also occur when an arterial gas embolism (see below) causes inert gas to come out of solution after a dive profile that would otherwise not be expected to cause decompression sickness (called type III). Some advocate the use of the alternate term *decompression illness*, instead of differentiating between decompression sickness and cerebral arterial gas embolism, to encompass all pathologic syndromes following a reduction in ambient pressure.
<table>
<thead>
<tr>
<th>Classification</th>
<th>Clinical Features</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I: &quot;pain-only&quot; DCS</td>
<td>Deep pain in joints and extremities, unrelieved but not worsened with movement</td>
<td>Usually single joint, most commonly knees and shoulders</td>
</tr>
<tr>
<td></td>
<td>Skin changes—mottling, pruritus, and color changes</td>
<td>Lymphatic obstruction can occur and takes days to resolve despite recompression therapy</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type II: &quot;serious&quot; DCS</td>
<td>Pulmonary (&quot;chokes&quot;)—cough, hemoptysis, dyspnea, and substernal chest pain</td>
<td>Has a tendency to affect the lower cervical and thoracic regions may see scattered lesions</td>
</tr>
<tr>
<td></td>
<td>Cardiovascular collapse can occur</td>
<td>Autonomic dysfunction seen</td>
</tr>
<tr>
<td></td>
<td>Neurologic—sensation of truncal constriction(^{\circ}) ascending paralysis, usually rapid in onset</td>
<td>Usually occurs after deep, long dives</td>
</tr>
<tr>
<td></td>
<td>Vestibular (&quot;staggers&quot;)—vertigo, hearing loss, tinnitus, and disequilibrium</td>
<td></td>
</tr>
<tr>
<td>Type III: combination of DCS and arterial gas embolism</td>
<td>Symptoms of DCS II noted above plus a variety of stroke syndromes, symptoms, and signs</td>
<td>Symptoms occur on ascent or immediately upon surfacing. Symptoms of arterial gas embolism may spontaneously resolve</td>
</tr>
</tbody>
</table>

**Generally, the symptoms of decompression sickness occur minutes to several hours after surfacing, but in rare cases, symptoms can occur days after diving.** Symptoms occurring between dives may improve during a subsequent dive (as recompression has occurred) but get worse upon resurfacing (as the inert gas load has increased and ambient pressure has decreased). Flying with the resultant decrease in ambient pressure may precipitate or worsen symptoms. For this reason, divers are generally advised to refrain from flying for at least 12 to 24 hours after the last dive depending on the nature of the diving exposure.\(^{9}\)

**Pain**

Divers with type I decompression sickness typically describe a deep pain, unrelieved but not worsened with movement. This pain can be attributed to or confused with pain caused by injury, potentially making accurate diagnosis difficult. Pain is thought to be due to distention from bubbles in ligaments or fasci,
intramedullary bubbles at the ends of long bones, or the activation of stretch receptors caused by bubbles in tendons. The mechanism of simple distention of tissues is supported by the rapid improvement of symptoms with recompression. Common pain locations are knees and shoulders, and most often, only a single joint is involved. Decompression sickness in commercial and military divers, caisson workers, and aviators tends to manifest most often as joint pain. Sport divers, who usually perform multiple dives, often over a period of days, are more prone to spinal cord effects.

Poorly localized and difficult-to-describe back or abdominal pain may herald the more serious signs of spinal cord involvement.

**Pulmonary Symptoms**

Pulmonary symptoms, generally seen usually only after more prolonged exposures, are caused by large numbers of pulmonary artery bubbles and include symptoms of cough, hemoptysis, dyspnea, and substernal chest pain. Cardiovascular collapse can occur.

**Neurologic Symptoms**

The classic description of divers with neurologic decompression sickness (type II) can begin with a sensation of truncal constriction or girdle-like pain. Often a wooly feeling begins in the feet, developing into an ascending paralysis, producing symptoms of transverse myelitis. This form is usually rapid in onset and has a tendency to affect the lower cervical and thoracic regions. However, in type II decompression sickness, neurologic deficits do not necessarily cause distinct spinal cord syndromes (i.e., an anterior or posterior spinal artery syndrome), nor will a definitive level necessarily be found, as lesions may be scattered throughout the spinal cord. Autonomic involvement, with resulting incontinence and sexual dysfunction, is not uncommon. The pathophysiology of spinal cord decompression sickness seems to be initial bubbling in the low-pressure venous plexus system that first impedes and then obstructs venous outflow from the cord. Decreasing venous blood flow prevents dissolved nitrogen in spinal cord tissues from egressing, and in situ bubbles within the spinal cord develop (called *autochthonous bubbles*).

**Vestibular Symptoms**

Vestibular decompression sickness usually occurs after deep, long dives, although it has been reported in sport divers. Signs are vertigo, hearing loss, tinnitus, and disequilibrium. The vestibular syndrome can be differentiated from inner ear barotrauma mainly by the history, because patients with inner ear barotrauma develop symptoms in the water and, generally, immediately after a forced Valsalva maneuver to equalize the middle ear pressure.

Other, nonspecific symptoms such as headache, nausea, dizziness, or unusual fatigue are also reported. It may be difficult to differentiate fatigue from decompression sickness from the expected fatigue from the exertion of diving.
Patent Foramen Ovale

The association between decompression sickness and patent foramen ovale is unclear. There appears to be an increased prevalence in patients with inner ear and cutaneous decompression sickness. It is reasonable to screen divers with recurrent, unexplained decompression sickness for a patent foramen ovale. Closure of a large defect will reduce arterialization of venous gas emboli, although it has yet to be shown if such closure will reduce the incidence of subsequent decompression sickness.\(^\text{12}\)

ARTERIAL GAS EMBOLISM

Arterial gas embolism occurs when air enters the left side of the vascular system. In the setting of diving, this most often results from pulmonary barotrauma. Arterial gas embolism can also occur as a complication of certain medical procedures, such as central vascular catheterization and cardiac bypass. Air inadvertently introduced into the venous circulation can cross from the right side of the circulation from intracardiac or pulmonary arteriovenous shunts. Air bubbles may also arterialize through these same shunts, sometimes making the source of arterial bubbles difficult to determine.\(^\text{13}\) Whatever the source, when air embolizes systemically, distribution depends mainly on blood flow and not gravity.

Clinical Features

The most dramatic effect of arterial gas embolism is on the brain, resulting in a variety of stroke syndromes, symptoms, and signs, depending on the part of the brain affected. Rarely, diving-related arterial gas embolism from pulmonary barotrauma causes immediate apnea and cardiac arrest. The mechanism of cardiovascular collapse appears to be air in the entirety of the large arteries and veins of the central vascular bed.\(^\text{14}\) The effects of arterial gas embolism secondary to pulmonary barotrauma usually occur on ascent or immediately upon surfacing. If the victim does not die immediately, the symptoms of cerebral arterial gas embolism often include loss of consciousness, seizure, blindness, disorientation, or hemiplegia. Symptoms may spontaneously improve as the gas enters the venous cerebral circulation after a spike in blood pressure. Sometimes, by the time the patient reaches the clinician, the only signs that remain are subtle defects. In particular, parietal lobe signs and symptoms are easily overlooked. A cascade of inflammatory processes also occurs in air embolism, just as in decompression sickness.\(^\text{4}\)

Laboratory Testing

The hematocrit may elevate from hemoconcentration and third spacing of fluids. The creatine phosphokinase (and other enzymes such as lactate dehydrogenase, alanine aminotransferase, and aspartate aminotransferase) will become elevated secondary to the systemic distribution of bubbles. The degree of elevation of creatine phosphokinase corresponds to the embolism severity. Cardiac troponins may also be elevated and most likely do not represent occlusive coronary artery disease.\(^\text{4}\)

Treatment of Decompression Sickness and Arterial Gas Embolism
The treatment includes administering 100% oxygen, increasing tissue perfusion with IV fluids, and rapid recompression. Some advocate placing patients with air embolism in the Trendelenburg position or in the left lateral decubitus position to "trap" air in the left ventricle. By the time the victim is brought onto the dive boat or the ambulance arrives, the air has usually been distributed, and the Trendelenburg position merely increases intracranial pressure, decreases cerebral perfusion, and interferes with other first aid measures. Nonetheless, some divers with arterial gas embolism have collapsed when placed in a sitting or standing position. As a result, a supine position—not Trendelenburg position—is recommended for patients with arterial gas embolism. Vomiting patients should be placed in the lateral decubitus position to prevent aspiration.

Recompression therapy with hyperbaric oxygen treats by several mechanisms. See the chapter 21, titled "Hyperbaric Oxygen Therapy" for detailed discussion. The administered pressure decreases the size of bubbles, and the high partial pressure of oxygen in solution increases inert gas washout from bubbles and tissue. Mass action dictates a gas will travel down pressure gradients; therefore, nitrogen will move from bubbles with a high partial pressure of nitrogen into plasma, where it will travel to the lungs and be exhaled. Conversely, oxygen from plasma with a high partial pressure of oxygen will enter bubbles, but ultimately will diffuse into cells and be metabolized, further reducing bubble size. Hyperbaric oxygen also decreases tissue edema, increases oxygen delivery to ischemic tissues, and reduces neutrophil adhesion to the endothelium and neutrophil activation.15

Recompression using the U.S. Navy Treatment Table 6 is a commonly used method of management for decompression sickness, employing a maximal treatment pressure of 2.8 ATA (60 fsw). Table 6 is also used for air embolism, although some advocate an initial pressurization to 6 ATA (165 fsw) to maximize bubble compression, then continuation at 2.8 ATA (U.S. Navy Table 6A). Different treatment tables are used in other parts of the world, and there is some experience using lower treatment pressures for decompression sickness in monoplace chambers with reportedly comparable results.16 Some patients may benefit from repeated treatments if symptoms do not fully resolve. Recompression should occur as soon as possible, and it should not be withheld in cases with delayed presentation.4 Additionally, for divers who have missed needed decompression stops because of an emergency ascent or nonadherence to appropriate diving tables, it may be appropriate for them to undergo recompression therapy even if asymptomatic. U.S. Navy Table 5 recompression would usually be adequate in such a circumstance.

The administration of IV lidocaine as a therapeutic adjunct for cerebral arterial gas embolism has been advocated, because it appears to decrease neuropsychiatric deficits when given during anesthesia for cardiac procedures requiring bypass,17,18,19 since bypass operations commonly cause the entry of air into the arterial system. Dosing of lidocaine in this setting is not standardized, although typical cardiac dosing is commonly used.20

The Divers Alert Network (telephone: 1-919-684-9111; Web site: http://www.diversalertnetwork.org) has staff available 24 hours a day to provide assistance to divers and to help clinicians treat patients with
decompression sickness or arterial gas embolism. The Divers Alert Network can provide information and the location of the nearest recompression facility around the world.

**SPECIAL CONSIDERATIONS**

**ASTHMA**

There is some debate over the safety of diving for individuals with asthma. Although the relative risk of pulmonary barotrauma may be higher in asthmatics (possibly as much as twice that of the general diving population), the absolute risk is still low because of the rarity of pulmonary barotrauma in diving (approximately 1 in 125,000 dives). A physician who specializes in diving medicine should examine divers or potential divers with asthma, and an exercise pulmonary function test should be performed. Asthmatics can be cleared for diving if, using their usual medications, they have a normal exercise pulmonary function test and if they understand the potential increased risk of pulmonary barotrauma. A diver who develops a lung injury that cannot be explained by the circumstances of the dive (i.e., the diver did not have a rapid, breath-holding ascent) should be evaluated for congenital or acquired structural lung disease and should probably no longer dive.

**IMMERSION PULMONARY EDEMA**

Pulmonary edema can occur while diving. Because the first reported cases occurred in cold water, this condition was first described as "cold water" or "cold-induced" pulmonary edema. However, many cases have subsequently been reported in warm water, up to 27°C (80.6°F). Typical symptoms of pulmonary edema (dyspnea, chest discomfort, coughing up pink frothy secretions) occur at depth and usually improve over time or with standard treatments for pulmonary edema. The cause is unknown despite human studies. This syndrome generally occurs in divers with no structural or ischemic heart disease, but diagnostic evaluation for heart conditions is indicated for those with risk factors for underlying heart disease. Immersion pulmonary edema is not caused by decompression and is not treated with recompression therapy. Interestingly, some divers will experience repeated episodes, whereas others may never experience another episode.

**NITROGEN NARCOSIS**

Inert gas narcosis occurs when air is breathed at a depth of 100 fsw or greater. Symptoms include loss of fine motor skills and high-order mental processes as well as behavior similar to that seen in alcohol intoxication. Symptoms increase as depth is increased beyond 100 fsw. Divers have codified this increase in symptoms as the "martini rule" (with many variations). A common description is that each 33 ft of depth (1 ATA) in excess of 100 fsw is the equivalent to drinking one martini. Nitrogen narcosis can cause divers to engage in dangerous or foolish activities during deep dives. At depths greater than 300 fsw, unconsciousness may occur from the anesthetic effect of nitrogen. At depths greater than 200 fsw, helium is often used in place of nitrogen in gas mixtures to prevent nitrogen narcosis.
OXYGEN TOXICITY

Oxygen toxicity usually affects the lungs or brain, depending on the partial pressure of oxygen delivered and duration of exposure. Pulmonary oxygen toxicity generally occurs at lower partial pressures of oxygen but with longer exposures, whereas cerebral oxygen toxicity occurs at high partial pressures with generally short exposures. Pulmonary oxygen toxicity can occur at partial pressures of oxygen at or below 1 ATA, for example, in patients requiring prolonged mechanical ventilation with high fractions of inspired oxygen. Pulmonary oxygen toxicity is unusual in diving.

Cerebral oxygen toxicity most often occurs with partial pressures of oxygen >1.4 ATA in the water. Some divers may breathe "nitrox" or oxygen-enriched air with fractions of oxygen of 32% to 36%. Therefore, cerebral oxygen toxicity can occur at lesser depths and actually is the factor that limits diving depth with nitrox. Additionally, there are rebreather systems (closed-circuit systems), with the diver breathing within a continuous circuit of gas that has a very high fraction of oxygen (>95%), with carbon dioxide being scrubbed out. With these systems, cerebral oxygen toxicity can occur at depths as little as 25 ft.

Signs and symptoms of cerebral oxygen toxicity include twitching, nausea, paresthesias, dizziness, and seizures. If a seizure develops in the water as the initial manifestation of cerebral oxygen toxicity, drowning may result. High partial pressures of oxygen are used clinically in hyperbaric chambers (2.4 ATA, 2.8 ATA, and sometimes 3.0 ATA), but cerebral oxygen toxicity in this setting is rare, reported in <1 per 1000 patients. This is because patients in hyperbaric chambers are dry, warm, and at rest, while divers are wet, often cold, and exerting themselves—and all of these latter factors exacerbate cerebral oxygen toxicity. Cerebral oxygen toxicity is affected by partial pressure of arterial carbon dioxide and cerebral blood flow and may be caused by an increase in nitric oxide production, although this is still an area of active investigation.

Besides nitrogen narcosis and oxygen toxicity, other gas-related conditions important in diving medicine are toxicity from carbon monoxide and the adverse effects of elevated partial pressures of carbon dioxide. Additional issues, especially with very deep dives, include heat loss from breathing helium and the high-pressure nervous syndrome, characterized by tremor and loss of fine motor function caused by the direct effects of pressure.

OTHER CONDITIONS

Injuries and medical conditions occurring during and immediately after compressed air diving are often misattributed as decompression sickness or cerebral arterial gas embolism. Be aware that any medical condition can occur under the water. Acute myocardial infarction, pulmonary embolism, stroke, seizure, encephalitis, and even appendicitis have been erroneously attributed to diving. True diving accidents or cardiac sudden death can be misattributed to drowning—a common final pathway of submersion.

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**USEFUL WEB RESOURCES**

Divers Alert Network—http://www.diversalertnetwork.org

Divers Alert Network Japan—http://www.danjapan.gr.jp

Divers Alert Network Europe—http://www.daneurope.org

Divers Alert Network Asia-Pacific—http://www.danasiapacific.org


Undersea and Hyperbaric Medical Society—http://www.uhms.org

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