Scuba Diving and Dysbarism

Richard L. Byyny and Lee W. Shockley

PERSPECTIVE

Underwater free diving to salvage wrecks and to harvest seafood, sponges, coral, and mother-of-pearl has been practiced for more than 5000 years. Historically, divers also used breathing tubes, such as hollow reeds; however, it is nearly impossible to use these at depths of more than 3 feet because of the restriction of inspiration by underwater pressure. Subsequent inventions from the 16th to the 19th centuries, including diving dresses, allowed divers to remain underwater for prolonged periods at depths of up to 12 fathoms (72 feet). The first diving dress (1715) was a reinforced, leather-covered barrel with watertight armholes and a viewing porthole.

With the advent of these technologies, the symptoms of diving-related illness began to be recognized. Colonel William Pasley, the officer in charge of a unit of the British Royal Engineers that salvaged the sunken warship HMS Royal George in 1840, observed symptoms in his divers. At approximately the same time, similar symptoms and even fatalities were observed among caisson workers. The ailment became known as caisson disease, but the construction workers on the Brooklyn Bridge (built from 1870 to 1883) attached the name “the bends,” characterizing the symptoms that often caused the victim to bend forward in pain. The first clinical description of caisson disease was by Paul Bert in 1878. He correctly attributed the disease to nitrogen gas coming out of solution in the tissues during decompression. This led to the recommendation of slow ascents for pressurized workers and the development of the first recompression chambers.

The significant breakthrough allowing diving at depth was the invention of the aqua-lung by Jacques-Yves Cousteau and Emile Gagnan in 1943. The lighter and less expensive equipment, widely known by its acronym SCUBA (self-contained underwater breathing apparatus), does not require a surface supply of air or the support personnel that are necessary for helmet diving. This innovation allowed widespread deep-sea diving, and millions of divers have become certified to date.

Most amateur divers use compressed air, open-circuit scuba equipment at depths of less than 130 feet of seawater (fsw). Systems with artificial mixtures of various gases, however, are used to extend the depths to which divers can descend. Some of these are used in sport diving, but their use is uncommon and is primarily limited to commercial applications (Table 143-1).

Other variations of supplying air for divers are closed-circuit and semiclosed-circuit diving apparatus (“rebreathers”) that use calcium hydroxide to absorb expired carbon dioxide. Oxygen is then added to the decarboxylated gas before rebreathing. The advantages of rebreathers over compressed air scuba are that they are more efficient (less gas is used for a given time), allow deeper dives and longer bottom times, and generate few if any bubbles.

The total number of diving-related injuries is unknown, but the absolute numbers of patients with decompression-related illnesses, one of the most serious dive-related injuries, continue to climb as the number of divers has increased despite the rate per 10,000 dives remaining relatively constant since 2001. This rate varies somewhat on the basis of the type of diver: 0.015% for scientific divers, 0.01 to 0.019% for recreational divers, 0.030% for U.S. Navy divers, and 0.095% for commercial divers. The rate of mortality in diving varies between 1.5 and 9 per 100,000 dives. With the popularity of diving and the relative ease of rapid travel from distant destinations, it behooves even the land-locked emergency physician to be aware of diving-related illnesses.

PRINCIPLES OF DISEASE

Scuba divers may encounter emergencies common to environmental exposures (e.g., hypothermia, sunburn, and physical trauma) or aquatic activities (e.g., submersion accidents, motion sickness, and marine envenomations), but they are also subject to the unique injuries related to dysbarisms and barotrauma. The pathophysiologic mechanism of these conditions primarily results from volume-pressure changes within the air-filled cavities of the body or from increased dissolution of gases, particularly nitrogen, in body tissues.

Atmospheric pressure varies with altitude and weather patterns, but 760 mm Hg (14.7 psi or 1 atm) is the standard used at sea level. Water is far denser than air. A mountain climber would need to ascend to 18,000 feet to reduce atmospheric pressure by 50% from sea level, but a diver needs to descend only 33 feet in seawater (34 feet in fresh water) to double the atmospheric pressure, an increase of 23 mm Hg per foot of depth.

To understand the pathophysiologic processes of dysbarisms and barotrauma, one should be familiar with several of the laws of physics that define the behavior of liquids and gases (Table 143-2; Figs. 143-1 to 143-5). The human body is composed mostly

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*Caissons are construction chambers filled with pressurized air that permit working in a dry environment. They have been used in the construction of bridge footings and tunnels in places that would otherwise have been underwater.
of water and behaves like a liquid subject to Pascal’s law, which states that a pressure applied to any part of a liquid is transmitted equally throughout. Pressure changes, however, do alter the volume within the air-filled spaces of the body, including the lungs, bowel, sinuses, and middle ear, according to Boyle’s law. This law states that at constant temperature, the absolute pressure and the volume of gas are inversely proportional \((PV = k)\). In other words, as pressure increases (with descent), the gas volume is reduced; as the pressure is reduced (with ascent), the gas volume increases.

Temperature also affects the pressure and volume of a gas as described by Charles’ law. At a constant pressure, the volume of a gas is directly proportional to the change in the absolute temperature \(\left(V_1/T_1 = V_2/T_2\right)\). Thus with heat the volume increases, and with cold the volume decreases. The general gas law \((P_1 \cdot V_1/T_1 = P_2 \cdot V_2/T_2)\) combines Boyle’s and Charles’ laws to predict the behavior of a given quantity of gas when any of these factors change.

Barotrauma results when a diver is unable to equalize the pressure within air-filled structures to the ambient pressure of the

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**Table 143-1 Mixed Gas Diving**

<table>
<thead>
<tr>
<th></th>
<th>OXYGEN</th>
<th>NITROGEN</th>
<th>HELIUM</th>
<th>OTHER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air</td>
<td>21%</td>
<td>78%</td>
<td>Trace</td>
<td>1%</td>
</tr>
<tr>
<td>Nitrox I (EAN32 or Nitrox32)*</td>
<td>32%</td>
<td>68%</td>
<td>Trace</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Nitrox II (EAN36 or Nitrox36)*</td>
<td>36%</td>
<td>64%</td>
<td>Trace</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Normoxic trimix (e.g., trimix 19/30)*</td>
<td>19%</td>
<td>51%</td>
<td>30%</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Hypoxic trimix (e.g., trimix 10/50)*</td>
<td>10%</td>
<td>40%</td>
<td>50%</td>
<td>&lt;1%</td>
</tr>
</tbody>
</table>

*Enhanced air nitrox, oxygen enriched air, nitrox, EANx, SafeAir, “devil gas,” “voodoo gas.”

†A normoxic mix, such as 19/30, is used in the depth range of 30 m (100 feet) to 60 m (200 feet).

‡A hypoxic mix, such as 10/50, is used for deeper diving, as a “bottom” gas only; it cannot safely be breathed at shallow depths where the oxygen partial pressure is less than 0.18.

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**Table 143-2 Laws of Physics**

<table>
<thead>
<tr>
<th>GAS LAW</th>
<th>FORMULA</th>
<th>SIGNIFICANCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pascal’s law: A pressure applied to any part of a liquid is transmitted equally throughout.</td>
<td>(\Delta P = \rho g (\Delta h))</td>
<td>Pressure increases in a contained space are transmitted throughout; significant for inner ear barotrauma and middle ear barotrauma (see Fig. 143-1)</td>
</tr>
<tr>
<td>Boyle’s law: At a constant temperature, the absolute pressure and the volume of gas are inversely proportional. As pressure increases, the gas volume is reduced; as the pressure is reduced, the gas volume increases.</td>
<td>(P_1 \cdot V_1 = P_2 \cdot V_2)</td>
<td>Relates to change in the volume of a gas caused by the change in pressure due to depth, which defines the relationship of pressure and volume in breathing gas supplies (see Fig. 143-2)</td>
</tr>
<tr>
<td>Charles’ law: At a constant pressure, the volume of a gas is directly proportional to the change in the absolute temperature.</td>
<td>(V_1/T_1 = V_2/T_2)</td>
<td>Increasing pressure (filling a scuba tank) causes heat; cooling a tank decreases the pressure (see Fig. 143-3)</td>
</tr>
<tr>
<td>The general gas law combines these concepts to predict the behavior of a gas when the factors change.</td>
<td>(P_1 \cdot V_1/T_1 = P_2 \cdot V_2/T_2)</td>
<td>A means of relating pressure, volume, and temperature together in one equation when variables are not constant</td>
</tr>
<tr>
<td>Dalton’s law: The total pressure exerted by a mixture of gases is equal to the sum of the pressures (partial pressures) of each of the different gases making up the mixture, with each gas acting as if it alone is present and occupies the total volume.</td>
<td>(P_{\text{total}} = P_1 + P_2 + P_3 + \ldots + P_n)</td>
<td>Nitrogen under pressure acts as if other gases are not present (see Fig. 143-4)</td>
</tr>
<tr>
<td>Henry’s law: The amount of a gas that will dissolve in a liquid at a given temperature is directly proportional to the partial pressure of that gas.</td>
<td>(e' = e^k )</td>
<td>More nitrogen is taken into solution (e.g., serum) at high pressures than comes out of solution at lower pressures (see Fig. 143-5)</td>
</tr>
</tbody>
</table>

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**Figure 143-1.** Pascal’s law. A pressure applied to any part of a liquid is transmitted equally throughout.
environment during ascent or descent. The fractional changes in volume are greater near the surface. Thus the greatest risk for barotrauma is in shallow water, where the proportional pressure changes are also the greatest.

Dalton’s law states that the total pressure exerted by a mixture of gases is equal to the sum of the pressures (partial pressures) of each of the different gases making up the mixture, with each gas acting as if it alone is present and occupies the total volume. Henry’s law states that the amount of any gas that dissolves in a liquid at a given temperature is directly proportional to the partial pressure of that gas. At higher ambient pressures, the concentration of each component of air in solution with blood and tissues increases until a new steady-state concentration is achieved.

The gases in a diver’s breathing mixture are dissolved into the body in proportion to the partial pressure of each gas in the mixture. The length of time the diver is breathing the gas at the increased pressure and the inherent solubility of the gas also govern the quantity of a particular gas that dissolves. The dissolved gas remains in solution as long as the pressure is maintained. As the diver ascends, however, increasingly more of the dissolved gas comes out of solution. A rapid ascent may reduce the pressure at a rate higher than the body can accommodate, and the bubbles (particularly nitrogen) may accumulate and disrupt body tissues and systems, a phenomenon termed decompression sickness (DCS). This is similar to the way that rapid opening of a bottle of
soda allows bubbles of carbon dioxide to come out of solution rapidly.
If the ascent rate is controlled (i.e., through the use of the safe
decompression tables or submersible dive computers), the gas is
carried to the lung vascular bed and is exhaled before it accumu-
lates to form significantly large or numerous bubbles in the tissues,
similar to how opening of a soda bottle slowly reduces the bub-
bling of the contained carbonated liquid.

**CLINICAL FEATURES**

 Disorders Related to Descent

**Middle Ear Barotrauma**

Middle ear barotrauma (MEBT), also known as barotitis or “ear
squeeze,” is the most common complaint of scuba divers. It is
experienced by 30% of novice scuba divers and 10% of experi-
denced divers. The middle ear may lead to a facial palsy in certain individuals
who the seventh cranial nerve passes unexposed through this
space. On occasion, this disorder can be life-threatening during a
dive as the subject becomes disoriented and can drown.

**External Ear Barotrauma**

External ear barotrauma is less common than MEBT and results
from the outward bulging of the tympanic membrane during
descent. Normally, the external auditory canal is filled with water
during descent. Obstruction of the external auditory canal because
of cerumen, stenosis, earplugs, or a tight-fitting wet suit hood can
trap air, causing a relative negative pressure. This may lead to
localized pain. Patients may have hemorrhages in the wall of the
external auditory canal on examination, but the symptoms are
typically self-limited.

**Inner Ear Barotrauma**

Inner ear barotrauma (IEBT) results in damage to the cochleoves-
tibular apparatus. It is much less common than MEBT but is
associated with greater morbidity. A large negative pressure gradi-
ent develops in the middle ear if the diver is unable to equalize
pressure during descent, similar to MEBT. Inward deflection of the
tympanic membrane is transmitted to the oval window of the
cochlea through the ossicles. Movement of the oval window
creates a pressure wave within the perilymph of the cochlea, which
causes an outward distention of the round window into the middle
ear. Sudden equilibration of pressure in the middle ear or a vigor-
os Valsalva maneuver may rupture the round window, lead to
hemorrhage into the inner ear, or tear the labyrinthine (Reissner’s)
membrane.

Symptoms associated with IEBT include variable hearing loss,
severe vertigo, nausea, tinnitus, and fullness in the affected ear.
Signs include severe nystagmus, positional vertigo, ataxia, and
vomiting. The degree of sensorineural hearing loss is variable.
Distinguishing IEBT from inner ear DCS (a type of DCS II) can
be challenging but should not delay recompression in a patient in
whom the diagnosis is in doubt.

**Barosinusitis**

Obstruction of the sinus ostia by mucosal thickening, polyps, pus,
or deviated septum predisposes to sinus barotrauma, the second
most common complaint among divers. The air-filled maxillary,
frontal, and ethmoidal sinuses are susceptible to volume-pressure
changes on ascent or descent; the most commonly affected is the
maxillary sinus, followed by the frontal. The most common
symptom is facial pain; epistaxis is common.

**Facial Barotrauma**

As water pressure increases during descent, a negative pressure
develops within the dive mask over the eyes and nose, which must
be equalized by forced exhalation through the nose. When this is
not adequately performed, the large negative pressure gradient
may lead to facial and conjunctival edema, diffuse petechial
hemorrhages on the face, and subconjunctival hemorrhages.
Very rarely, optic nerve damage can result from severe facial
barotrauma.

**Disorders Arising at Depth**

**Nitrogen Narcosis**

Nitrogen narcosis, known as rapture of the deep, results from the
intoxicating effects of increased tissue nitrogen concentration at
depth. Symptoms include euphoria, false feeling of well-being,
confusion, loss of judgment or skill, disorientation, inappropriate laughter, diminished motor control, and tingling and vague numbness of the lips, gums, and legs. With breathing of compressed air, symptoms typically begin to occur at approximately 100 feet and often become profound at depths of more than 150 feet. Because of these dangers, the use of compressed air is not recommended for sport diving to depths of more than 120 feet. Although the effects of nitrogen narcosis resolve with ascent to shallower depths and are variable between individuals, the diver may drown because of poor judgment or seriously impaired motor skills in the presence of a dive emergency.

**Oxygen Toxicity**

At elevated partial pressures for extended periods, oxygen can be toxic to the central nervous system (CNS) or lungs. Oxygen becomes toxic to the CNS when its partial pressure exceeds 1.6 ATA. Oxygen partial pressures below 1.4 ATA are unlikely to produce CNS toxicity. A diver breathing compressed air would attain a partial pressure of 1.6 ATA of oxygen at a depth of 218 fsw. This far exceeds the depth to which sport divers would go. Deep divers prevent oxygen toxicity by breathing mixed gases with decreased oxygen content (e.g., hypoxic trimix).

Pulmonary oxygen toxicity (low-pressure oxygen poisoning) can occur after 24 hours of exposure to partial pressures of oxygen in excess of 0.6 ATA. The symptoms of pulmonary oxygen toxicity include a burning sensation or pain on inspiration and coughing. Pulmonary function gradually becomes normal after the exposure is terminated, but pneumonitis and permanent fibrosis are possible. It is extremely unlikely that a sport diver would ever be exposed for the duration that is required to produce toxicity; however, long exposures to higher levels of oxygen, such as those administered for certain recompression protocols, may lead to pulmonary oxygen toxicity.

**Contaminated Air**

Rarely, other gases, such as carbon monoxide and carbon dioxide, can contaminate the air that is compressed into a tank. This can happen, for example, if the compressor intake is placed too close to the compressor’s engine exhaust. As in the case of oxygen and nitrogen, the partial pressure of these contaminants in the tissues increases dramatically with depth, potentiating their clinical effects. The symptoms of hypercarbia or carbon monoxide poisoning are more severe at elevated partial pressures. Hypercarbia increases a diver’s susceptibility to CNS oxygen toxicity.

Rebreathers release microscopic calcium hydroxide or “soda lime” dust particles into the apparatus. These particles are small enough and have geometric characteristics that allow them to be deposited in the alveoli. When soda lime comes into contact with water, it forms a caustic liquid. In the event of a hose rupture allowing seawater contamination of the circuit, caustic burns to the mouth, throat, and airways may result. Chronic exposure to soda lime dust may contribute to long-term effects on respiratory function.

**Disorders Arising on Ascent**

**Alternobaric Vertigo**

Alternobaric vertigo (ABV) results from an inability to equalize pressure within the middle ear during ascent. Although equalization during descent requires active maneuvers to maintain eustachian tube patency, air normally exits the middle ear without difficulty during ascent because the pressure within the middle ear exceeds ambient pressure. In the setting of mucosal edema or thickening within the eustachian tube, however, the passage of air may be impeded. The problem is typically unilateral. When the pressure gradient within the middle ear reaches 60 cm H₂O, increased labyrinthine discharge produces nystagmus. Clinically, the patient experiences a profound but transient sense of vertigo during ascent that may be associated with nausea and vomiting. Unlike those of IEBT, the symptoms are self-limited.

**Barodontalgia**

On occasion, air that is trapped beneath a poorly filled dental cavity expands on ascent, leading to dental pain. This condition is relatively benign and self-limited.

**Gastrointestinal Barotrauma**

Serious gastrointestinal barotrauma is a rare condition in scuba divers. It results from the expansion of bowel gas in the small intestine and colon on ascent. Predisposing factors include consumption of carbonated beverages, large meals, or gas-producing foods before diving as well as performance of the Valsalva maneuver in the head-down position. Symptoms include eructation, flatulence, bloating, and crampy abdominal pain. In divers with inguinal or other hernias, the potential for expansion of trapped gas within the hernia exists, and expansion may result in incarceration or strangulation. Gastric rupture is a rare complication. Although gastrointestinal barotrauma is a rare entity, it should be suspected in the diver-patient with a provocative history and abdominal pain.

**Pulmonary Barotrauma**

Without continuously expiring on ascent, a scuba diver who takes a full breath at 33 fsw will have twice the lung volume at the surface (Boyle’s law). Because the volume expansion of the alveoli is limited, the increase in pressure either forces gas bubbles across the alveolar-capillary membrane or causes the wall of the alveoli to rupture. A differential pressure of only 80 mm Hg between the alveoli and chest wall, corresponding to a change in depth of 3 to 4 feet, is all that is required to force air bubbles across the alveolar-capillary membrane.

The greatest risk for pulmonary barotrauma occurs in less than 10 feet of water. It is therefore important for the clinician to evaluate patients with symptoms that are consistent with disease entities related to pulmonary barotrauma even after exposure to shallow depths. Pulmonary barotrauma can result in the following four conditions: pneumothorax, pneumomediastinum, subcutaneous emphysema, and alveolar hemorrhage. Risk factors elicited from the dive and medical history may suggest the diagnosis of pulmonary barotrauma. In most cases, fast ascent, panic, problems in regulating buoyancy, or running out of air is noted.

Asthmatics have a twofold increased risk for pulmonary barotrauma compared with the general diver population. There are six mechanisms that contribute to the increased risk in asthmatics:

1. Bronchospasm and mucus plugging predispose local regions of lung to injury.
2. When air is compressed, it becomes denser. This may contribute to greater turbulent flow through narrow airways.
3. During scuba diving, there is a reduction in breathing capacity related to the effects of immersion. At 33 feet underwater, the maximum breathing capacity of a normal scuba diver is only 70% of the surface value. At 100 feet underwater, the reduction is approximately 50%.
4. When compressed air (from the scuba tank) expands in the regulator before delivery to the lungs, it cools (Charles’ law). Breathing of chilled air may trigger bronchospasm in
asthmatics who have a cold-induced component of their disease.
5. Scuba diving takes some effort; asthmatics who have an exercise-induced component of their disease may experience bronchospasm.
6. Compressed air may be contaminated by pollen and other allergens.

Traditionally, asthmatics were advised not to dive. Several experts, however, propose more liberal guidelines. The risk of diving is probably acceptable if the diving candidate with asthma demonstrates normal pulmonary function at rest (forced vital capacity [FVC], midexpiratory flow, forced expiratory volume in 1 second, and forced expiratory flow between 25 and 75% of FVC) and after strenuous exercise. Asthma severity can wax and wane. Because symptoms may worsen for 4 to 6 weeks after an upper respiratory infection or during certain seasons, asthmatics should refrain from diving until they are completely free of symptoms despite the pulmonary function criteria.

Pneumomediastinum and subcutaneous emphysema result when air crosses the alveolar endothelium and dissects into the pulmonary interstitium. Most commonly, the air then travels into the neck, mediastinum, or pericardium. The manifestations of pneumomediastinum may include fullness in the neck, palpable subcutaneous crepitance, and a change in voice quality or timbre. Unless evidence of either hemodynamic instability or airway compromise exists, interstitial air or subcutaneous emphysema is not a life-threatening condition.

Pneumothorax may occur when the air that escapes from alveoli as a result of pulmonary barotrauma crosses the visceral pleura. A tension pneumothorax is a rare complication. The symptoms and signs of a pneumothorax secondary to pulmonary barotrauma are typical for a pneumothorax of any cause.

Pulmonary barotrauma can also cause alveolar hemorrhage. Patients may present with hemoptyis coincident with chest pain and dyspnea. Chest radiography may reveal an interstitial infiltrate.

Decompression Sickness
The term decompression sickness (DCS) refers to a spectrum of clinical illnesses resulting from the formation of small bubbles of nitrogen gas in the blood and tissues on ascent. The clinical expression of DCS depends on the location, destination, and degree of nitrogen bubble formation in blood and tissues. Small, asymptomatic venous gas emboli are common in the ascending diver and are filtered by the lungs without apparent permanent damage. Persistent intravascular bubbles, however, elicit inflammatory cascades, cytokines, the complement system, platelet aggregation, and thrombosis. Furthermore, the bubbles can cause mechanical obstruction, ischemia, and tissue hypoxia. Nitrogen is highly fat soluble, and the heavily myelinated white matter of the CNS is at particular risk for DCS.

The incidence of DCS is estimated to be 2.8 cases per 10,000 dives. The potential for development of DCS increases with the length and depth of a dive. Other risk factors include age, obesity, fatigue, heavy exertion, dehydration, fever, cold ambient temperatures after diving, diving at high altitude, and flying after diving. Tobacco and ethanol use may also increase susceptibility to DCS. The risk of DCS is 2.6 times greater for men than for women.

This difference is possibly due to risk-taking behaviors. There appears to be no increase in DCS in women who are taking oral contraceptive agents or menstruating during diving.

A patent foramen ovale (PFO) is a risk factor for increased susceptibility to DCS. Sixty-five percent of divers who present with serious DCS have a PFO. Brain lesions occur in 27% of sport divers. This percentage is roughly the same as the prevalence of PFO or other right-to-left shunts in the general population. These multiple brain lesions may be caused by bubbles in the venous circulation that are not filtered by the vasculature of the lungs but enter the arterial circulation, even in the absence of other DCS symptoms. Most sport divers do not undergo screening for PFO with echocardiographic bubble studies; some PFOs may open only at increased ambient pressures so that bubble studies conducted at 1 ATA would be normal.

The U.S. Navy dive tables estimate the amount of nitrogen that accumulates in the body during a dive to a particular depth and duration. The tables calculate a maximal dive time, called the no-decompression limit. If the no-decompression limits are exceeded, underwater decompression stops are recommended. Many sport scuba divers use submersible dive computers to calculate maximum dive times in lieu of the tables. These computers use mathematical algorithms to model nitrogen saturation in human tissues. Although they remove human calculation errors, such computers also tend to extend no-decompression times to their maximum limits.

These tables and computers are meant to reduce the likelihood of exceeding the solubility of nitrogen at sea level to produce DCS. The diver still must ascend in a slow, controlled manner to allow the gradual release of nitrogen. Off-gassing continues after the diver has surfaced. Repetitive dives within several hours result in accumulation of tissue nitrogen and shorter no-decompression limits. The diving tables are based on several assumptions about nitrogen elimination, even strict adherence to these tables does not ensure that DCS will not occur. The risk of DCS increases as a diver approaches the no-decompression time limits and almost never occurs after dives to less than 6 meters and rarely at less than 10 meters. It is critical for the evaluating physician to realize that divers can develop DCS even when they are within any calculated no-decompression limits.

DCS typically is manifested within hours after surfacing. The U.S. Navy Diving Manual notes that 42% of symptoms occur within 1 hour after diving, 60% within 3 hours, 83% within 8 hours, and 98% within 24 hours. Flying shortly after diving or ascending to altitude, however, may cause symptoms in patients later than expected, and some patients present days after diving with DCS.

Traditionally, DCS has been divided into two categories, type I and type II. Type I DCS affects the musculoskeletal system, skin, and lymphatic vessels. Type II DCS involves any other organ system. The more inclusive “decompression-related illness” is now also used to encompass DCS I, DCS II, and arterial gas embolism (AGE). This terminology is adapted to aid clinicians in remembering that the distinction is irrelevant in the selection of treatment. All types of decompression illness require recompression. DCS I, DCS II, and AGE can coexist. The symptoms of DCS may be subtle and resolve by the time of evaluation. A distribution of the most common symptoms and the first symptoms is presented in Figure 143-7.

Type I DCS can be experienced as variable and periartricular pain in the arms and legs. The elbow and shoulder joints are most commonly affected. Local tenderness and erythema are uncommon. The placement of a blood pressure cuff inflated to 150 to 200 mm Hg on an affected joint produces relief of pain and confirms the diagnosis; however, the sensitivity of this maneuver was as low as 61% in one study. Skin marbling, known as cutis marmorata (patchy cyanotic marbling of the skin), results from venous stasis. It may begin as severe pruritus and progress into an erythematous rash and then skin mottling. Cutis marmorata does not follow a dermatomal distribution and commonly involves the trunk and torso. Lymphatic obstruction by air bubbles can also occur, causing extremity edema.

Type II DCS includes symptoms beyond those described for type I. These symptoms can involve the CNS, the inner ear, and the lungs. The CNS is particularly susceptible to decompression
illness because of its high lipid content. The spinal cord, especially the upper lumbar area, is more often involved than cerebral tissue. Symptoms of spinal DCS include limb weakness or paralysis, paresthesias, numbness, and low back and abdominal pain. Limb symptoms often begin as a distal prickly sensation that advances proximally, followed by progressive sensory or motor loss. A dermatome sensory level occurs in some spinal DCS patients, often at the T12 to L1 dermatomes. Bladder symptoms, fecal incontinence, and priapism may occur. Unlike patients with spinal cord trauma, patients experiencing DCS may have patchy or unequally distributed sensory and motor findings.

Spinal DCS can occur alone or in combination with cerebral, inner ear, or pulmonary symptoms. Cerebral symptoms include mild to moderate headache, blurred vision, diplopia, dysarthria, unusual fatigue, inappropriate behavior, and a sense of detachment. Loss of consciousness in CNS DCS is rare (in marked contrast to AGE). Magnetic resonance imaging, computed tomography (CT), and single-photon emission CT with technetium (Tc-99m)–labeled hexamethylpropyleneamine can identify the bubbles of CNS DCS. No imaging studies are sensitive enough to exclude DCS, however, and normal imaging results should not delay transfer for definitive therapy.

Inner ear DCS is commonly called the staggers. The symptoms of inner ear DCS are the same as those of IEBT and include nausea, dizziness, vertigo, and nystagmus. This can usually be distinguished from IEBT because the onset happens during ascent or after surfacing.

Pulmonary DCS is called the chokes. All divers are exposed to some degree of microbubble emboli to the lungs on ascent. The progression to symptoms depends on the number and volume of bubbles. The deposition of venous gas emboli in the pulmonary arterial circulation produces progressive dyspnea, cough, and chest pain. The cough may progress to paroxysmal fits with worsening pain.

The physical examination of patients suffering from pulmonary DCS may reveal cyanosis and hypotension in association with increased central venous pressure and pulmonary arterial pressure, right-sided strain on an electrocardiogram, and decreased end-tidal carbon dioxide level. The condition may progress to respiratory arrest. Ancillary tests for pulmonary DCS not only are insensitive but also lead to unnecessary treatment delays. Even after ascent from very shallow saturation dives, microbubbles in the venous circulation can be routinely detected by M-mode ultrasonography; however, their presence does not necessarily correlate with symptoms.

DCS may be particularly dangerous to a developing fetus of a scuba diving mother because the majority of the fetal circulation bypasses the pulmonary bed through the foramen ovale and the ductus arteriosus. This bypass prevents the fetal lungs from acting as a filter for microbubbles. In addition, venous gas emboli may appear in the fetal circulation before they are apparent in the maternal circulation. Data on the effects of diving on pregnant women suggest a higher incidence of low-birth-weight infants, prematurity, congenital malformations, stillbirths, and spontaneous abortions. There are no safe-diving tables that would protect a fetus from DCS; therefore, pregnant women should be advised to refrain from scuba diving.

Arterial Gas Embolism

AGE results when air bubbles are forced across the alveolar-capillary membrane, escape into the pulmonary venous circulation, and proceed through the left atrium and ventricle into the arterial circulation. It is one of the most common causes of death.
in divers. Clinical symptoms and signs are in part the result of mechanical obstruction by gas bubbles. AGE can also result from a right-to-left shunt of venous bubbles, such as in a diver with a PFO. AGE may occur alone or in conjunction with DCS.

Although air bubbles may embolize to any organ, the coronary and cerebral arteries are associated with the most serious consequences. Emboli to the coronary arteries may cause cardiac ischemia, myocardial infarction, dysrhythmias, or cardiac arrest. Dysrhythmias are also indirectly caused by centrally mediated autonomic dysfunction from cerebral emboli. Mechanical occlusion of the cerebral vasculature from emboli, most commonly to the anterior and middle cerebral arteries, causes a variety of symptoms and signs similar in appearance to an acute stroke.

The clinical manifestations of AGE may be sudden, dramatic, and life-threatening. Divers who have supposedly “drowned” may actually have lost consciousness during ascent as a result of cerebral gas emboli. Any diver breathing compressed air at any depth underwater and who surfaces unconscious or who loses consciousness within 10 minutes of reaching the surface should be assumed to be suffering from AGE. The most common presentation of AGE includes a global alteration of consciousness, headache, dizziness, convulsions, and visual changes. Other common presenting symptoms and signs include cranial nerve symptoms, unilateral weakness, unilateral or bilateral sensory loss, ataxia, and speech changes. Pulmonary symptoms, including dyspnea, pleuritic chest pain, and hemoptysis, occur in 25 to 50% of cases.

Pulmonary Edema

Pulmonary edema while scuba diving was first reported in 1981. An increase in Afterload (from vascular hyper-reactivity, possibly triggered by cold) combined with an increase in preload (from the hyperbaric underwater environment) may be the cause.

DIAGNOSTIC STRATEGIES

Focused questions concerning the dive profile, including the depth and length of the dive and a careful assessment of when the symptoms first occurred, may provide important diagnostic clues (Box 143-1). A physician unfamiliar with the specifics of scuba diving may rely on the assessment of members of the dive group to determine whether maximum dive limits were approached. In making the diagnosis of a dive injury, it is helpful to think of the injuries in terms of occurring during descent, while at depth, or during ascent (Fig. 143-8). Because recompression therapy is time sensitive, it is important to concentrate on treatment decisions rather than on securing an absolute diagnosis. The primary question to address is whether this dive injury is likely to be helped by recompression. Early consultation with a hyperbaric specialist is recommended.

DIFFERENTIAL CONSIDERATIONS

Most diving injuries have limited differential diagnoses that include medical disorders and trauma unrelated to dysbarism. The differential diagnosis of IEBT includes inner ear DCS, ABV, and isolated MEBT with a rupture of the tympanic membrane. It is relatively easy to distinguish IEBT from MEBT and ABV because the vestibular symptoms associated with the last two entities are transient and self-limited. When IEBT occurs simultaneously with MEBT, the presence of both may be documented by an audiogram, which demonstrates both a conductive and a sensorineural hearing loss (Table 143-3).

The differentiation of IEBT from inner ear neurologic DCS is crucial because the treatments differ. An IEBT is more likely when symptoms begin during descent or the diver relates a history of difficulty equilibrating or performing a vigorous Valsalva maneuver. If the dive profile is examined and the no-decompression limits are approached and symptoms began soon after surfacing, inner ear DCS is more likely. A history of difficulty in equalizing the ears on descent, or the onset of symptoms early in the dive, suggests barotrauma. A history of a dive that approaches decompression limits, or the onset of symptoms during or soon after ascent, and the presence of other neurologic findings suggest DCS. A trial of recompression therapy is prudent if concerns for DCS exist.

![Figure 143-8. The approach to the injured diver. ABV, alternobaric vertigo; AGE, arterial gas embolism; DCS, decompression sickness; GI, gastrointestinal; POPS, pulmonary overpressure syndrome.](image-url)
The differential diagnosis of pulmonary DCS includes AGE (Table 143-4). Although the treatment of both requires recompression therapy, they may be differentiated by the timing of the onset of symptoms. Almost all cases of AGE present within the first 10 minutes of surfacing, whereas DCS presents more typically after 10 minutes: 42% of pulmonary DCS symptoms begin within 1 hour of surfacing, 60% within 3 hours, 83% within 8 hours, and 98% within 24 hours.3

| Table 143-4 Middle Ear Barotrauma, Inner Ear Barotrauma, and Alternobaric Vertigo |
|---------------------------------|---------------------------------|---------------------------------|
| **MEBT** | **IEBT** | **ABV** |
| Symptoms | Ear pain during descent | Ear pain during descent | Ear pain during descent |
| | Hearing loss | Hearing loss | Transient |
| | | | hearing loss |
| Possible transient vertigo | Severe vertigo and nausea | Nausea |

| Signs | Conductive hearing loss | Nystagmus | Nystagmus |
| | TM injury | Emesis | Emesis |
| | Unilateral face paralysis | Ataxia | TM injury |
| | | Romberg’s sign | Neural hearing loss |

Abnormal tympanic membrane.

<table>
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<th>Table 143-5 Decompression Sickness versus Arterial Gas Embolism</th>
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<td><strong>DECOMPRESSION SICKNESS</strong></td>
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<td>Treatment</td>
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The intravenous fluid treatment recommendations differ in AGE. Divers with AGE are less likely to be dehydrated than are divers with DCS. This may be because of a shorter period of immersion or because they experience less bubble-induced endothelial damage.7 Further, CNS injuries in AGE may be worsened

**Box 143-2 Diving Disorders That Require Recompression Therapy**

- Decompression sickness type I
- Decompression sickness type II
- Arterial gas embolism
- Contaminated air (carbon monoxide poisoning)

**Diving Disorders Requiring Recompression Therapy**

Diving disorders that require recompression therapy are listed in Box 143-2.

Treatment with 100% oxygen replaces inert gases in the lungs with oxygen. By establishment of a large gradient from the tissues to the alveoli, removal of the inert gases is enhanced and bubble size is reduced.52 In addition, oxygen administration treats the tissue hypoxia caused by gas bubbles. Normobaric oxygen treatment during preparation for recompression therapy is associated with a need for fewer recompressions.51

Dehydration can increase the seriousness of DCS.52 Hydration decreases bubble formation after decompression.53 It also decreases the endothelial inflammation and damage associated with hemoconcentration and hemodynamic instability seen in dehydrated DCS patients.54,55 Therefore, intravenous fluids should be administered to divers suspected of having nonpulmonary (“choke”) DCS. Overly aggressive hydration in divers with pulmonary DCS may worsen pulmonary edema. Similarly, fluid overload should be avoided in patients with cerebral or spinal cord edema. There is no clear consensus that the best fluid is crystalloid (normal saline or lactated Ringer’s solution) or colloid; many experts, however, advise against dextrose in water without electrolytes (e.g., D,W). Fluids should be given to ensure a urinary output of 0.5 mL/kg/hr.

Divers with AGE are less likely to be dehydrated than are divers with DCS. This may be because of a shorter period of immersion or because they experience less bubble-induced endothelial damage.7 Further, CNS injuries in AGE may be worsened

**MANAGEMENT**

Patients with stable vital signs and suspected dive injuries should receive 100% oxygen until the clinician can exclude pulmonary barotrauma or decompression illness. Patients with unstable vital signs or in cardiac arrest should be treated according to advanced cardiac life support (ACLS) guidelines. The clinician should take a thorough history, including elements in Box 143-1, and then perform a thorough physical examination. Diving-related illnesses are diagnosed and treated on the basis of the history and physical examination, and several invaluable resources are available for advice. The Divers Alert Network (DAN), located at Duke University in Durham, North Carolina, is a membership association that provides courses on diving-related emergencies and publishes data on diving accidents and fatalities. Clinicians may obtain treatment advice by calling DAN. DAN provides a 24-hour medical emergency hotline at 1-919-684-9111 (collect calls are accepted) and a nonemergency advisory line Monday through Friday, 8:30 AM to 5 PM Eastern time, at 1-919-684-2948 or 1-800-446-2671. In addition, DAN international contacts are in Europe, Brazil, Japan, Asia Pacific, and southern Africa. DAN maintains a website with links to key information at www.diversalertnetwork.org. DAN uses a telephone intake form, the DAN On-Site Neurological Assessment for Diver’s History (Fig. 143-9); familiarity with this form may facilitate the emergency physician’s communications with DAN.

Hyperbaric therapy for AGE should be initiated as soon as possible, although there are cases of significant improvement even in the face of long delays until recompression.\textsuperscript{58} Patients with AGE who are recompressed within 5 minutes of surfacing have a mortality rate of 5%, and there is an extremely low risk of morbidity among the survivors.\textsuperscript{32} If recompression is delayed by 5 hours or more, the mortality rate increases to 10%, with 50% morbidity.\textsuperscript{32} Although spontaneous resolution of symptoms may occur in patients with AGE, all patients should be recompressed. The rationale is that although microbubbles may clear from the cerebral circulation, secondary capillary edema and swelling may result in a delayed recurrence of symptoms. Furthermore, more subtle

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**Figure 143-9.** The DAN On-Site Neurological Assessment for Diver’s History.
symptoms may be appreciated only after the resolution of more prominent ones. Finally, minor symptoms may progress, and the relapse rate is high in untreated cases.

Similarly, the prognosis for DCS when it is treated with recompression is generally good but depends on the severity of symptoms at onset and the delay to recompression. A delay to definitive recompression treatment is associated with a worse outcome in cases of severe DCS. Patients can obtain some benefit from recompression, however, even if treatment is initiated more than 24 hours after the dive. Recompression therapy for DCS may be initiated as late as 10 to 14 days after exposure if necessary.

Recompression for DCS and AGE is most often performed in a multiplace chamber with one or more in-chamber attendants. DCS and AGE have also been successfully treated in monoplace chambers. Monoplace chambers are compact, lightweight, and more widely available than multiplace chambers. Unfortunately, because of their design, most cannot be pressurized beyond 3 ATA (100 fsw) or deliver air-oxygen mixtures. The most common recompression schedule is U.S. Navy treatment (or an equivalent procedure); the diver is compressed to 2.8 bar (60 fsw pressure) while breathing 100% oxygen. The time to complete treatment is 4 hours 45 minutes, not including descent time.

Ground transport to a hyperbaric facility is preferred to air transportation, if feasible, because an increase in altitude lowers the ambient pressure and allows microbubbles to expand. If air transportation must be used, it is important to maintain cabin pressure at less than 1000 feet. Commercial aircraft are typically pressurized to a cabin altitude of 5000 to 8000 feet in cruise flight (30,000 feet). Many of these aircraft are capable of near-sea level cabin pressures if flying no higher than 15,000 to 20,000 feet. Because helicopters are not pressurized, it is recommended that they maintain an altitude of no more than 500 feet above the departure facility.

In addition to recompression therapy, several adjunctive treatments are proposed in the treatment of DCS and AGE. The treatment or prevention of hypothermia may increase tissue perfusion and off-gassing. The nonsteroidal anti-inflammatory drug tenoxicam (Mobiflex) decreases the number of recompressions required for symptom resolution in DCS but does not change the ultimate outcome. Aspirin therapy (as a platelet aggregation inhibitor) has previously been recommended for DCS and AGE. Because of the lack of studies demonstrating safety and efficacy for DCS or AGE treatment, however, it cannot be recommended. Steroids have previously been advocated clinically for neurologic DCS and AGE. No significant improvements in neurologic sequelae are now demonstrated; however, the elevated blood glucose levels associated with steroid treatment may worsen the outcome of CNS injury.

Anticoagulants should not be used routinely in the treatment of DCS. The one exception is in divers with lower extremity paralysis caused by neurologic DCS or AGE. Low-molecular-weight heparin (e.g., enoxaparin, 30 mg, subcutaneously every 12 hours; dose adjustment should be made for patients with renal impairment) after recompression therapy may be used in patients with inability to walk to prevent venous embolic disease. Intermittent pneumatic compression devices are alternatives.

Lidocaine is not effective in DCS but may have neuroprotective effects in patients with AGE and serious neurologic symptoms. It is administered as an intravenous bolus of 1 mg/kg, followed by a drip infusion of 2 to 4 mg/min.

Cardiac dysrhythmias may be refractory to standard treatments until the diver is recompressed. Defibrillation in a hyperbaric environment may be a fire hazard and is not recommended. Seizures may be managed with benzodiazepines; however, mannitol should be avoided. Spinal DCS patients often develop urinary retention requiring bladder catheterization. Of note, endotracheal tube and urinary catheter balloons should be inflated with sterile saline (not air) before recompression therapy is initiated. Nitrous oxide should be avoided because it can increase the size of tissue bubbles by inward diffusion.

Although the head-down position (Trendelenburg) has previously been advocated to prevent migration of intra-arterial bubbles to the brain, it is not effective and may result in worsening cerebral edema and intracranial pressure. Transport of the patient with AGE in a flat supine position is recommended to maximize arterial-venous flow.

There is limited experience with carbon monoxide poisoning from contaminated air supplies in a diving environment. This condition should be treated immediately with normobaric 100% oxygen and may require hyperbaric oxygen therapy. Consultation with a hyperbaric specialist or toxicologist is recommended.

### Diving Disorders Not Requiring Recompression Therapy

Diving disorders that do not require recompression therapy are listed in Box 143-3.

Prevention of MEBT requires that the diver equalize the pressure in both middle ears. Any diver who cannot clear both ears on the surface should not dive. The diver should never perform a forceful Valsalva maneuver during descent or ascent to clear the ears because of the risk of ABV, round or oval window rupture (descent), or pulmonary barotrauma (ascent). The prophylactic use of pseudoephedrine (60 mg taken 30 minutes before diving) or oxymetazoline nasal spray may reduce the incidence and severity of MEBT. The use of these medications to facilitate diving with symptoms of an upper respiratory infection, however, is not recommended. Antihistamines should be avoided before diving. Sinusitis and upper respiratory infections increase the likelihood of suffering barotitis. Diving should be avoided for 2 weeks after the resolution of an upper respiratory infection.

Treatment of uncomplicated serous otitis from MEBT includes topical nasal vasoconstrictors, such as phenylephrine and oxymetazoline hydrochloride, and repeated Frenzel maneuvers to displace the fluid through the eustachian tube. The Frenzel maneuver is performed by pinching the nose, placing the tongue on the roof of the mouth, as far forward as possible, and gently moving the back of the tongue upward, as when starting to swallow; this is repeated as many times as necessary until equalization occurs. If the physical examination reveals a ruptured tympanic membrane, prophylactic treatment should also include an oral antibiotic. Oral steroids (prednisone) may speed recovery when a seventh nerve palsy is diagnosed in conjunction with a perforated tympanic membrane, although this disorder is typically self-limited. Diving must also be suspended until the tympanic membrane heals to prevent calorically induced vertigo.

### Box 143-3 Diving Disorders That Do Not Require Recompression Therapy

- Middle ear barotrauma
- External ear barotrauma
- Inner ear barotrauma
- Barocongestive syndrome
- Baroaurora
- Baroembolism
- Baroaneurysm
- Baroreflux
- Baroencephalitis
- Gastrointestinal barotrauma
- Avascular osteonecrosis
Treatment of external ear barotrauma includes cleaning of the external canal and removal of foreign bodies. Earplugs should never be worn when diving.

A conservative treatment approach to IEBT includes bed rest for 5 to 7 days with the head elevated, avoidance of straining and the Valsalva maneuver, and decongestants to facilitate drainage of the middle ear. Early surgical intervention may benefit patients with total or near-total hearing loss but not isolated high-frequency hearing loss. All patients with IEBT should be referred to an otolaryngologist because IEBT suggests significant injury to the cochleovestibular system.

Treatment of baro sinusitis is typically conservative, including the use of decongestants and, occasionally, antibiotics. If symptoms persist, referral for antrostomy should be considered, particularly to prevent future recurrences. The patient should be advised not to dive until any underlying respiratory infection or acute inflammatory process has resolved.

The victim of facial barotrauma may have a dramatic appearance, but the condition is usually benign and requires no specific treatment. The patient should be advised not to resume diving until facial edema resolves.66

Nitrogen narcosis symptoms should resolve on surfacing when the partial pressure of nitrogen decreases. Seemingly persistent symptoms should prompt a search for other causes, such as DCS, cerebral AGE, contaminated air, and near-drowning.

With the exception of AGE, none of the pulmonary barotrauma disorders (pneumothorax, pneumomediastinum, subcutaneous emphysema, and alveolar hemorrhage) requires recompression therapy. Treatment with 100% oxygen may aid in the resolution of the disorders. Although tube thoracostomy may not be required for the treatment of a small pneumothorax, a tube should be placed if the diver is to undergo recompression therapy for concomitant AGE or DCS to prevent a tension pneumothorax. Catheter aspiration of the pneumothorax is an acceptable alternative to tube thoracostomy if the patient will not receive positive-pressure ventilation or recompression therapy.

The evaluation and management of pneumomediastinum include serial chest radiographs to ensure that no coexisting pneumothorax develops. One hundred percent oxygen therapy may hasten the resolution of symptoms. In the rare case of respiratory compromise, tracheal intubation may be required. Most important, the presence of interstitial pulmonary emphysema should alert the physician to the possible coexistence of more severe forms of pulmonary barotrauma. The need for a surgical decompressive treatment of subcutaneous emphysema alone is extremely unlikely. Supportive therapy to correct hypoxia is indicated in the treatment of alveolar hemorrhage.19

Careful equalization during a slow ascent can prevent the occurrence of ABV. Oral and intranasal decongestants may be indicated if symptoms persist. Myringotomy is occasionally required.87

DISPOSITION

Decompression tables are based on the premise that the diver returns to an ambient pressure of 1 atm on surfacing. However, with ascent in altitude after diving or on diving at altitude, a further reduction in ambient pressure occurs. Commercial airliners may be pressurized to a cabin altitude of 5000 to 8000 feet in cruise flight. Many cases of DCS have a delay in the onset of symptoms in divers who fly after diving even if they are symptom free before departure. Divers who experience DCS symptoms before departure but nevertheless fly are more likely to have type II DCS, less likely to achieve complete relief after recompression, and more likely to have residual symptoms for at least 3 months.

The recommended postdive preflight surface interval (PFSI) before ascending to higher altitudes (or flying) depends on the diver’s repetitive group designator (residual nitrogen time). The relative risk for development of DCS increases with greater residual nitrogen times and shorter PFSIs (Fig. 143-10).68 For example, the risk for development of DCS is seven times greater for PFSIs of 12 hours after a dive to 130 fsw than for PFSIs of 24 hours after a dive to 60 fsw.34 Long PFSIs (up to 48 hours) may be necessary to reduce the risk of DCS after deep, multiday repetitive diving, particularly if the dives required decompression stops (exceeding the no-decompression limits).68 Some dive computers calculate time-to-flight intervals, which tend to be somewhat shorter than most guidelines based on residual nitrogen timetables. Flying should be delayed for at least 12 hours after diving if less than 2 hours of total dive time was accumulated in the preceding 48 hours. For multiple-day, unlimited diving, flying should be delayed for at least 24 hours. It is prudent to admit all recompressed patients or to advise them to remain within 60 minutes of the hyperbaric facility for 24 hours. Patients recompressed after DCS or AGE should not fly for 72 hours.

The U.S. Navy’s guidelines recommend that the patient not return to diving for 7 days after recompression for type I DCS and...
for 4 weeks after type II DCS. The sport diver who experiences DCS type II symptoms or AGE should probably never dive again. After treatment for pulmonary barotrauma, evaluate the diver with a spiral volumetric chest CT scan to determine whether there are any preexisting pathologic conditions (e.g., bullae) that could put the diver at risk for recurrence before further diving. Chest CT is not recommended in routine medical screening before participation in scuba diving without a history of pulmonary barotrauma. Echocardiography to assess for a PFO is often recommended for divers with severe or recurrent neurologic DCS. A substantial amount of venous gas must embolize through the PFO for significant symptoms to occur; that is unlikely to happen in conservative depth-time profiles. A diver with a diagnosed PFO should weigh the risks of continuing to dive and the risks of surgical repair.

**KEY CONCEPTS**
- Treatment with 100% oxygen is the initial therapy for all diving emergencies until the diagnoses can be determined. It has been demonstrated to reduce the morbidity and mortality related to decompression illness and can be helpful in patients with pneumothorax.
- Intravenous hydration is important for DCS but may not be as important for AGE.
- The Trendelenburg position, once thought to reduce arterial gas cerebral embolization, is no longer recommended.

The majority of dive injuries are diagnosed on the basis of the focused dive history and physical examination alone and are best differentiated into disorders of descent, disorders of depth, and disorders of ascent. The U.S. Navy Diving Manual and the Divers Alert Network (DAN) are valuable resources for the clinician presented with a diving emergency. DAN provides a 24-hour medical emergency hotline at 1-919-684-9111 (collect calls are accepted) and a nonemergency advisory line Monday through Friday, 8:30 AM to 5 PM Eastern time, at 1-919-684-2948 or 1-800-446-2671. Imaging and laboratory studies are not useful for ruling in decompression and should not delay definitive recompression therapy. With the exception of DCS, AGE, and possibly carbon monoxide poisoning from contaminated air, most dive-related disorders can be treated without recompression therapy. Recompression treatment is recommended for patients with DCS and AGE. Before initiation of recompression therapy, ensure that endotracheal tube and urinary catheter balloons are not inflated with air. After multiple-day unlimited diving, flying should be delayed for at least 24 hours. Unlike patients with spinal cord trauma, patients experiencing DCS may have patchy or unequally distributed sensory and motor findings. If inflation of a blood pressure cuff to 150 to 200 mm Hg relieves joint pain, type I DCS is suggested.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.


