Medical Problems Associated with Underwater Diving

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MEDICAL PROBLEMS ASSOCIATED WITH UNDERWATER DIVING

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THE underwater environment, with its rapidly changing ambient pressures, presents pathophysiologic challenges that may lead to a variety of unusual diseases for which rapid diagnosis and treatment are critical. In the United States there are more than 5 million people certified as recreational scuba divers — that is, divers who use a self-contained underwater breathing apparatus. Given the popularity of scuba diving the world over and the number of diving accidents,[1] every physician should be aware of the specific hazards and medical conditions encountered underwater.

The barometric pressure at sea level is 100 kPa (the equivalent of 1 atmosphere absolute, 760 mm Hg, or 14.7 psi). Because pressure increases linearly underwater, every additional 10 m (33 ft) of descent adds 100 kPa (1 atmosphere). To make breathing possible for scuba divers, the changing ambient pressure is balanced by a supply of air delivered by a demand regulator from a compressed-air diving apparatus.

Since most morbidity involving diving is related to the behavior of gases under changing conditions of pressure, a brief mention of the two most relevant gas laws is appropriate. Boyle's law states that at a constant temperature, the volume of gas varies inversely with the pressure applied. The physiologic ramifications of this law underlie the characteristics of pressure-related diving diseases (barotrauma). Henry's law states that the amount of a given gas that is dissolved in a liquid at a given temperature is directly proportional to the partial pressure of that gas. Henry's law provides the basic explanation of decompression sickness and nitrogen narcosis.

BAROTRAUMA

Barotrauma refers to tissue injury resulting from the failure of a gas-filled body space (e.g., the lungs, the middle ear, and the sinuses) to equalize its internal pressure to correspond to changes in ambient pressure. During a diving descent, failure to equalize pressure leads to a decrease in the volume of these body spaces in accordance with Boyle's law. Since cavities located within a bone cannot collapse, the space they take up is filled by engorgement of the mucous membrane, often followed by hemorrhage. The risk of barotrauma is more pronounced near the surface of the water, where a small change in depth (of even a few meters) may lead to a large change in relative gas volume.[2]

Pulmonary barotrauma occurring during ascent is the most severe and life-threatening form of barotrauma. In this disorder, mediastinal and subcutaneous emphysema may produce changes in the voice, a feeling of fullness in the chest, dyspnea, dysphagia, supraclavicular crepitus, and typical radiographic findings on chest and neck films. Pneumothorax may cause sudden pleuritic pain and dyspnea. A rapidly developing large pneumothorax may impair cardiac function and in rare cases lead to shock and death.[2] All the manifestations of pulmonary barotrauma may occur singly or in combination. During ascent, as the ambient pressure decreases, gas within the lungs expands. If a diver does not permit expanding gas to escape from the lungs by exhalation, distention and rupture of the lungs may occur.[2] Air escaping from the intraalveolar spaces may cause pneumothorax, pneumomediastinum, or subcutaneous emphysema of the neck and upper chest.[2,3] Air may also enter torn pulmonary veins, resulting in arterial gas emboli.[4] The main precipitating factor is the trapping of air due to various obstructive pulmonary lesions. The most severe cases of pulmonary barotrauma are seen after an emergency ascent.[4,5] However, the condition may occur in healthy divers who have no detectable underlying pathologic conditions and who have carried out appropriate techniques of exhalation.

The most serious sequela of pulmonary barotrauma is arterial air embolism as a result of the passage of gas into the pulmonary veins and from there into the systemic circulation.[4] Gas bubbles lodged in small arteries may occlude segments of the cerebral, coronary, and other systemic vascular beds. The clinical picture is usually that of a rapidly developing stroke-like syndrome, ranging from a focal neurologic deficit beginning hours after the dive to unconsciousness, collapse, and death immediately after the diver surfaces.[2,4,5] In some patients, myocardial ischemia or arrhythmias due to coronary-artery occlusion dominate the clinical picture.[6] There may also be skin marbling and gas bubbles in the retinal vessels.[2] The occurrence of unconsciousness or any other neurologic manifestation in a scuba diver surfacing from a deep dive should also suggest the possibility of another diving complication, namely decompression sickness. This complication can occur either alone or in combination with air embolism.

The maintenance of respiration and adequate arterial oxygen tension is essential in all patients with pul-

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monary barotrauma. In cases of mediastinal or subcutaneous emphysema, the administration of high concentrations of oxygen increases the rate of removal of nitrogen, enhancing the clearance of nitrogen from the emphysematous area. A large pneumothorax that interferes with pulmonary function requires thoracocentesis. A chest drain is indicated in patients with pneumothorax and air embolism who are undergoing recompression therapy, since pneumothorax is likely to expand again during ascent.

Patients with air embolism should be transferred to the nearest hyperbaric-oxygen facility for emergency therapy while breathing the highest available concentration of oxygen.[2,4] Some authorities maintain that the patient should be transported in the Trendelenburg position in order to decrease the risk of additional cerebral embolism. Others argue that the head-down position may augment cerebral edema, and they suggest that the supine position is the best compromise.[7] Recompression treatment is carried out in hyperbaric chambers, which are used to administer oxygen at high ambient pressures. Treatment with hyperbaric oxygen both results in a mechanical reduction of the volume of gas emboli (Boyle's law) and increases the nitrogen pressure gradient between emboli and blood, accelerating the absorption of the emboli. In addition, hyperbaric oxygen increases the oxygenation of hypoxic brain tissue and decreases postembolic brain edema. Although the optimal pressure and gas mixture for this treatment are still under debate,[8] most authorities use the protocol developed by the U.S. Navy[2,4] (Fig. 1), which includes rapid recompression with air to 600 kPa (6 atmospheres absolute) for 30 minutes and subsequent exposure to 100 percent oxygen at 280 kPa (2.8 atmospheres absolute). A common variation we use includes an equal mixture of oxygen and nitrogen as the first stage of the Navy protocol. In this regimen, oxygen is used at the highest possible subtoxic partial pressure.

Barotrauma of the middle ear during descent is the most common disorder in divers.[9] The symptoms of middle-ear barotrauma vary from a sensation of pressure, followed by pain and conductive hearing loss, to rupture of the tympanic membrane, usually with an acute relief of pain, followed by whirling vertigo due to uneven caloric stimulation.[9] The condition is caused by an inability to equalize the pressure in the middle ear because of faulty clearing techniques, upper respiratory infection, or anatomical variations in the nasal skeleton.[10] When a difference in pressure of 90 mm Hg builds up between the middle-ear cleft and the nasopharynx, the eustachian tube cannot be opened. In this situation, edema, exudation, and hematotympanum may occur.[11,12] A forceful Valsalva maneuver results in further locking forces, which may lead to rupture of the tympanic membrane or inner-ear barotrauma.[11] With the diver's deeper descent, the eardrum may rupture.[13] The failure of the expanding gas to vent through the eustachian tube

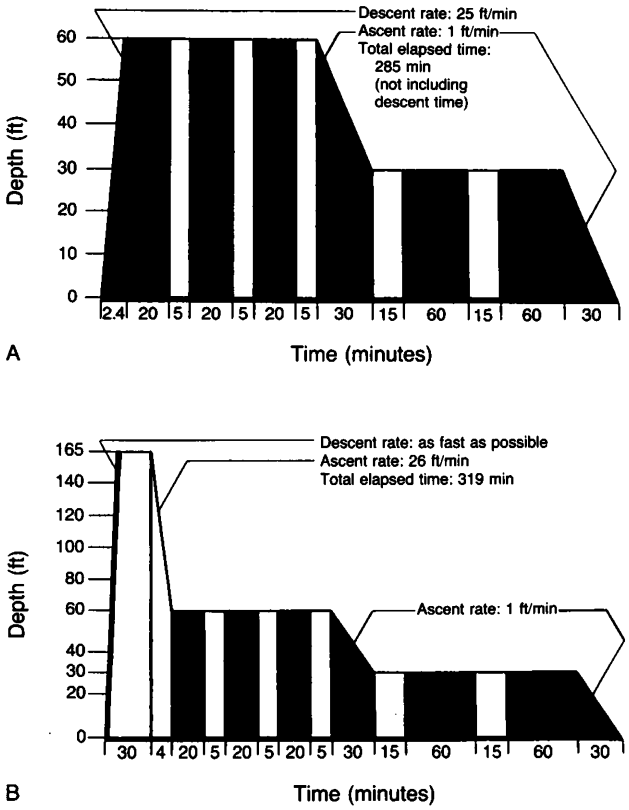

Figure 1. U.S. Navy Profiles of Pressure and Time Required for Therapeutic Recompression after Severe Decompression Sickness (Panel A) and Air Embolism (Panel B).

Solid areas denote intervals of exposure to pure oxygen, and open areas intervals of exposure to air. The values on the vertical axis in Panel B are shown slightly out of scale. To convert values in feet to meters, multiply by 0.3048.

may occasionally lead to middle-ear barotrauma during ascent.[9,11]

The management of middle-ear barotrauma consists of topical nasal and systemic decongestants. If purulent otorrhea is observed, antibiotics should be prescribed. Most tympanic-membrane perforations heal spontaneously if infection is controlled and normal eustachian-tube function is restored.[9,11] Concomitant inner-ear barotrauma should be ruled out in all cases of middle-ear barotrauma.[11]

Inner-ear barotrauma may be a consequence of forceful efforts to equalize middle-ear pressure when the eustachian tube is locked and blocked.[11] The main symptoms are persistent vertigo, sensorineural hearing loss, and loud tinnitus. This increases intracranial pressure markedly and may cause the rupture of inner-ear membranes or the creation of labyrinthine window fistulae, with consequent impairment of cochlear and vestibular function. Inner-ear barotrauma may also occur independently of middle-ear barotrauma.[14,15] The primary treatment is complete bed rest, with the patient's head elevated to avoid an increase in the pressure of the cerebrospinal fluid that might aggravate a possible perilymphatic leak or the admixture of endolymph and perilymph, if there are

tears in the labyrinthine membrane.[14] The deterioration of inner-ear function indicates a need for exploratory tympanotomy and the patching of a round or oval window.[9,11]

Alternobaric vertigo is a condition manifested by an asymmetric increase in pressure in the right and left middle ears that exceeds a threshold difference of 45 mm Hg. This pressure difference is transferred to the labyrinth, resulting in nausea, vomiting, and disorientation. Although these symptoms are usually mild, when they occur underwater they may lead to aspiration of water and drowning.[16]

Paranasal-sinus barotrauma, the second most common disease of divers,[9] is frequently related to chronic dysfunction of the nasal or paranasal sinus, with blockage of the sinus ostia. Pain over the frontal sinus is the predominant symptom, often followed by epistaxis. The relatively long and tortuous course of the nasofrontal duct may explain the vulnerability of the frontal sinus.[17] Treatment for sinus barotrauma includes the use of topical and systemic decongestants or adrenergic agents, and of antibiotics if a purulent nasal discharge is present. Although recovery is usually uneventful with conservative therapy,[17] the patient should be followed closely for the potential spread of infection to orbital and intracranial structures.[18]

Several uncommon forms of pressure-related diving diseases, not discussed in detail here, are listed in Table 1.

Table 1. Some Forms of Barotrauma.

Condition (Reference)	Symptoms and Signs	Pathogenesis
Face squeeze (Edmonds and Thomas[19])	Edema and hemorrhage of facial skin and conjunctiva	The pressure of gas in the space inside the diving mask was not equalized during descent
Skin barotrauma (Edmonds et al.[2])	Linear bruises to trunk and skin of extremities	The volume of air pockets between the dry diving suit and the skin was reduced during descent
Dental barotrauma (Edmonds et al.[2] and Edmonds and Thomas[19])	Pain, possible breakdown of tooth	The volume of air spaces at the roots of infected teeth or adjacent to fillings changed rapidly during descent or ascent
Facial baroparesis (Eidsvik and Molvaer[20])	Transient ischemic neurapraxia	A dehiscent intratympanic segment of the facial nerve compressed because of overpressure
Stomach rupture (Edmonds et al.[2])	Severe pain, shock, generalized peritonitis	Gas overexpanded during rapid and uncontrolled ascent

Nitrogen Narcosis

A sufficiently raised partial pressure of nitrogen in the tissue of the nervous system induces signs and symptoms of narcosis, which worsen in proportion to increases in the ambient partial pressure (the "rapture of the depths").[21,22] The clinical picture of nitrogen narcosis is similar to that of alcohol intoxication, and is characterized by temporary impairment of intellectual and neuromuscular performance and by changes in personality and behavior. The condition may occur at any depth exceeding 30 m (100 ft). At an extreme depth of 90 to 100 m it may lead to hallucinations, unconsciousness, or death.[21] Divers recover rapidly from nitrogen narcosis when they ascend to a shallower depth, where the narcotic effects of the gas are reduced.[23] Alcohol, fatigue, cold, and increased arterial carbon dioxide tension all increase susceptibility to nitrogen narcosis.[21,23] The chief danger of this condition stems from the impairment of the diver's reactions to the environment, which severely diminishes the ability to function in an emergency. Nitrogen narcosis is therefore a common precipitant of diving accidents and drowning. It is easily prevented by avoiding air diving (with compressed air) to a depth of more than 30 to 40 m.

Decompression Sickness

When a diver breathes air under increased pressure, the tissues are loaded with increased quantities of oxygen and nitrogen (Henry's law). Oxygen is used in tissue metabolism, whereas nitrogen, which is physiologically inert, is not. Thus, the nitrogen content of a tissue increases in proportion to the ambient pressure and also in relation to the tissue's fat content, since nitrogen is about five times more soluble in fat than in water.[24]

When the ambient pressure decreases as the diver returns to the surface, the sum of the gas tensions in the tissue may exceed the absolute ambient pressure. At this point, a state of supersaturation is created that may lead to the liberation of free gas from the tissues and to the onset of decompression sickness. The liberated gas can disturb organ function by blocking arteries, veins, and lymphatic vessels; in addition, its expansion can rupture or compress tissues. When gas is found in a space with rigid boundaries, as in a muscle enveloped by fascia, it may lead to compartmental syndrome. Gas may also form within cells, causing the rupture of the cell membrane. Finally, reactions at the interface of blood and gas bubbles can activate the clotting cascade.[24,25]

The appearance of clinical symptoms during decompression can be largely avoided if rates of ascent are controlled with the use of stops during the ascent, as specified in decompression tables such as those of the U.S. Navy[26] or the Royal Navy.[27] However, severe decompression sickness may occur despite meticulous adherence to decompression tables.[28] Moreover, "silent" (asymptomatic) bubbles have been detected ultrasonically in the pulmonary circulation of divers during decompression from dives that do not require stops during ascent[29,30] or from a series of dives made without equipment while the diver's breath is held ("breathhold dives") that do not require such stops.[31] The long-term importance of silent bubbles is unclear. Risk factors that may enhance the accumulation of inert gases in tissues or slow their release include advanced age, female sex,[32] obesity, elevated arterial carbon dioxide tension, low water temperature, poor physical fitness, and level of physical activity during the dive. Dives repeated in a series are also a major source of difficulty, since after one dive a considerable quantity of nitrogen may remain in the tissues. This

exposes the diver to an increased risk of decompression sickness during subsequent dives.[26]

It is customary to classify cases of decompression sickness into two types on the basis of clinical manifestations. Type I involves a mild insult, and Type II is the more severe variety, which may lead to permanent neurologic injury and death.[33] The most common manifestation of Type I is localized joint pain ("the bends"), which generally develops within 1 hour of surfacing and ranges from mild discomfort ("niggles") to very severe pain, which may gradually increase during the following 24 to 36 hours. Joint pain is attributable to the supersaturation and separation of an inert gas within the poorly perfused periarticular tight connective tissues during decompression. The joints most commonly affected in scuba divers are the elbow and shoulder, and they are involved three times more often than the knee and hip. The pain is usually not exacerbated by motion, and local tenderness or signs of inflammation are very uncommon.[24,25]

Other manifestations of Type I decompression sickness include pruritus and skin rashes due to the presence of gas bubbles in subcutaneous glands. Pruritus generally resolves within 10 to 30 minutes, even without treatment. Skin marbling may occur when subcutaneous microbubbles cause venous stasis. These mild symptoms signal possible systemic involvement, which may progress rapidly and require recompression treatment. Lymphatic obstruction can also occur, leading to lymphadenopathy, pain, and localized edema, which are only rarely alleviated by recompression.

Symptoms of Type II decompression sickness most commonly appear 10 to 30 minutes after the diver surfaces[24,25] and often start with a feeling of malaise and fatigue. However, the symptoms may develop insidiously over several days. Central nervous system involvement is usually characterized by damage to the spinal cord. Paresthesias that progress to numbness are the most common presenting symptoms. In addition, the involvement of motor pathways may lead to paraparesis and paraplegia. The spinal lesion is usually associated with bladder paralysis, fecal incontinence, and occasionally priapism. Referred abdominal pain, as well as girdle and back pain, is quite common.[34]

The symptoms of cerebral involvement include severe headache, blurred vision, diplopia, tunnel vision or scotomas, dysarthria, dizziness, and vertigo, as well as mental and personality changes. Severe cerebral involvement may lead to convulsions and death.[24,25] In addition to the separation of a free gas, neurologic manifestations of decompression sickness are also attributed to paradoxical gas embolization; namely, bubbles that escape pulmonary filtration[35] enter the arterial circulation through a patent foramen ovale or an atrial–septal defect,[36] and may even be generated de novo as microemboli within the arterial circulation. These may occlude capillaries or damage the blood–brain barrier.[37]

Poorly perfused areas of the nervous system with a high fat content are at a particularly high risk of decompression damage from underwater diving. This explains the typical appearance of extensive demyelinating lesions in the central nervous system.[38,39] Laboratory[40] and clinical[41,42] studies suggest that subclinical cerebral damage occurs in divers. In this regard, delayed resolution of decompression symptoms may also be associated with permanent cerebral lesions, observable in studies using computed tomography and nuclear magnetic resonance imaging.[43] Finally, a recent scintigraphic study using single-photon-emission tomography has suggested that there is multifocal cerebral involvement in Type II decompression sickness, even when clinical symptoms and signs are confined to damage to the spinal cord.[42]

Inner-ear decompression sickness has been reported after recreational scuba diving, although most cases are the result of decompression with mixtures of helium and oxygen or of very deep air diving.[44,45] Blockage of the microcirculation of the inner ear, hemorrhage, and exudation of protein in the cochlea irritate the endosteum in the bony semicircular canals. This initiates osteoblastic differentiation, leading to fibro-osseous labyrinthitis. The main manifestations of this condition are severe sensorineural hearing loss, tinnitus, and vertigo.[46]

Pulmonary manifestations of Type II decompression sickness occur when excessive numbers of gas bubbles liberated during decompression are trapped in the pulmonary arteriolar circulation. As little as 5 ml of air accumulating as a bolus in the pulmonary arterial circulation may cause retrosternal discomfort, whereas 25 ml causes severe retrosternal pain, accompanied by extreme fatigue, dry cough, and even severe respiratory distress (known among divers as "the chokes").[24,25]

Contrary to what was previously believed, clinical reports supported by new neurodiagnostic techniques have shown that among those engaging in air diving Type II decompression sickness is just as common as Type I.[28,41,42] It should be emphasized that although decompression sickness is a diffuse, multifocal disease, the differentiation between Type I and Type II determines the urgency of treatment and the selection of the appropriate treatment protocol.

First aid for decompression sickness includes the administration of inhaled oxygen at the highest possible concentration and proper hydration. The patient must be transported immediately to a recompression chamber, with a portable one-person chamber used for the transfer if such a chamber is available, because the time to the administration of treatment is one of the main determinants of outcome.[47] Even after a long delay, however, recompression treatment should never be denied, because there remains a chance of favorable results.[48]

The rationale for the use of hyperbaric oxygen to treat decompression sickness is similar to that described earlier to treat air emboli — namely, the need to eliminate gas bubbles and alleviate damage to hypoxic tissue. In principle, recompression protocols use

oxygen at pressures sufficient to produce a therapeutic effect without causing oxygen toxicity. In most centers providing hyperbaric-oxygen therapy, treatment is given according to the U.S. Navy treatment tables.[26] A recompression protocol is selected according to the severity of illness (Type I or Type II) and the patient's response during therapy. The most commonly used initial treatment regimen is shown in Fig. 1. If this regimen fails, an extension of this protocol is possible. Other therapeutic regimens are available for severe cases.[25] Subsequently, short (90 minute) treatments with hyperbaric oxygen are usually given daily until there is no further improvement in symptoms.

Another therapeutic option is the use of mixtures of helium and oxygen. This technique was first suggested by the U.S. Navy in 1958,[49] and it is currently used by some professional diving companies and treatment centers, including our institute. It seems that when such mixtures are used for recompression, nitrogen is eliminated faster, because it is much more soluble than helium in the structural fats of the central nervous system. The superiority of oxygen and helium to oxygen alone in the treatment of decompression sickness has recently been reported in several laboratory studies,[50] and it awaits further clinical support.

Because of hemodynamic changes encountered during severe decompression sickness that lead to hemoconcentration, intravenous fluids should be administered. Others have also recommended high doses of steroids and mannitol to reduce local edema in the nervous system and maintain an intact blood–brain barrier.[51]

Because of the unique medical problems encountered in scuba diving, the importance of providing medical screening for applicants to diving courses cannot be overemphasized. In principle, diving is absolutely contraindicated in persons subject to spontaneous pneumothorax, as well as in those with air-trapping pulmonary lesions or bronchial asthma. The same applies to persons with conditions that impair the level of consciousness (e.g., epilepsy, diabetes mellitus, and drug addiction) or affect the person's ability to equalize pressure in the middle ear and the sinuses.

In view of the growing number of scuba divers and diving accidents, any clinician working in general practice or in an emergency department should be acquainted with the basics of diving medicine. In the United States, the Divers Alert Network at Duke University Medical Center provides a 24-hour information and consultation service; the telephone number is (919) 684-8111.[1] Basic knowledge, combined with a high index of suspicion, may prevent unnecessary delays in the diagnosis and proper treatment of potentially hazardous and irreversible conditions related to scuba diving.

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