Diving Medicine

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Abstract

Exposure to the undersea environment has unique effects on normal physiology and can result in unique disorders that require an understanding of the effects of pressure and inert gas supersaturation on organ function and knowledge of the appropriate therapies, which can include recompression in a hyperbaric chamber. The effects of Boyle's law result in changes in volume of gas-containing spaces when exposed to the increased pressure underwater. These effects can cause middle ear and sinus injury and lung barotrauma due to lung overexpansion during ascent from depth. Disorders related to diving have unique presentations, and an understanding of the high-pressure environment is needed to properly diagnose and manage these disorders. Breathing compressed air underwater results in increased dissolved inert gas in tissues and organs. On ascent after a diving exposure, the dissolved gas can achieve a supersaturated state and can form gas bubbles in blood and tissues, with resulting tissue and organ damage. Decompression sickness can involve the musculoskeletal system, skin, inner ear, brain, and spinal cord, with characteristic signs and symptoms. Usual therapy is recompression in a hyperbaric chamber following well-established protocols. Many recreational diving candidates seek medical clearance for diving, and healthcare providers must be knowledgeable of the environmental exposure and its effects on physiologic function to properly assess individuals for fitness to dive. This review provides a basis for understanding the diving environment and its accompanying disorders and provides a basis for assessment of fitness for diving.

Keywords: diving medicine; barotrauma; decompression sickness; fitness for diving

Recreational scuba diving is a popular sport throughout the world. In the United States alone, there are an estimated 1.2 million sport divers (1). Recreational diving was originally centered in coastal areas, but with the increasing popularity of the sport, divers are now found increasingly in inland lakes, pools, and quarries. Therefore, divers can present with diving-related disorders in any area of the country. The popularity of diving in tropical waters and the ease of travel to and from these tropical sites can result in local physicians encountering unfamiliar disorders acquired in distant locations. Incorrect diagnosis and treatment, particularly of neurologic injury related to diving, can result in permanent injury to the brain and spinal cord. Additionally, individuals from 12 to 80 years old are seeking medical clearance to dive from their primary care physicians, who should be aware of recommendations for fitness to dive.

The Environment

Common to underwater exposure and exposure in hyperbaric chambers, caissons, and underwater habitats is an increase in ambient pressure. Pressure underwater is directly proportional to depth (Table 1), and pressure effects dominate the disorders sustained by divers. Intrathoracic blood volume increases with water immersion (2). Hong and colleagues estimated the blood volume shift related to head-out water immersion to be about 700 ml (2), with a resulting increase in cardiac output (3) and central venous pressure. Diuresis results from an increase of natriuretic hormones and suppression of antidiuretic hormone.

Shallow water blackout, the development of unconsciousness during ascent from long breath-hold dives (4), is caused by reduction in arterial P_{O_2} due to metabolic consumption of oxygen and decreasing ambient pressure during ascent. Excess hyperventilation pre-dive increases the risk of shallow water blackout. Comprehensive reviews of the physiology of breath-hold diving have been published (5).

Divers are exposed to water temperatures that result in progressive heat loss during the dive. Without protective

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This review follows the outline and expresses the concepts regarding diving medicine and physiology that can be found in more detail in Reference 75.

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Table 1. Pressure Equivalents for Altitude and Depth

Depth in Sea Water (*ft*)	ATA	psi
0*	1	14.7
33	2	29.4
66	3	44.1
99	4	58.8

Definition of abbreviations: ATA = atmospheres absolute; psi = pounds per square inch.
*Sea level.

garments, hypothermia occurs when water temperature is below thermoneutral temperature (93–95°F). Even tropical water temperatures (76–82°F) require some form of thermal protective garment for safe diving. Thermal protection is effective in preventing hypothermia and is tailored to the expected water temperature. Cold stress evokes a rise in V_{O_2} to generate metabolic heat and minimize core temperature change. Vasoconstriction occurs as a reflex response to facial immersion (5) and lower body core temperature. Energy demands for underwater swimming also require that divers maintain a reasonable level of physical fitness (6).

The Equipment

The self-contained underwater breathing apparatus (scuba) is the most popular form of breathing support for sport divers. A metal cylinder containing compressed air (2,500–3,000 pounds per square inch [psi]) connected to a pressure regulator provides ambient pressure breathing air to the diver when inhalation is initiated and provides flow that matches the ventilatory rate of the diver. Expired air is exhaled into the surrounding water. Because of the limited air supply, scuba use is usually limited to 200 feet of seawater (fsw) depth. In addition, the high partial pressures of oxygen and nitrogen have important physiologic effects at deeper depths. A rebreather (closed circuit scuba) conserves inert gas by recycling the exhaled gas through a carbon dioxide absorbent and replenishes only the consumed oxygen. Although commercial and military divers have traditionally used closed-circuit scuba, it is increasing in popularity in recreational diving.

Commercial divers use a closed helmet attached at the collar to a diving suit. Compressed air or other gas mixtures flow from the surface to the helmet and from the helmet into the suit to maintain an air layer for thermal protection. Further thermal protection can be provided by pumping hot water through a specially designed diving suit.

Diving Nomenclature

Decompression sickness (DCS) is a disorder resulting from supersaturation of inert gases in blood and tissues that results in free gas formation. Golding and coworkers (7) classified DCS into a minor form (Type I), affecting the musculoskeletal system, and a major form (Type II), involving the neurologic system. Arterial gas embolism (AGE) is a separate disorder that can result from diving and from other mechanisms that result in injection of air into the circulation. Francis and Smith (8) developed the term "decompression illness" for these two disorders because they require similar therapy and can be difficult to separate clinically in divers.

Pressure Effects: Boyle's Law

Boyle's law states that volume and pressure are inversely related in a fixed mass of an ideal gas at constant temperature. Therefore, gas volume is reduced to one half of the original volume when the absolute pressure is doubled. Based on Boyle's law, a relative volume change from 2 to 3 atmospheres absolute (ATA) (33–66 fsw) is less than the change from 1 to 2 ATA (surface to 33 fsw); thus, for a given change in depth the gas volume change is greater when closer to the surface.

Barotrauma

Barotrauma can occur on descent or ascent. With increased pressure during descent, gas volume in air-containing body cavities, such as the lungs, middle ear, paranasal sinuses, and gastrointestinal tract, is diminished. If the pressure in these spaces does not equalize with the ambient pressure, tissue injury results from the forces generated by the pressure difference between the ambient pressure and the body cavity. Compression of the lung to volumes below the residual volume during deep breath-hold dives can result in pulmonary hemorrhage. The most common diving-related disorder encountered in divers is barotrauma of the middle ear (9). Barotrauma can also involve the paranasal sinuses, the diving mask, and air pockets associated with tooth fillings. Facial nerve injury has been reported as a complication of middle ear barotrauma (10). The most serious form of barotrauma affects the lungs.

Pulmonary Barotrauma

Behnke (11) and Polak and Adams (12) first reported lung barotrauma in an ascending diver due to inadequate exhalation and overexpansion of the lungs. Ascent to the surface from depths as shallow as 4 feet can cause pulmonary barotrauma after breathing compressed air. Elevated intrapulmonary pressure that results in a transpulmonary gradient of 95 to 110 cm H_2O can rupture alveoli (13). Gas in the lung interstitial space can enter the mediastinum (mediastinal emphysema) and the pleural space, producing pneumothorax (13). Interstitial gas dissecting along the esophagus can also result in pneumoperitoneum. Subcutaneous emphysema in the neck results when gas dissects upward from the mediastinum into cervical tissues. With high alveolar pressure, gas can be forced into the pulmonary capillaries and enter the arterial circulation as gaseous emboli. Pulmonary barotrauma can occur in the absence of an evident overpressure event.

Tetzlaff and colleagues (14) noted a reduced midexpiratory flow at 25% of vital capacity in divers who presented with lung barotrauma and suggested that increased risk for pulmonary barotrauma could be identified with pulmonary function testing. These investigators identified 13 lung abnormalities among 15 patients with pulmonary barotrauma who underwent CT examination of the chest and suggested that unexplained lung barotrauma may stem from occult lung disease.

In a study of 31 patients with pulmonary barotrauma from diving, Harker and coworkers (15) reported 25% with pneumomediastinum: 10% with subcutaneous emphysema, 6% with pneumopericardium, 3% with pneumoperitoneum, and 3% with pneumothorax. Pulmonary infiltrates caused by aspiration were present in over half of the subjects.

Substernal pain exacerbated by coughing or swallowing is a common

symptom of mediastinal emphysema. Auscultation that reveals a crunching sound (Hamman's sign) that is synchronous with the heartbeat is also indicative of mediastinal air. The diagnosis can be confirmed by chest radiograph, which also helps exclude pneumothorax and aspiration. Elimination of the mediastinal air can be hastened by breathing 100% oxygen, but the free gas will eventually resolve spontaneously without oxygen therapy. Subcutaneous emphysema causes swelling and crepitus in soft tissues of the neck and supraclavicular fossa. Throat pain, hoarseness, and dysphagia may also result. The most serious complication of pulmonary barotrauma results from cerebral embolization, which can result in loss of consciousness and usually occurs within minutes after surfacing. Less severe symptoms include hemiplegia, stupor and confusion, visual disturbances, seizures, vertigo, and headache. Apnea, unconsciousness, and cardiac arrest may occur in about 5% of victims due to filling of the cardiac chambers and great vessels with air. Therapy requires emergent recompression in a hyperbaric chamber.

Middle Ear Barotrauma

Middle ear barotrauma occurs when the middle ear does not equilibrate with ambient pressure, especially when anatomic variation and local edema from allergies or upper respiratory infection result in Eustachian tube dysfunction. As middle ear pressure becomes more negative relative to the ambient pressure, the tympanic membrane is displaced inward, causing pain in the affected ear. As the pressure gradient increases with increasing depth, the tympanic membrane can rupture. When cold water enters the middle ear, the resulting unilateral vestibular dysfunction causes acute vertigo. Otitis media and chronic tympanic membrane perforation can also result (9). Most divers are trained to perform a Valsalva maneuver during descent to equilibrate middle ear pressure.

Inner Ear Barotrauma

Inner ear barotrauma occurs on descent when the diver does not equilibrate middle ear pressure in spite of repeated forceful Valsalva maneuvers. Because the Valsalva maneuver increases spinal fluid pressure and inner ear pressure, the gradient between the inner ear perilymph and the middle ear can become large enough to rupture the round or oval window, causing perilymph to leak from the inner ear (9). The diver experiences the sudden onset of vertigo, tinnitus, and loss of hearing on the affected side. Vestibular involvement can result in nausea and vomiting. Treatment may require surgical repair of the round or oval window. If untreated, tinnitus and reduced hearing may become chronic.

Sinus Barotrauma

Failure to equalize the air in the paranasal sinuses during descent causes pressure within the sinus to become negative relative to ambient pressure. Vascular engorgement and rupture causes sinus pain during descent and epistaxis during ascent when sinus pressure exceeds ambient pressure. Persistence of blood in the sinus may result in bacterial sinusitis. During ascent, compression of the maxillary branch of the trigeminal nerve that courses through the maxillary sinus can result in infraorbital paresthesias that resolve in several hours without treatment (16).

Less Common Forms of Barotrauma

Failure to equalize air in the diving mask during descent results in facial barotrauma. Edema and ecchymoses in the distribution of the diving mask and conjunctival hemorrhages are noted. Butler (17) reported a retro-orbital hematoma resulting from mask barotrauma, and Latham and colleagues (18) described diplopia complicating this injury. No therapy is required. Air in the stomach due to air swallowing during diving distends the stomach or intestine on ascent as ambient pressure drops. The stomach may rupture, resulting in pneumoperitoneum (19). The diver experiences abdominal pain during ascent that increases in severity as depth becomes shallower. Surgical repair of the ruptured viscous is usually needed. Prior gastric surgery may increase the risk of gastric air trapping (20).

Dissolved Inert Gas Effects

Henry's Law states that the partial pressure of a gas determines the dissolved gas content of tissues. Thus, as ambient pressure increases, dissolved gas concentration in blood and tissues increases. Solubility of the gas (Table 2) also determines dissolved gas content. During ascent, inert gas in the tissues becomes supersaturated and forms a gas phase. Because of the metabolic activity of oxygen and carbon dioxide, oxygen and carbon dioxide saturation rarely contributes to gas phase formation. Recent studies suggest that blood microparticles may act as a nidus for bubble formation in blood (21).

Inert Gas Kinetics

Because gas transfer into tissues is a dynamic process, time is required to achieve equilibrium between tissues and ambient inert gas partial pressure. Tissue gas concentration approaches the equilibrium concentration for a given pressure after time has elapsed. Washout of inert gas from tissues follows similar kinetics when ambient pressure is reduced. Mathematical models incorporating several body compartments with different gas exchange characteristics are used to characterize whole body inert gas kinetics (22). Free gas formation occurs on exposure to altitude (23). At altitudes above 18,000 feet (0.5 ATA), gas supersaturation results in free gas formation. Divers going to altitude (e.g., flying in a commercial aircraft) within 12 to 18 hours after diving may develop free gas in tissues even though they follow established protocols for safe decompression.

Usual diving exposures that are relatively short in duration and not deeper

Table 2. Characteristics of Inert Gases*

Gas	Molecular Weight	Lipid Solubility	Water Solubility	Narcotic Potential†
Helium	4	0.015	0.009	0.23
Hydrogen	2	0.036	0.018	0.55
Nitrogen	28	0.067	0.013	1.00

*Solubility of gases in lipid and water expressed as gas volume/solute volume at 1 atmosphere absolute.
†Values relative to nitrogen.

than 200 fsw allow only a few tissues to reach equilibrium. Divers follow schedules for ascent based on depth and time to minimize free gas formation (22).

When diving exposures exceed 12 hours, many tissues reach equilibrium with the higher ambient pressure. Bond (24) demonstrated that divers could experience extended exposures (weeks) to increased pressure (saturation diving) without serious physiologic effects. Using Bond's techniques, divers have been exposed to depths over 1,000 feet for periods of 3 to 4 weeks. Decompression from these prolonged exposures requires multiple days to avoid free gas formation.

Inert Gas Supersaturation in Tissues

As ambient pressure falls during ascent from a dive breathing pressurized gas, the diver's tissues become supersaturated. Excessive supersaturation causes gas to leave solution and form free gas. Boycott and colleagues (25) demonstrated that staged decompression involving timed stops during ascent (Figure 1) could minimize the degree of supersaturation. Decompression schedules based on the Haldane concept of a critical pressure ratio are used to minimize the risk of decompression sickness. Boycott and colleagues (25) proposed that a set of tissue compartments with different rates of gas uptake and elimination could be used to model gas kinetics. These tissue compartments provide a convenient means to understand the kinetics of inert gas exchange, but they do not represent discrete anatomic structures. Schedules for safe decompression have been developed for air and a number of gas mixtures (nitrogen–oxygen, helium–oxygen, nitrogen–helium–oxygen, hydrogen–oxygen, and rare earth gases such as argon and neon).

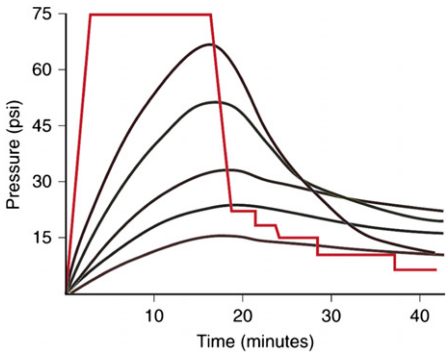

Figure 1. Nitrogen uptake and washout curves in five tissue compartments after exposure to increased ambient pressure (75 pounds per square inch above atmospheric pressure, equivalent to 168 ft of seawater depth). The *red line* shows the pressure exposure with several stops during pressure reduction to avoid excess supersaturation. Tissue gas uptake of five different tissue compartments is shown by the *curved lines*. Uptake and washout rates are different for each tissue. Redrawn with permission from Reference 25.

DCS

Excess supersaturation on ascent with expansion of gases in blood and tissues results in damage to tissues and organs. In his classic work, *La Pression Barometrique*, published in 1878, Bert (26) described the pathophysiology of DCS. Autopsy studies on divers and caisson workers in the early 20th century suggested that DCS was caused by free gas in blood and tissues. Blood clotting and platelet activation, intravascular coagulation, and capillary breakdown with plasma leakage into the extravascular space were found by Hallenbeck and colleagues to be related to bubble surface activity (27). Venous gas emboli are usually the first evidence of free gas and can be detected by ultrasound interrogation of the pulmonary artery (28). Severe cases of DCS can result in plasma loss, hemoconcentration, hypovolemia, and pulmonary edema. When studied by ultrasound, divers are often noted to have venous gas emboli without clinical evidence of DCS. High volumes of venous gas emboli in asymptomatic divers and aviators are often precursors of clinical DCS (29, 30).

Patent Foramen Ovale

Venous bubbles transiting the atrial septum through a patent foramen ovale (PFO) can cause arterial gas embolism (31). The presence of a PFO significantly increases the risk of DCS, and this risk parallels PFO size (32). Compared with control subjects, divers with evidence of altered cerebral function due to DCS have a high prevalence of large PFOs (33). Similarly, Billinger and colleagues found a high incidence of MRI lesions in divers with PFOs; these authors also showed that PFO closure in continuing divers decreases the risk of symptomatic DCS and asymptomatic ischemic neurological events (34). Honek and colleagues (35) demonstrated a high incidence of venous gas emboli in divers exposed to stressful experimental diving exposures. Divers with large PFOs demonstrated an increased incidence of clinical DCS, and in divers who underwent PFO closure there was a significant reduction in arterial bubbles and a trend toward reduction of clinical DCS. Their data suggest that in divers with extreme diving exposures who have a large PFOs, the risk of DCS is likely to be increased. However, the 25 to 30% prevalence of PFOs and the very low incidence of DCS indicate that a PFO plays a minor role in DCS associated with usual sport diving exposures and that closing a PFO in a sport diver as a prophylactic measure is not indicated (36) but might be considered in commercial divers who experience repeated DCS and have a large PFO.

Age and Other Risk Factors

A United States Air Force study on altitude exposure demonstrated a 3-fold increase in susceptibility to altitude DCS in aviators 42 years of age and older compared with 18- to 21-year-old aviators (37). In divers, Carturan and colleagues (38) found that increased age and decreased physical condition were related to venous bubble formation. Dehydration, exercise during diving, hypothermia, and hyperthermia are additional risk factors.

Clinical manifestations. DCS occurs in about 1 in 5,000 dives for the sport diver (39, 40), which is comparable to the risk in military divers. Commercial divers are at the highest risk for DCS. Pulmonary vascular obstruction occurs when large amounts of free gas transit the venous system; this results in chest pain, dyspnea, and cough (41). Injury to the spinal cord, usually at levels below T-11 and T-12 (42), causes paresthesia, weakness, or paralysis of the lower extremities; urinary retention; bowel or bladder incontinence; and sexual impotence (43). Acute neurologic hearing loss and vestibular dysfunction are uncommon but important manifestations of type II decompression sickness. These manifestations of DCS usually follow deep, prolonged diving exposures and often result in permanent deafness (44, 45). Musculoskeletal DCS, which is the most common type of DCS, causes joint pain that is often confused with pain from an injury (46, 47). Osteonecrosis may occur in divers who have experienced deep, prolonged exposures in caisson work (47), in diving instructors (48), and

in commercial diving operations. A history of musculoskeletal DCS also increases risk for osteonecrosis. Cutaneous DCS is associated with an erythematous or purpuric skin rash (cutis marmorata) that usually resolves within 24 hours.

Therapy of DCS and AGE. Arterial gas embolism and DCS produce similar clinical manifestations. Therapy of DCS and arterial gas embolism requires recompression in a hyperbaric chamber with administration of oxygen (49, 50). Fluid replacement should be instituted. Antiplatelet agents have been recommended, but clinical trials demonstrating their efficacy are lacking (50, 51). Bennett and colleagues (52) found that the use of a nonsteroidal anti-inflammatory medication was associated with fewer recompression treatments for DCS, but clinical outcomes were not improved. Once stabilized, the patient is decompressed slowly to surface pressure. Recompression therapy for DCS and air embolism minimizes risk for permanent injury. The severity of injury and time to effective therapy affect outcome, but treatment even several days after injury has demonstrated efficacy. For most cases of DCS, hyperbaric therapy at 60 fsw (2.8 ATA) lasting about 6 hours is used. Recompression therapy for cutaneous DCS has been recommended, but most cases resolve spontaneously. Treatment gases are oxygen and air for divers using air as the breathing gas. Enriched nitrogen-oxygen (nitrox) or helium-oxygen (heliox) may be used for treatment depths greater than 60 fsw. Hyperbaric oxygen treatments can be repeated several times, although the end points for repetitive therapy are not well defined. Although clinical outcome is thought to be improved in severe cases by multiple therapies (42), controlled clinical trials to evaluate the efficacy of repetitive treatments have not been done. Arterial gas embolism therapy is usually initiated by recompression to 60 fsw and oxygen therapy (53). When symptoms persist after recompression to 60 fsw, recompression to 165 fsw (6 ATA) is recommended (54). Administration of 100% oxygen at 1 ATA before recompression treatment is recommended for DCS and arterial gas embolism (55). Initial treatment should include oral or intravenous fluid administration. Urine specific gravity and hematocrit are useful guides for fluid therapy.

Diagnostic testing. Evaluation of the clinical status of an injured diver starts with a detailed history of the diving exposure and physical examination. Water aspiration, pneumothorax, and pneumomediastinum associated with pulmonary barotrauma can be confirmed by chest radiography or by computed tomography (CT). Imaging modalities also confirm the diagnosis of pneumoperitoneum. Imaging studies should not prolong time to recompression therapy. The diving exposure usually allows the seasoned clinician to distinguish inner ear barotrauma from inner ear DCS. Inner ear DCS requires recompression; inner ear barotrauma is initially managed medically with later surgical repair if indicated. Although audiography or electronystagmography are not specific for either disorder, these studies may aid in a decision for surgery to repair a ruptured round window caused by inner ear barotrauma. Magnetic resonance imaging (MRI) can sometimes confirm the presence of diving-related brain injury, but MRI images should be correlated with neurologic findings in light of their low specificity (56, 57). MRI images of the brain in divers without a history of DCS or AGE also show abnormalities (58), the significance of which is unclear.

Emergency treatment. Emergency treatment during transit to a recompression chamber should include 100% oxygen and fluid replacement with a crystalloid solution administered orally in a conscious diver or intravenously if aspiration is a concern. Antiplatelet therapy with aspirin can be administered to counter the platelet activation caused by free gas in the blood. The efficacy of emergency therapy in improving clinical outcome is unproven (55). Recompression therapy should not be delayed when emergency treatment measures are associated with a significant improvement in clinical status.

Inert Gas Narcosis

Breathing air at pressures exceeding 4 ATA (100 fsw) produces nitrogen narcosis (59). High partial pressure of nitrogen results in neurologic dysfunction that can present with impaired cognitive skills, reduced motor coordination, altered emotional states, and unconsciousness. Nitrogen at partial pressures exceeding 10 to 12 ATA (300–400 fsw) produces an anesthetic effect resulting in unconsciousness. Other inert gases also produce narcosis. Lipid solubility (Table 2) determines their relative narcotic potential.

Oxygen Toxicity

Oxygen at partial pressures exceeding 1.4 ATA can produce acute neurotoxicity (60). Tolerance to higher oxygen partial pressures depends on exposure time. Auditory and visual hallucinations are frequently reported. The most serious effect, a grand mal seizure, can lead to drowning. The risk of oxygen neurotoxicity is increased when divers use breathing mixtures with partial pressures of oxygen higher than that in air (i.e., 32 or 40% oxygen in nitrogen) or with rebreather systems. Lung injury from oxygen toxicity is uncommon in divers.

Fitness for Diving

Requirements for fitness depend on operational needs, with tactical military divers requiring the highest levels of fitness among various communities of divers. Standards have been created for commercial divers, tunnel and caisson workers, and support staff for clinical hyperbaric chambers. Disorders that lower exercise capacity, such as compromised cardiopulmonary function, poor physical condition, and physical disabilities, can increase the risk for diving-related injury.

Exercise Requirements

Recreational diving is not usually associated with high workloads, but with the possibility of unpredicted adverse diving conditions, a steady-state oxygen consumption of about 20 ml/kg/min allows the diver to manage adverse diving exposures. Divers who cannot sustain this level of aerobic activity should plan diving exposures that are not likely to create this demand.

Disorders That Increase the Risk of Sudden Unconsciousness

An underwater seizure can result in drowning and can risk the safety of other divers. A diving candidate should be free of seizures for 4 years before being approved for diving (61). Candidates with insulin-dependent diabetes mellitus are excluded from commercial and military diving due to the risk of hypoglycemia. Sport divers who

receive special training in managing insulin when diving can dive safely (62).

Pulmonary Disorders

Asthma has generated significant concern in the diving community because of the risk for pulmonary barotrauma during ascent related to air trapping. Clinical observations have not demonstrated an increased incidence of pulmonary barotrauma in divers with asthma (63), although isolated cases have been reported (64). Diving is often associated with increased exercise demands due to the need for swimming either underwater or on the surface breathing through a mouthpiece. Diving is not recommended in patients with poorly controlled asthma, including those with frequent need for rescue bronchodilators, with cold or exercise-induced symptoms, or with significantly reduced exercise capacity due to airway obstruction. Clearly, asthma exacerbation is a contraindication to diving. The use of controller medications to maintain unobstructed airways is not a contraindication to diving. Criteria for safe diving by patients with a history of asthma include evidence that pulmonary function measures (FEV_1, FVC) are not reduced during exercise. In this case, risk for lung barotrauma is low (65). Divers with stable asthma who have normal exercise capacity should not be restricted from diving. A spontaneous pneumothorax history increases the risk for pneumothorax during diving. A diagnosis of COPD with reduced exercise capacity and significant airflow obstruction precludes safe diving.

Cardiac Disorders

Under conditions of increased exercise demand while diving, patients with obstructive coronary disease can develop angina pectoris, myocardial infarction, or sudden death. Divers in the 60- to 70-year age range demonstrate an increased incidence of diving-related death from cardiovascular disease (66). Mebane and colleagues (67) reported 33 cases of diving-related sudden death from the Divers Alert Network. Coronary disease was found in 31 cases, a stroke occurred in one case, and aortic stenosis was present in one case. Coronary disease screening is important in recreational divers who are likely to have an increased risk for coronary disease due to age and the presence of coronary risk factors (e.g., hypertension, hyperlipidemia, and smoking). After coronary bypass surgery or coronary stenting, divers can return to sport diving if they have adequate exercise tolerance and have no evidence of ischemia or arrhythmias during exercise. Commercial and military standards preclude return to diving after coronary revascularization, although waivers are possible in some cases. Central fluid shifts due to water immersion can result in acute symptoms in subjects with reduced left ventricular function, but acute pulmonary edema (which is also referred to as immersion pulmonary edema) can occur in fit divers with normal cardiac and pulmonary function (68). The etiology is unclear, but central fluid shifts, left ventricular diastolic dysfunction, hypertension, and negative intrapulmonary pressure may contribute. Echocardiography to rule out cardiomyopathy and valvular disorders and exercise testing to rule out ischemia may be useful. Symptoms resolve with diuretic therapy. Patients with cyanotic congenital heart disease are at risk for arterialization of venous bubbles; they commonly have reduced exercise tolerance and should not dive (69).

Diving-induced arrhythmias. Humans and other mammals have a diving reflex that is stimulated by facial immersion (70, 71). In marine mammals, autonomic activation by apneic diving is an oxygen-conserving reflex, but oxygen conservation in humans has not been demonstrated (72). Ackerman and colleagues (73) first reported drowning associated with an inherited long QT syndrome. Although their cases were related to swimming, a similar risk would occur in diving.

Implanted devices. Sport divers occasionally seek clearance for diving after receiving a pacemaker or an implantable cardioverter defibrillator (ICD). Most devices are pressure tested, and their pressure tolerance is published with the device specifications. ICDs may fire when a diver is underwater, causing panic and risk for drowning. Divers with reduced left ventricular function may be at risk for acute pulmonary edema due to water immersion effects. Recent data on athletes with ICDs due to arrhythmia risk (74) suggest that such individuals with normal heart function can safely participate in sport diving.

Conclusions

Diving is an ancient skill that was first limited to commercial and military interests, but over the last 60 years diving has become an increasingly popular sport. Because of the unique environment and the well-defined disorders related to this environment, physicians are likely to encounter an occasional patient with a diving-related disorder. Some knowledge of this area of medicine is essential for recognizing these disorders. In addition, evaluations for diving fitness have become an issue for physicians. Knowledge of the underwater environment and the diving disorders provides a basis for assessment of fitness for diving. A detailed review of diving medicine can be found in Reference 75. ■

Author disclosures are available with the text of this article at www.atsjournals.org.

References

1. How many recreational scuba divers in the world? [accessed 2014 Mar 14]. Available from: http://wiki.answers.com/Q/How_many_recreational_scuba_divers_in_the_world
2. Hong SK, Cerretelli P, Cruz JC, Rahn H. Mechanics of respiration during submersion in water. *J Appl Physiol* 1969;27:535–538.
3. Arborelius M Jr, Ballidin UI, Lilja B, Lundgren CE. Hemodynamic changes in man during immersion with the head above water. *Aerosp Med* 1972;43:592–598.
4. Craig AB Jr. Causes of loss of consciousness during underwater swimming. *J Appl Physiol* 1961;16:583–586.
5. Ferrigno M. Breath hold diving. In: Bove AA, editor. Bove and Davis' diving medicine. Philadelphia, PA: Elsevier; 2004. pp. 77–94.
6. Pendergast DR, Tedesco M, Nawrocki DM, Fisher NM. Energetics of underwater swimming with SCUBA. *Med Sci Sports Exerc* 1996;28: 573–580.
7. Golding FC, Griffiths P, Hempleman HV, Paton WD, Walder DN. Decompression sickness during construction of the Dartford Tunnel. *Br J Ind Med* 1960;17:167–180.
8. Francis TJR, Smith DH. Describing decompression illness (42nd UHMS Workshop). Kensington, MD: Undersea and Hyperbaric Medical Society; 1991.

9. Hunter SE, Farmer JC. Ear and sinus problems in diving. In: Bove AA, editor. Bove and Davis' diving medicine, 4th ed. Philadelphia, PA: WB Saunders; 2004. pp. 431–460.
10. Molvaer OI, Eidsvik S. Facial baroparesis: a review. *Undersea Biomed Res* 1987;14:277–295.
11. Behnke AR. Analysis of accidents occurring in training with the submarine "lung." *U S Nav Med Bull* 1932;30:177–184.
12. Polak B, Adams H. Traumatic air embolism in submarine escape training. *U S Nav Med Bull* 1932;30:165–177.
13. Schaffer KE, McNulty WP Jr, Carey C, Liebow AA. Mechanisms in development of interstitial emphysema and air embolism on decompression from depth. *J Appl Physiol* 1958;13:15–29.
14. Tetzlaff K, Reuter M, Leplow B, Heller M, Bettinghausen E. Risk factors for pulmonary barotrauma in divers. *Chest* 1997;112: 654–659.
15. Harker CP, Neuman TS, Olson LK, Jacoby I, Santos A. The roentgenographic findings associated with air embolism in sport scuba divers. *J Emerg Med* 1993;11:443–449.
16. Butler FK, Bove AA. Infraorbital hypesthesia after maxillary sinus barotrauma. *Undersea Hyperb Med* 1999;26:257–259.
17. Butler FK, Gurney N. Orbital hemorrhage following face-mask barotrauma. *Undersea Hyperb Med* 2001;28:31–34.
18. Latham E, van Hoesen K, Grover I. Diplopia due to mask barotrauma. *J Emerg Med* 2011;41:486–488.
19. Cramer FS, Heimbach RD. Stomach rupture as a result of gastrointestinal barotrauma in a SCUBA diver. *J Trauma* 1982;22: 238–240.
20. Hayden JD, Davies JB, Martin IG. Diaphragmatic rupture resulting from gastrointestinal barotrauma in a scuba diver. *Br J Sports Med* 1998; 32:75–76.
21. Thom SR, Milovanova TN, Bogush M, Bhopale VM, Yang M, Bushmann K, Pollock NW, Ljubkovic M, Denoble P, Dujic Z. Microparticle production, neutrophil activation, and intravascular bubbles following open-water SCUBA diving. *J Appl Physiol (1985)* 2012;112: 1268–1278.
22. Vann RD. Mechanisms and risk of decompression sickness. In: Bove AA, editor. Bove and Davis' diving medicine, 4th ed. Philadelphia, PA: WB Saunders; 2004. pp. 127–164.
23. Ryles MT, Pilmanis AA. The initial signs and symptoms of altitude decompression sickness. *Aviat Space Environ Med* 1996;67: 983–989.
24. Bond GF. New developments in high pressure living. *Arch Environ Health* 1964;9:310–314.
25. Boycott AE, Damant GCC, Haldane JS. The prevention of compressed air illness. *J Hyg (Lond)* 1908;8:342–443.
26. Bert P. Barometric pressure: researches in experimental physiology. Columbus Book Co.; 1943. Reprinted by the Undersea Medical Society, Bethesda, MD, 1978.
27. Hallenbeck JM, Bove AA, Moquin RB, Elliott DH. Accerlerated coagulation of whole blood and cell-free plasma by bubbling in vitro. *Aerosp Med* 1973;44:712–714.
28. Eftedal OS, Lydersen S, Brubakk AO. The relationship between venous gas bubbles and adverse effects of decompression after air dives. *Undersea Hyperb Med* 2007;34:99–105.
29. Conkin J, Powell MR, Foster PP, Waligora JM. Information about venous gas emboli improves prediction of hypobaric decompression sickness. *Aviat Space Environ Med* 1998;69:8–16.
30. Kumar VK, Billica RD, Waligora JM. Utility of Doppler-detectable microbubbles in the diagnosis and treatment of decompression sickness. *Aviat Space Environ Med* 1997;68:151–158.
31. Wilmshurst PT, Treacher DF, Crowther A, Smith SE. Effects of a patent foramen ovale on arterial saturation during exercise and on cardiovascular responses to deep breathing, Valsalva manoeuvre, and passive tilt: relation to history of decompression illness in divers. *Br Heart J* 1994;71:229–231.
32. Torti SR, Billinger M, Schwerzmann M, Vogel R, Zbinden R, Windecker S, Seiler C. Risk of decompression illness among 230 divers in relation to the presence and size of patent foramen ovale. *Eur Heart J* 2004;25:1014–1020.
33. Germonpré P, Dendale P, Unger P, Balestra C. Patent foramen ovale and decompression sickness in sports divers. *J Appl Physiol (1985)* 1998;84:1622–1626.
34. Billinger M, Zbinden R, Mordasini R, Windecker S, Schwerzmann M, Meier B, Seiler C. Patent foramen ovale closure in recreational divers: effect on decompression illness and ischaemic brain lesions during long-term follow-up. *Heart* 2011;97:1932–1937.
35. Honek J, Sramek M, Sefc L, Januska J, Fiedler J, Horvath M, Tomek A, Novotny S, Honek T, Veselka J. Effect of catheter-based patent foramen ovale closure on the occurrence of arterial bubbles in scuba divers. *JACC Cardiovasc Interv* 2014;7:403–408.
36. Moon RE, Bove AA. Transcatheter occlusion of patent foramen ovale: a prevention for decompression illness? *Undersea Hyperb Med* 2004;31:271–274.
37. Sulaiman ZM, Pilmanis AA, O'Connor RB. Relationship between age and susceptibility to altitude decompression sickness. *Aviat Space Environ Med* 1997;68:695–698.
38. Carturan D, Boussuges A, Vanuxem P, Bar-Hen A, Burnet H, Gardette B. Ascent rate, age, maximal oxygen uptake, adiposity, and circulating venous bubbles after diving. *J Appl Physiol (1985)* 2002; 93:1349–1356.
39. Hart AJ, White SAS, Conboy PJ, Bodiwala G, Quinton D. Open water scuba diving accidents at Leicester: five years' experience. *J Accid Emerg Med* 1999;16:198–200.
40. Bove AA. Risk of decompression sickness with patent foramen ovale. *Undersea Hyperb Med* 1998;25:175–178.
41. Neuman TS, Spragg RG, Wagner PD, Moser KM. Cardiopulmonary consequences of decompression stress. *Respir Physiol* 1980;41: 143–153.
42. Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. *Lancet* 2011;377:153–164.
43. Hawes J, Massey EW. Neurologic injuries from scuba diving. *Neurol Clin* 2008;26:297–308; xii.
44. Farmer JC, Thomas WG, Youngblood DG, Bennett PB. Inner ear decompression sickness. *Laryngoscope* 1976;86:1315–1327.
45. Gempp E, Louge P. Inner ear decompression sickness in scuba divers: a review of 115 cases. *Eur Arch Otorhinolaryngol* 2013;270: 1831–1837.
46. Gempp E, Blatteau JE, Simon O, Stephant E. Musculoskeletal decompression sickness and risk of dysbaric osteonecrosis in recreational divers. *Diving Hyperb Med* 2009;39:200–204.
47. McCallum RI, Walder DN; Report of Decompression Sickness Panel Medical Research Council. Bone lesions in compressed air workers, with special reference to men who worked on the Clyde Tunnels 1958 to 1963. *J Bone Joint Surg Br* 1966;48: 207–235.
48. Cimsit M, Ilgezdi S, Cimsit C, Uzun G. Dysbaric osteonecrosis in experienced dive masters and instructors. *Aviat Space Environ Med* 2007;78:1150–1154.
49. Moon RE. Treatment of decompression illness. In: Bove AA, editor Bove and Davis' diving medicine, 4th ed. Philadelphia, PA: WB Saunders; 2004. pp. 195–224.
50. Bennett MH, Lehm JP, Mitchell SJ, Wasiak J. Recompression and adjunctive therapy for decompression illness. *Cochrane Database Syst Rev* 2012;5:CD005277.
51. Bennett MH, Lehm JP, Mitchell SJ, Wasiak J. Recompression and adjunctive therapy for decompression illness: a systematic review of randomized controlled trials. *Anesth Analg* 2010;111:757–762.
52. Bennett M, Mitchell S, Dominguez A. Adjunctive treatment of decompression illness with a non-steroidal anti-inflammatory drug (tenoxicam) reduces compression requirement. *Undersea Hyperb Med* 2003;30:195–205.
53. Bove AA, Clark JM, Simon AJ, Lambertsen CJ. Successful therapy of cerebral air embolism with hyperbaric oxygen at 2.8 ATA. *Undersea Biomed Res* 1982;9:75–80.
54. US Navy Department. US Navy diving manual. Rev. 6, Vol. 5. Diagnosis and treatment of decompression sickness and arterial gas embolism. Publication no. NAVSEA 0910-LP-106-0957. Washington, DC: US Navy Department; 2008.
55. Longphre JM, Denoble PJ, Moon RE, Vann RD, Freiberger JJ. First aid normobaric oxygen for the treatment of recreational diving injuries. *Undersea Hyperb Med* 2007;34:43–49.
56. Hennedige T, Chow W, Ng YY, Chung-Tsing GC, Lim TC, Kei PL. MRI in spinal cord decompression sickness. *J Med Imaging Radiat Oncol* 2012;56:282–288.

57. Gempp E, Blatteau JE, Stephant E, Pontier JM, Constantin P, Pény C. MRI findings and clinical outcome in 45 divers with spinal cord decompression sickness. *Aviat Space Environ Med* 2008;79: 1112–1116.
58. Reuter M, Tetzlaff K, Hutzelmann A, Fritsch G, Steffens JC, Bettinghausen E, Heller M. MR imaging of the central nervous system in diving-related decompression illness. *Acta Radiol* 1997; 38:940–944.
59. Bennett PB. Inert gas narcosis and HPNS. In: Bove AA, editor. Bove and Davis' diving medicine, 4th ed. Philadelphia, PA: WB Saunders; 2004. pp. 225–240.
60. Clark JM, Thom SR. Toxicity of oxygen, carbon dioxide, and carbon monoxide. In: Bove AA, editor. Bove and Davis' diving medicine, 4th ed. Philadelphia: WB Saunders; 2004. pp. 241–260.
61. Massey EW, Greer HD. Neurologic consequences of diving. In: Bove AA, editor. Bove and Davis' diving medicine, 4th ed. Philadelphia, PA: WB Saunders; 2004. pp. 461–474.
62. Scott DH, Marks AD. Diabetes and diving. In: Bove AA, editor. Bove and Davis' diving medicine. Philadelphia, PA: WB Saunders; 2004. pp. 507–518.
63. Neuman TS, Bove AA, O'Connor RD, Kelsen SG. Asthma and diving. *Ann Allergy* 1994;73:344–350.
64. Weiss LD, Van Meter KW. Cerebral air embolism in asthmatic scuba divers in a swimming pool. *Chest* 1995;107:1653–1654.
65. Elliott DH. Are asthmatics fit to dive. Kensington, MD: Undersea and Hyperbaric Medical Society; 1996.
66. Denoble PJ, Caruso JL, Dear GdeL, Pieper CF, Vann RD. Common causes of open-circuit recreational diving fatalities. *Undersea Hyperb Med* 2008;35:393–406.
67. Mebane GY, Low N, Dovenbarger J. A review of autopsies on recreational scuba divers: 1989–1992. *Undersea Hyperb Med* 1993;20:70.
68. Hampson NB, Dunford RG. Pulmonary edema of scuba divers. *Undersea Hyperb Med* 1997;24:29–33.
69. Bove AA. The cardiovascular system and diving risk. *Undersea Hyperb Med* 2011;38:261–269.
70. Irving L. Bradycardia in human divers. *J Appl Physiol* 1963;18:489–491.
71. Irving L, Solandt OM, Solandt DY, Fischer KC. The respiratory metabolism of the seal and its adjustment to diving. *J Cell Comp Physiol* 1935;7:137–151.
72. Lindholm P, Lundgren CE. The physiology and pathophysiology of human breath-hold diving. *J Appl Physiol (1985)* 2009;106:284–292.
73. Ackerman MJ, Tester DJ, Porter CJ. Swimming, a gene-specific arrhythmogenic trigger for inherited long QT syndrome. *Mayo Clin Proc* 1999;74:1088–1094.
74. Lampert R, Olshansky B, Heidbuchel H, Lawless C, Saarel E, Ackerman M, Calkins H, Estes NA, Link MS, Maron BJ, *et al*. Safety of sports for athletes with implantable cardioverter-defibrillators: results of a prospective, multinational registry. *Circulation* 2013;127:2021–2030.
75. Bove AA, Neuman TS. Diving medicine. In: Mason RJ, Broaddus VC, Martin TR, King TE, Schraufnagel DE, Murray JF, Nadel JA, editors. Murray & Nadel's textbook of respiratory medicine, 5th ed. Philadelphia, PA: Elsevier; 2010. pp. 1674–1690.