Chapter 18

PHYSIOLOGY AND MEDICAL ASPECTS OF DIVING

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INTRODUCTION

Diving as a military activity is sufficiently common that any military physician has a significant probability of involvement in diving operations or in treating or examining divers during his or her career. In addition, military physicians may be called on to treat civilian recreational divers with diving-related problems when no appropriate facilities exist in the civilian community.

Diving is not new to military operations. The first recorded use of breath-hold divers for military purposes dates from the 5th century BCE, when Scyllus and his daughter Cyane saved the fleet of Xerxes by freeing the ships' anchors, allowing them to get underway rapidly when threatened by a sudden storm.[1] The Syracusans are said to have trained divers to swim underwater and damage enemy ships.[2] Divers of Tyre were employed to cut the anchor ropes of Alexander the Great's ships during the siege of Tyre in 332 BCE.[3] However, not until the 19th century, after many technological advances, did diving become a military, as well as an industrial, specialty. Military diving operations today include Special Operations; explosive ordnance disposal and mine clearance; salvage of ships; location and retrieval of aircraft or other equipment lost in the water; clearance of wrecks and debris from harbors and waterways; ship-hull maintenance and repairs (ship's husbandry); repair of piers, locks, dams, and other associated structures; and reconnaissance. The military services employ military and civilian divers to fulfill these varied missions worldwide. See also Chapter 17, Military Diving Operations, for additional discussion.

Figure 18-1 shows the relationship of the depth of the oceans to the attempts humans have made to enter them. Water is an unforgiving and difficult environment in which to work. Most diving operations are done in cold water with a minimum of visibility. The diver's ability to work is also degraded by the physically challenging equipment used, the respiratory effects of increased breathing resistance, and other physiological changes due to pressure. The risk of injury or illness is largely dependent on the differential in ambient pressure experienced by the diver. Many advances in technology and procedures have helped mitigate the dangers of diving, but there will always be challenges to putting humans in the sea. This chapter discusses the physics and physiology of diving as they relate to humans living and working in the underwater environment, while highlighting common health effects and mitigation strategies.

PHYSICAL PRINCIPLES

Certain fundamental physical principles need to be understood before the deleterious effects of diving can be appreciated. Foremost among these are the behaviors of gases under pressure. Barometric pressure is the force per unit of surface area exerted by the atmosphere. Under natural conditions, this pressure is the result of gravity acting on the column of air that stands several miles above the surface of the Earth; its value was initially measured against a column of mercury and is thought to have been first described by Italian physicist Torricelli in the mid-1600s. It was given an average value of 760 mm Hg at sea level.

The primary unit of pressure in the International System of Units (SI units) is the Pascal (Pa, a unit derived from newtons per square meter), where 1 atmosphere (atm) equals 101.3 kPa. Other units are also sometimes used (Table 18-1).

Many pressure gauges (eg, blood pressure gauges) read zero at ambient (surface) pressure, thereby measuring only differential pressure. However, physiological calculations require the use of absolute pressure, where zero corresponds to a complete vacuum. Therefore, every expression of pressure must be designated as gauge (g) or absolute (a). Conversion between the two requires knowledge of ambient barometric pressure at the time the measurement was made.

Actual pressure underwater differs between fresh and salt water and varies slightly from one area of the ocean to another, depending on local salinity and temperature. One atmosphere is equivalent to 10.13 m of depth in seawater (msw) or 33.08 ft of depth in seawater (fsw). In fresh water, 1 atm = 10.38 msw, or 33.83 fsw.

The following formula may be used when converting from diving depth to atmospheres:

$$\text{absolute pressure} = (D + De) / De$$

where D represents the depth in units of length and De represents the number of those units equivalent to 1 atm. When solving for pressure using atmospheres (atm) as the base unit of measure, absolute pressure is described as atmospheres absolute (ata). NOTE: this equation assumes a pressure of 1 atm at the surface of the water and requires correction for high-altitude diving.

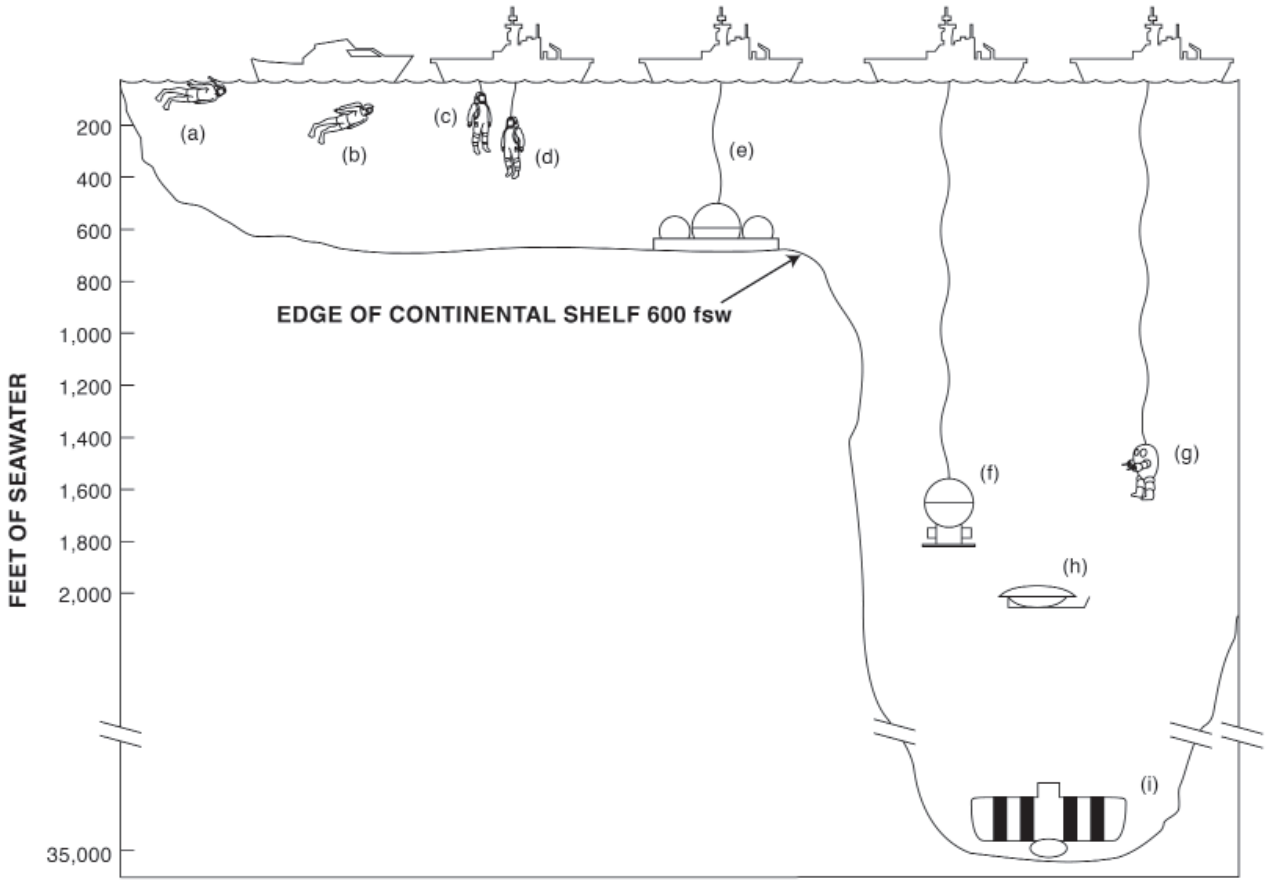

Figure 18-1. Various forms of diving and other submarine activities in relation to ocean depth. (**a**) Breath-hold diving is usually not deeper than 100 feet of seawater (fsw) or 30 meters of seawater (msw), although some divers can go deeper. (**b**) Self-contained underwater breathing apparatus (scuba) diving with air is usually limited to a normal working depth of 130 fsw (40 msw). (**c**) Surface-supplied air diving is usually limited to a normal working depth of 190 fsw (58 msw) for the US Navy, and 220 fsw (67 msw) for commercial or technical diving. (**d**) Surface-supplied helium–oxygen diving without a diving bell is usually limited to a depth of about 300 fsw (91 msw). (**e**) The deepest seafloor habitat operation is at 610 fsw (185 msw). (**f**) The deepest saturation diving at sea operation is at 1,752 fsw (534 msw). (**g**) The deepest dive in an armored 1-atm suit is at 2,000 fsw (610 msw). (**h**) Research submersibles can operate to depths of about 2,000 fsw (610 msw). (**i**) The bathyscaphe Trieste has been to the deepest-known depth of the ocean, 35,814 fsw (10,916 msw), in the Marianas Trench. More recently the Deepsea Challenger descended to 35,756 fsw (10,898 msw) in the Challenger Deep area of the trench. Note that the ordinate, marking depth, has been shortened. If this graphic were drawn to scale, a page at least 1.26 m long would be required to display ocean depth to the bottom of the Marianas Trench.

Gas Laws

Changes in barometric pressure and in the composition of inspired gas affect the human body in ways that reflect the physical behavior of gases in gaseous mixtures and liquid solutions. A good grasp of gas laws and related physical principles is therefore prerequisite to understanding the physiological effects of the pressure changes imposed by flying and diving.

Pressure Effect (Boyle's Law)

The volume of a given mass of gas varies inversely with absolute pressure when temperature is held constant. In mathematical terms:

$$P_1 / P_2 = V_2 / V_1$$

or the formula can be rewritten:

$$P_1 \bullet V_1 = P_2 \bullet V_2$$

TABLE 18-1

PRESSURE EQUIVALENTS

Atmospheres	Bars	10 Newton per cm^2	Lbs per Square Inch	Columns of Mercury at 0°C		Columns of Water at 15°C			
				Meters	Inches	Meters	Inches	Feet (FW)	Feet (FSW)
1	1.01325	1.03323	14.696	0.76	29.9212	10.337	406.966	33.9139	33.066
0.986923	1	1.01972	14.5038	0.750062	29.5299	10.2018	401.645	33.4704	32.6336
0.967841	0.980665	1	14.2234	0.735559	28.959	10.0045	393.879	32.8232	32.0026
0.068046	0.068947	0.070307	1	0.0517147	2.03601	0.703386	27.6923	2.30769	2.25
1.31579	1.33322	1.35951	19.33369	1	39.37	13.6013	535.482	44.6235	43.5079
0.0334211	0.0338639	0.0345316	0.491157	0.0254	1	0.345473	13.6013	1.13344	1.1051
0.09674	0.09798	0.099955	1.42169	0.073523	2.89458	1	39.37	3.28083	3.19881
0.002456	0.002489	0.002538	0.03609	0.001867	0.073523	0.02540	1	0 .08333	0.08125
0.029487	0.029877	0.030466	0.43333	0.02241	0.882271	0.304801	12	1	0.975
0.030242	0.030643	0.031247	0.44444	0.022984	0.904884	0.312616	12.3077	1.02564	1

1. Fresh water (FW) = 62.4 lb/ft^3; salt water (FSW) = 64.0 lb/ft^3.
2. The SI unit for pressure is kilopascal (kPa); 1 kg/cm^2 = 98.0665 kPa and by definition 1 BAR = 100.00 kPa @ 4°C.
3. In the metric system, 10 MSW is defined as 1 BAR. Note that pressure conversion from MSW to FSW is different than length conversion; ie, 10 MSW = 32.6336 FSW and 10 m = 32.8083 ft.

Reproduced from: US Department of the Navy. *US Navy Diving Manual.* Rev 6. Washington, DC: Naval Sea Systems Command; 2011: 2-32, Table 2-10. NAVSEA 0994-LP-100-3199.

where P represents pressure, V represents volume, and the subscripts 1 and 2 refer to the condition before and after a pressure change. For example, if a flexible container is filled with 1 L of gas at 1 ata and then subjected to increasing pressure, at 2 ata the volume will be halved (0.5 L) and at 3 ata it will be one third of its original volume (0.33 L). Ascent to an altitude of 18,000 ft (0.5 ata) would cause the volume to double (2 L).

Because gas in body cavities is saturated with water vapor at body temperature, Boyle's law is often applied in the following form:

$$(P_1 - P_{H_2O}) / (P_2 - P_{H_2O}) = V_2 / V_1$$

where P_{H_2O} represents water vapor pressure at body temperature (47 mm Hg). The addition of the term P_{H_2O} is important when doing respiratory studies and in high-altitude work but is insignificant when calculating pressure volume relationships in diving.

Temperature Effect (Charles's Law)

The volume of a given mass of gas is directly proportional to its absolute temperature when pressure is held constant. According to Charles's law, this relationship can be expressed mathematically: $V_1 / V_2 = T_1 / T_2$, or the formula can be rewritten:

$$V_1 \bullet T_2 = V_2 \bullet T_1$$

where T_1 and T_2 represent initial and final temperature, respectively. For this purpose the temperatures must be expressed in absolute units, which can be calculated as T°K (Kelvin) = T°C (Celsius) + 273.

Universal Gas Equation

Boyle's and Charles's laws are often expressed in a single equation for calculating the effects of concurrent changes in pressure and temperature, as follows:

$$(P_1 \bullet V_1) / T_1 = (P_2 \bullet V_2) / T_2$$

The gas laws listed above are discussed in terms of a flexible container that fully accommodates alterations in volume due to changes in pressure and temperature. However, considering real-world applications for gas in a rigid container when volume is held constant, the increasing ambient pressure will eventually cause structural collapse of the container, whereas decreasing pressure will force the walls to burst. For this reason, the inability to ventilate semi-rigid, gas-filled body compartments such as the lungs and middle ear during severe pressure changes can cause incapacitating or fatal injury in the form of a diving "squeeze" or overinflation.

Partial Pressure in Gaseous Mixtures (Dalton's Law)

The total pressure exerted by a mixture of gases is equal to the sum of the pressures that each gas would exert if it alone occupied the container. According to Dalton's law, this can be expressed mathematically:

$$P_t = P_1 + P_2 + P_3 + P_n$$

where P_t represents the total pressure of the mixture, and $P_1 + P_2 + P_3 + P_n$ represent the component partial pressures. The partial pressure of a single gas in a mixture can be calculated from the following relationship:

$$P_x = F_x \bullet P_t$$

where P_x represents the partial pressure of gas x, and F_x represents the fractional concentration of that gas in the mixture. At 1 ata, oxygen and nitrogen exert partial pressures of 0.21 and 0.79 atm, respectively. On raising barometric pressure to 3 ata, these partial pressures triple, to 0.63 and 2.37 atm.

Gases in Solution (Henry's Law)

The quantity of a gas that dissolves in a liquid is directly proportional to its partial pressure in the gas phase, and inversely proportional to the absolute temperature, assuming that no chemical reaction takes place. When the partial pressure of the gas is reduced, a proportional amount of that gas will emerge from solution and may form bubbles in the liquid phase. This is the primary initial mechanism for the development of decompression sickness (DCS).

Transmission of Pressure in a Liquid (Pascal's Law)

Pressure exerted at any point on a confined liquid is transmitted uniformly in all directions. As opposed to hollow organs, which may contain an airspace, solid organs are mostly liquid, and therefore, pressure exerted on them is distributed equally throughout the body and does not change tissue volume.

Buoyancy

As stated in Archimedes's principle, any object immersed in liquid will be buoyed up by a force equal to the weight of the water displaced. For example, an object that weighs 100 lb in air and displaces 90 lb of water will be 10 lb heavier than the water displaced and, therefore, sink. If the amount of water displaced is equal in weight to the object, its depth remains constant because it is neutrally buoyant.

Buoyancy is important to a diver because it affects the amount of work needed to change or maintain depth. A positively buoyant diver rises, and a negatively buoyant diver sinks. If the buoyancy is not appropriate, a catastrophic accident can occur. The desired state of buoyancy depends on operational requirements. For example, a combat diver swimming into an enemy harbor at 20 fsw will try to achieve neutral buoyancy, both to make the best speed and to conserve breathing gas. If negatively buoyant, the diver might swim too deep and develop oxygen toxicity; if positively buoyant, the diver might break the surface and be detected. On the other hand, a diver doing salvage work on a hull of a ship may want to be strongly negatively buoyant so that he or she can handle the equipment and tools required without being unsteady or easily movable in the water.

Control of buoyancy is achieved by various methods. The most common is by adding or removing gas from the diving dress (a garment that provides thermal and mechanical protection) or the buoyancy compensator (an inflatable vest for adjusting buoyancy). Weights or weighted equipment are also used to provide initial negative buoyancy for entering the water. When a diver wishes to return to the surface, he or she may establish positive buoyancy by dropping weights or inflating the buoyancy compensator. Certain types of protective clothing, such as the closed-cell neoprene suit, are positively buoyant at shallow depths but become less so at greater depths as the material collapses.

UNDERWATER PERTURBATIONS OF VISION AND HEARING

Vision

Vision underwater is affected by the mask a diver wears, absorption of light by the water, intensity of light, and turbidity of the water, which depends on the amount of material suspended in it. Looking through a diving mask or helmet magnifies objects underwater by 25% to 35% and therefore makes them look closer than they are (Figure 18-2). This displacement of the image is a result of refraction of light as it passes from water through the faceplate material to gas, and it can be confusing to novice divers. A mask or helmet also restricts the peripheral vision of the diver by as much as 50%. Masks or helmets with larger viewing areas increase the field of view but introduce visual distortion.

Stereoacuity, or depth perception, is the ability to determine the relative distance between objects. It is also adversely affected underwater as a result of nar-

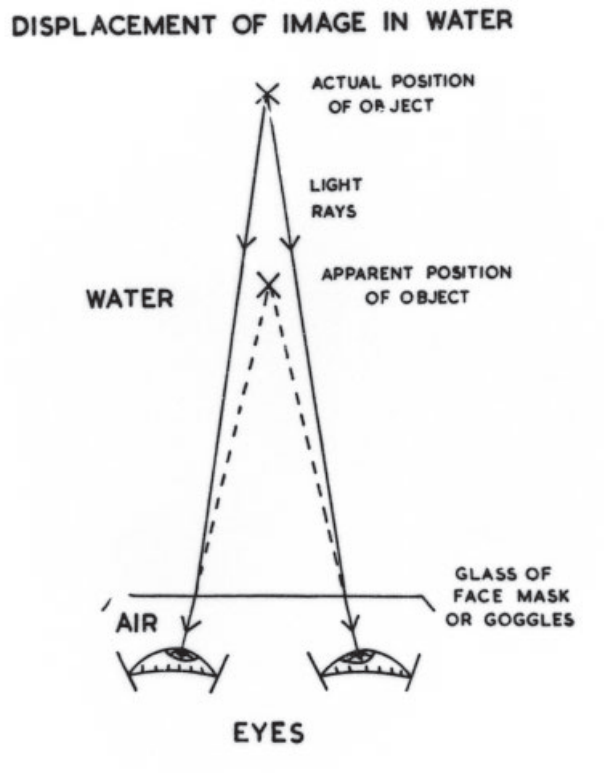

Figure 18-2. When seen underwater, images are displaced toward the diver. The closer apparent position is caused by the refraction of light rays as they pass through the water, the material of the face mask, and the gas inside the mask.

rowed field of view due to mask, change in refraction of light causing relative magnification, decreased contrast due to increased light absorption, and decreased clarity of image due to increased turbidity. This is especially noticeable in clear, well-illuminated water and worsens with decreasing illumination and increasing turbidity.

Absorption of light affects underwater vision by decreasing available illumination. In clear water, only about 20% of incident light penetrates to a depth of 33 ft (10 m). Available light also decreases with a decreasing angle of incidence of sunlight to the water surface (as the sun approaches the horizon), as more light is reflected instead of penetrating the surface. However, in clear water with a high sun angle, useful illumination may be found as deep as 198 to 297 ft (60–90 m). As the turbidity increases, the penetration of light decreases. In many rivers, lakes, and harbors, there may be no usable light even within a few feet of the surface.

Changes in color perception occur because water selectively absorbs the very short and very long wavelengths of light. At fairly shallow depths, the red-orange spectrum is lost, and most objects appear blue-green. For example, at a depth of about 10 ft (3 m), blood appears greenish rather than red. If an underwater light source is used, however, the colors appear normal.

Vision in dry, hyperbaric environments is not significantly changed. The only vision-related problems that may arise in repetitive or prolonged saturation dives (see Chapter 17, Military Diving Operations) are a slight loss in visual acuity and a tendency to esophoria.

Hearing

In a dry, hyperbaric environment the only effect on hearing appears to be an increase in the hearing threshold with increasing depth. This is not due to changes in bone conduction, which remains normal at depth, but to changes in conductivity in the middle ear as a result of increasing gas density. This occurrence reverses with decreasing pressure, and occurs in both nitrogen–oxygen and helium–oxygen environments.

Hearing underwater is a completely different situation. Because the densities of tissue and water are similar, a submerged head is "transparent" to sound energy. In addition, water in the external ear canal damps the vibrations induced in the tympanic membrane. Therefore, hearing underwater occurs through bone conduction rather than through the middle ear, with a 50% to 75% decrease in the hearing threshold.

Sound is difficult to localize underwater. It travels much faster in water than in air, reducing the difference in time at which sound arrives at each ear. The human brain is uniquely tuned to interpret this time difference in air, but at shorter intervals, as experienced underwater, localization can often be impossible even though the sound is heard well.

Speech intelligibility is a problem in diving. In water, it is almost impossible for divers to converse without a communications device because too much power is required for through-water sound transmission by voice alone. Divers wearing rigid helmets can converse by touching helmets, so that sound is transmitted directly from helmet to helmet. While speech intelligibility decreases somewhat as the density of a nitrogen–oxygen mixture increases, speech is almost impossible when a helium–oxygen mixture is used. This is because helium shifts the resonance of vocalizing structures and gas-filled cavities, drastically changing the timbre of the vowels. This results in a high pitched, nasal quality referred to as "Donald Duck" speech. Most saturation divers find that their understanding of such speech improves after several days under pressure, but it is still poor.

Permanent hearing loss was a common problem among divers in the past because of high noise levels in helmets and chambers. Although divers who wear a "hard hat" (the classic rigid diving helmet) may still have this problem, newer diving equipment is quieter and hearing loss is not common. If a hyperbaric chamber does not have muffling for the compression and exhaust systems, divers inside the chamber and operators outside should use individual hearing protection to prevent temporary or permanent hearing loss.

A submarine's sonar uses sound signals propagated into water for certain aspects of operation (eg, active sonar). Standards for diving operations near active sonar sources are defined in appendix 1A of the *US Navy Diving Manual*.[4] Exposure to sonar can produce both auditory and nonauditory effects. Exposure to high sound pressure in water is similar to exposure to loud noise in air, causing either a temporary threshold shift or permanent sensorineural hearing loss. Whether decreased hearing is temporary or permanent depends on the sound pressure level and the frequency of the sonar signal.

Unless a sonar signal is unexpectedly energized at close range, there is little danger because a diver who is swimming in the area of a sonar source can hear or feel the signal and stay clear of the danger zone. Divers exposed to sonar have reported feeling vibrations (in parts or the entire body), vertigo, nausea, general discomfort, disorientation, decreased ability to concentrate, fatigue, and transient joint pain. The vibration is a mechanical phenomenon, whereas the vertigo, nausea, and discomfort indicate effects on the vestibular system. Similarly, nausea, vomiting, and vertigo are occasionally described in patients with vestibular signs or symptoms induced by loud noise (the Tullio phenomenon). The combination of disorientation, decreased concentration, and fatigue are similar to symptoms of motion sickness and probably result from sound stimulation of the vestibular system. The cause of joint pain is uncertain. Adherence to the published exposure standards should prevent these symptoms.

BAROTRAUMA

Barotrauma refers to injury caused by changes in pressure. Barotrauma can occur on descent or ascent when a gas-filled cavity fails to equilibrate with changing ambient pressure (see also Chapter 16, Aerospace Medicine). Almost any gas-filled cavity in the body can be affected by barotrauma. For example, gastrointestinal barotrauma can occur if gas is swallowed. The gas expands during ascent, causing abdominal distress, cramping, flatus, or eructation. No therapy is usually required because the gas will be expelled over a short time, but several cases of gastric rupture after decompression have required emergency attention.[5,6]

Less obvious body cavities are restored dental caries and gingivitis, which can also become gas-filled. Often referred to as aerodontalgia, dental barotrauma results from poor or eroded fillings or gum infection, and can occur on both ascent and descent. On descent, an air space can be filled with gum tissue or blood, and pain may occur. Descent also may cause the thinned filling or cementum over a carious tooth to collapse. If gas has become trapped under a filling during a dive, the pressure change during ascent may cause the cavity walls to explode.

Barotrauma that occurs when pressure increases is commonly referred to as a "squeeze." For example, suit squeeze occurs in a poorly fitted wetsuit or in a dry suit with an insufficient gas supply to keep it slightly expanded. There may be no symptoms at all, or the diver may notice some pinching of the skin. Upon removal of the suit, irregular linear wheals or ecchymosis may be seen where the skin was pinched in folds of the suit material. No therapy is required. Mask squeeze can occur if a diver fails to equalize the pressure in the mask during descent. The space inside the mask is subjected to a relative vacuum and the skin under the mask becomes puffy and edematous, and may show small hemorrhages. Mask squeeze is more common around the eyes and in the conjunctivae. A severe squeeze will bruise the entire area under the mask.

Middle Ear Barotrauma

Descent

The most common form of barotrauma is middle ear squeeze. Anyone who has flown in an airplane or ridden in an elevator in a tall building has felt fullness in the ears during descent to ground level. This usually resolves by swallowing or yawning, which opens the eustachian tube and allows pressure equalization of the air on both sides of the tympanic membrane: ambient air and the air within the middle ear (see also Chapter 16, Aerospace Medicine). The consequences of not achieving equilibration are much worse in diving, where pressure changes far exceed the 1-atm maximum change in descent from altitude. The initial symptom of fullness in the ear progresses to pain if descent continues without equalization. The tympanic membrane retracts and small hemorrhages occur (Fig-

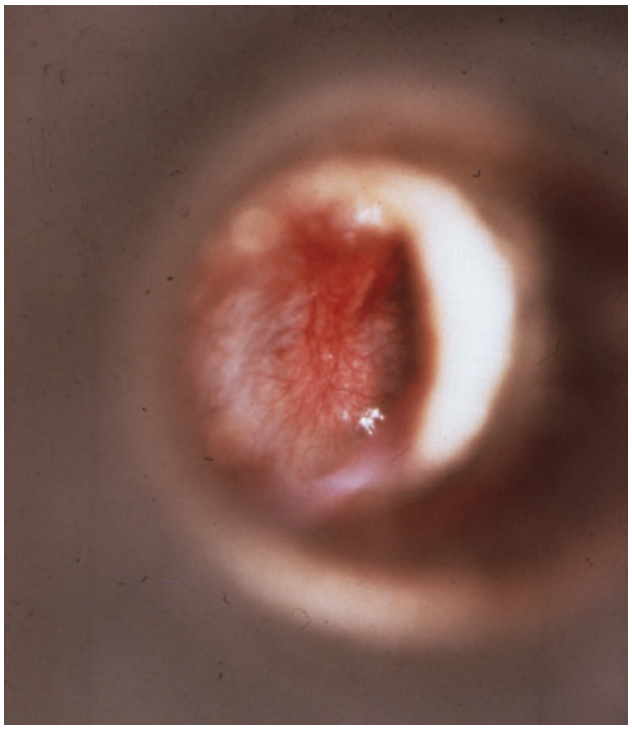

Figure 18-3. Photograph showing moderate barotrauma of the middle ear. Note the hemorrhaging around the handle of the malleus and surrounding tympanic membrane, and the slight retraction and dullness of the tympanic membrane.

ure 18-3). If the process continues, the relative vacuum in the middle ear causes serum and blood to fill the space, and eventually the tympanic membrane ruptures. If cold water then enters the middle ear, sudden severe vertigo may occur, producing disorientation, nausea, vomiting, and panic. Predisposing factors to eustachian tube dysfunction and middle ear squeeze are conditions that prevent easy opening and closing of the eustachian tube, such as upper respiratory tract infections, allergies, mucosal polyps, mucosal irritation from smoking, otitis media, or anatomical variations.

The diver should begin equilibration as soon as descent begins and continue every few feet. If the pressure differential is allowed to become too large, the eustachian tube will collapse to a point where it is "locked" and cannot be opened by any method. Methods of opening the eustachian tubes, or clearing the ears, include sliding the jaw around, opening the mouth, yawning, swallowing, and performing the Valsalva and Frenzel maneuvers. The Valsalva maneuver consists of closing the mouth, blocking the nostrils, and exhaling gently to increase internal gas pressure. A forceful Valsalva maneuver may produce pressure high enough to cause rupture of the round window with cochlear and vestibular damage (see **Inner Ear Barotrauma**, below). The Frenzel maneuver consists of closing the mouth, glottis, and nose, and contracting the muscles of the mouth and pharynx or swallowing. If these maneuvers do not work, the diver should ascend a few feet and try again. If repeated attempts do not allow equilibration, the dive should be aborted.

Prevention of middle ear barotrauma includes not diving when any condition exists that prevents proper eustachian tube function. Some divers often have "sticky ears," a condition in which eustachian tube function is present but equilibration is difficult to achieve. Nonsedative oral decongestants can be helpful but may not always be effective. Topical decongestants should be used with care because overuse can make matters worse.

There is no specific therapy for middle ear barotrauma except the prohibition of significant pressure change or the use of autoinflation techniques until the problem resolves. In the case of tympanic membrane rupture, close observation is needed to detect infection when it first occurs. For severe barotrauma, serial audiograms are recommended to exclude hearing loss that may require further investigation and therapy. Generally, a diver who has had an ear squeeze without perforation can return to diving within 2 weeks. The basis for return is complete resolution of signs and symptoms as well as demonstration of the ability to equilibrate pressure in the middle ear. If rupture has occurred, a month or longer may be needed before return to diving can be allowed. Recurrence is common if exposure to pressure is allowed too soon after rupture.

Ascent

Middle ear barotrauma of ascent is infrequent because the middle ear can normally vent passively through the eustachian tube when its pressure reaches about 50 cm of water over ambient. When it does occur, middle ear barotrauma of ascent is usually a consequence of middle ear barotrauma of descent. If blockage is severe, ascent without trauma may be impossible. Symptoms are pressure, pain, tinnitus, vertigo, or hearing loss. The Valsalva maneuver should be avoided while ascending because it increases pressure in the middle ear space and can also lead to pulmonary barotrauma (see below). If pain or vertigo develops on ascent, the diver should halt the ascent and descend a few feet to relieve symptoms before attempting ascent at a slower rate. There is usually little to observe clinically, but hyperemia or hemorrhage in the tympanic membrane may occur. No specific therapy is required unless the vertigo persists after pressure equalization.

Continued vertigo requires immediate investigation, especially if there is hearing loss.

Inner Ear Barotrauma

Any diver who has had difficulty equalizing the ears or has had barotrauma of the middle ear may also develop inner ear barotrauma, probably due to a rupture of either the round window or the vestibular membrane. Tinnitus is the most common manifestation, but hearing loss may occur at the time of barotrauma or within a few days. Some divers may experience vertigo, nausea, and vomiting. Hearing loss is usually sensorineural and is probably due to cochlear membrane rupture or hemorrhage.[7] If deafness is instantaneous and present in all frequencies, it is probably due to severe round-window rupture. If it is mild and develops over several days, a round-window fistula may be present. If air leaks into the perilymph, the deafness may change with head position. Immediate referral to an otolaryngologist is recommended to prevent permanent hearing loss. Bed rest with head elevation is required. The diver should be cautioned not to do anything that will raise the pressure of the cerebrospinal fluid, such as nose blowing, performing a Valsalva maneuver, straining at the stool, or coughing.

If there is no improvement within 24 to 48 hours or if the casualty's condition deteriorates, then surgical therapy should be considered. If improvement is steady, bed rest is continued for 1 week after improvement plateaus. Aeromedical evacuation, if required, should occur in an aircraft that can be pressurized to sea level. If helicopter transfer is the only method available, the pilot should be instructed to fly as low as is safely possible, preferably below 1,000 ft (305 m).[4] If deafness, vertigo, or tinnitus persist despite all therapy, the casualty should be advised against any future diving. Inner ear barotrauma and inner ear DCS can have similar symptoms (see **Decompression Sickness**, below), and their differentiation is important because DCS is treated by recompression, which is contraindicated in barotrauma.

Sinus Barotrauma

Sinus barotrauma during descent is probably the next most common problem after ear squeeze and involves the same predisposing factors. Symptoms include a feeling of pressure or tightness and pain over the face during descent. If the maxillary sinuses are affected, pain may be referred to the teeth. Often the diver may not realize that a mild squeeze has occurred until ascent, when the ostia may open, expelling blood and fluid.[7] Sometimes fluid, blood, or gas can be felt escaping from the sinuses. Divers who are unfamiliar with the problem can become extremely worried if this occurs. Although pain is usually relieved on ascent, it may persist for several hours. Persistent pain indicates that equilibration has probably not occurred and there is a risk of acute sinusitis. Valsalva maneuvers will usually allow equilibration of the sinuses. Nasal decongestants may also be used.

Sinus barotrauma rarely occurs during ascent. However, if the pressure in the sinuses is high enough, there may be a painful rupture of the ostia or mucosa into the nasal cavities. Treatment is the application of nasal decongestants.

Palsies of the fifth and seventh cranial nerves can result from sinus barotrauma (cranial nerve V) or middle ear barotrauma (cranial nerve VII).[8]

Pulmonary Barotrauma

Descent

Pulmonary barotrauma of descent occurs under two circumstances:

1. When a hard-hat diver accidentally descends faster than the gas can be supplied to the helmet or suit.
2. In breath-hold diving at depths so great that gas in the lungs is compressed to less than the residual volume.

The second circumstance is a very rare event in conscious divers because chest pain alerts the diver of excessive depth. For most people the maximum breath-hold depth is about 100 fsw (30 msw), although competitive free divers have exceeded 650 fsw (200 msw). See **Breath-hold Diving**, below.

Ascent

Pulmonary barotrauma of ascent is potentially the most severe type of barotrauma and can occur in all types of diving and in submarine escape (as well as in rapid decompression to high altitudes). Pulmonary barotrauma of ascent is caused by overinflation of the lung as the gas expands during ascent, either by breath-holding or by some local pathology that prevents gas from escaping from a portion of the lung. Pulmonary barotrauma is believed to be the second most common cause of mortality in divers (drowning is the most common). It has been reported in ascent from a depth as shallow as 3 to 6 ft (1–2 m) of water.[9,10] During naval submarine escape training, ascents are done under close supervision after medical screening, with emergency medical care immediately available.

Predisposing conditions for pulmonary barotrauma are breath-holding during ascent, previous spontaneous pneumothorax, asthma, blebs, pulmonary tumors, pleural adhesions, infections, pulmonary fibrosis, or any other disease that weakens the lung tissue or interferes with the free passage of gas.[4,11,12]

Pulmonary barotrauma may result in local or wide-ranging damage to lung tissue, characterized by disruption of the alveolar-capillary membrane and consequent passage of alveolar gas into (*a*) the mediastinum, causing mediastinal or subcutaneous emphysema; (*b*) the pleural space, causing pneumothorax; and (*c)* the capillaries, with probable resultant cerebral arterial gas embolism (AGE). These traumata can occur alone or in combination. Patients with pulmonary barotrauma but no signs or symptoms of AGE should be kept under observation for at least 24 hours, even if no therapy is required, to ensure that medical attention is available if the patient worsens, or if a new condition such as DCS emerges.

Pulmonary Tissue Damage

There may be no symptoms with local pulmonary tissue damage, but if the damage is widespread, the diver may surface with cough, hemoptysis, and dyspnea. Respiratory failure may result if enough alveoli have been ruptured. Severe symptoms of pulmonary damage require immediate respiratory support with 100% oxygen, as in the treatment of nonfatal drowning or acute respiratory distress syndrome. If pulmonary barotrauma is suspected, positive pressure ventilation should be avoided unless it is absolutely necessary for survival because it may cause further lung damage or other complications. Recompression is not needed unless symptoms or signs of AGE or DCS are present.

Mediastinal Emphysema

Mediastinal emphysema occurs when gas enters the mediastinum along perivascular sheaths or tissue around the airways. The gas may extend to the subcutaneous tissue of the neck (subcutaneous emphysema), the pericardium (pneumopericardium), or the retroperitoneal space (pneumoperitoneum). There may be no symptoms in mild cases, or symptoms may appear immediately or over hours, depending on the severity of the condition. Signs and symptoms include crepitus under the skin, faint heart sounds, paralysis of the recurrent laryngeal nerve, tachycardia, hypotension, cyanosis, coma, fullness in the throat, retrosternal discomfort, voice changes, dyspnea, dysphagia, syncope, shock, and unconsciousness. Crepitus that is heard over the precordium and is related to heart sounds is called Hamman sign. Gas that has dissected into the pericardial sac or between the pleura and pericardium may cause cardiac tamponade. A chest radiograph will confirm the diagnosis, or it may be the only evidence of mediastinal emphysema.

Mediastinal emphysema in divers is generally not life-threatening and usually requires no treatment. Breathing 100% oxygen will accelerate absorption of the inert gas in mild cases and is required for life support in severe cases. Severe cases may benefit from recompression to reduce the volume of gas in the tissues. Compression on 100% oxygen to a depth of relief (normally 5–10 ft [1.5–3 m]) for 1 hour should suffice.[4]

Pneumothorax

Pneumothorax occurs when increased pressure in the lung ruptures the pleura with results similar to a spontaneous pneumothorax. A hemopneumothorax may occur if blood vessels are torn. Symptoms include sudden onset of chest pain (possibly pleuritic) and dyspnea. Bilateral pneumothoraces may be present after a fast ascent, as in submarine escape training or in an emergency ascent from a dive. Signs include increased respiratory rate, decreased breath sounds, and increased resonance. If a tension pneumothorax is present, the classic tracheal shift to the unaffected side may occur, with shock and severe respiratory distress. If the pneumothorax is small, no therapy is necessary, although breathing 100% oxygen at the surface should accelerate pleural gas absorption.

If the patient is under pressure and decompression continues, a tension pneumothorax will result. If the patient is in a chamber, treatment includes recompression to reduce symptoms, 100% oxygen breathing (or a gas mix with increased partial pressure of oxygen), and the insertion of a chest tube or valve to allow decompression while avoiding expansion of the pneumothorax. If the diver is in the water when the pneumothorax occurs, however, none of the above treatments are possible.

If the patient is on the surface and severe symptoms are present, recompression should not be initiated unless required for treatment of AGE or DCS. Treatment at sea level does not differ from that for a pneumothorax resulting from other causes. If recompression is required for therapy of AGE or DCS, insertion of a chest tube should be considered before recompression. For emergency recompression, a needle with a nonreturn valve may need to be inserted prior to or during decompression. If recompression is needed and a chest tube with drainage is in place, care is required to prevent water and gas from being sucked into the chest during compression. If the system allows gas or

fluid into the chest, the chest tube should be clamped close to the chest wall until treatment depth is reached. Due to the possibility of infection, chest tubes should not be used in a recompression chamber unless absolutely necessary.

Arterial Gas Embolism

AGE can be life threatening or result in permanent injury, and requires immediate recompression. AGE is a complication of pulmonary barotrauma caused by the entry of gas through tissue tears into the pulmonary venous system, with further distribution through the left side of the heart to the arterial system. The gas bubbles generally come to rest in the cerebral circulation, or uncommonly in the coronary circulation. In the coronary system, immediate death can result through cardiac failure. In the cerebral circulation, the typical signs are of sudden arterial block, as in a stroke. The onset of signs and symptoms is sudden and dramatic, appearing almost always during the ascent or within 10 minutes of surfacing. The most common signs are altered mental status, unconsciousness, disorientation, and paresis. Paresis can occur in any distribution but is most often unilateral.

Other symptoms, depending on the anatomical location of the blockage, include vertigo, visual disturbances, dysphasia, sensory abnormalities, and convulsions. If the coronary system is involved, cardiac chest pain and dysrhythmias may be present. AGE, whether in the brain or elsewhere, causes ischemia in the surrounding tissue and induces coagulopathies, hemorrhages, and endothelial damage.[13] If recompression treatment is delayed, reperfusion injury may occur once the circulation has been reestablished.

Studies may show abnormal electrocardiographic, electroencephalographic, and brain scan findings, but if AGE is suspected, there is no reason to delay treatment while these tests are obtained. Immediate recompression is the key to adequate treatment of this syndrome. If treatment is delayed, the chances for a good outcome decrease and therapy becomes more difficult. In severe cases death may occur despite immediate and aggressive therapy. In mild cases the symptoms may resolve without any therapy. In some cases a patient may improve after initial symptom onset but relapse despite treatment.[14] There is no way to predict which case will resolve and which will not. Therefore, all cases of suspected AGE should be recompressed as soon as possible. Iatrogenic AGE can occur during interventions such as vascular catheterization, mechanical ventilation, open heart surgery, brain surgery, and kidney dialysis.[13,15] Treatment in these cases is no different from that in cases resulting from ambient pressure changes.

The treatment for AGE is immediate recompression, as discussed in further detail below. The *US Navy Diving Manual* recommends initial recompression to 60 fsw (18 msw) on 100% oxygen. If the symptoms resolve, treatment may be completed in accordance with US Navy Treatment Table 6. If the patient does not respond satisfactorily, further compression to a depth not to exceed 165 fsw (50 msw) is an option for attempting to reduce the size of remaining bubbles.[4] If immediate recompression is not available, 100% oxygen breathing by mask should be immediately initiated.

Transportation to a recompression facility should be done as gently and quickly as possible to prevent further distribution of bubbles to the brain or elsewhere. Air evacuation, if required, should occur at an altitude below 1,000 ft (305 m) and preferably in an aircraft that can be pressurized to sea level.[4] Other supportive therapy should be used as required.

In certain cases, there may be difficulty in determining whether the diver has AGE or cerebral DCS (see **Decompression Sickness**, below).

PATHOPHYSIOLOGICAL EFFECTS OF COMMON DIVING GASES

The nature of diving requires the creation of an artificial environment, one that simulates the very narrow parameters required to sustain life, where small variations can have drastic effects. With increasing depth comes increasing complexity due to changes in physiologic responses as the body is exposed to changing stressors.

The pathophysiology of all metabolically active gases (oxygen, carbon dioxide, carbon monoxide) depends on the gas's partial pressure, not on its percentage in the breathing mixture. For example, at 1 atm, a mixture containing 5% oxygen is not compatible with life, but at 5 atm this mixture is perfectly adequate. Just as life-sustaining gases need to be adequately controlled, so do gases considered to be contaminants. A small fraction of contaminants at sea level can cause significant levels of toxicity at high pressure because the partial pressures increase with depth, as described by Dalton's law (see **Gas Laws**, above). The current US Navy standards for the purity of air, oxygen, nitrogen, and helium are found in the *US Navy Diving Manual*.[4] Breathing gases that do not meet these standards are not to be used for diving. This section deals with the pathological conditions associated with commonly

used breathing gases and some common contaminants (carbon dioxide, carbon monoxide).

Oxygen Toxicity

Oxygen, at its most fundamental requirement for life, is used for cellular respiration and the production of adenosine triphosphate, the body's energy currency. However, it has long been known that breathing high partial pressures of oxygen (greater than that experienced in atmospheric air at sea level) can lead to toxicity issues that particularly affect the lungs, central nervous system (CNS), and eyes. The risk is dose dependent. Effects can be seen even on shorter dives and should be a serious consideration for longer exposures such as during saturation dives and hyperbaric oxygen therapy.

Pulmonary Oxygen Toxicity

Pulmonary oxygen toxicity was first described by Lorrain-Smith in 1899 as the result of breathing pure oxygen with a partial pressure of oxygen (Po_2) of 1 ata at sea level for a prolonged time.[16] Clinically, the symptoms of pulmonary oxygen toxicity follow the tracheobronchial tree and progress to the interstitial lung tissue. Patients typically first report the symptoms of a mild "tickle" in their throat and mild cough. This is followed by tracheal and substernal burning with cough on deep inspiration. Persistence of high dose oxygen can eventually lead to dyspnea and death. Even though the patient may be breathing pure oxygen, the cause of death is asphyxia. A measurable sign of pulmonary oxygen toxicity is a decrease in vital capacity (VC), with increasing loss of inspiratory capacity and increasing residual volume. The pathophysiology can be divided into two phases. The first is an exudative phase (< 5 days) and includes an initial general inflammatory response that results in endothelial damage, inflammatory cell infiltration, exudative fluid leakage, hemorrhage, and consolidation. This is followed by the second, proliferative phase (> 5 days) characterized by alveolar type 2 cell and fibroblast proliferation, alveolar membrane thickening, and eventual fibrosis of interstitial lung tissue. The first phase is considered reversible, while the second phase is not.[17]

It has also been shown that there is an association between CNS and pulmonary oxygen toxicity at higher partial pressures of oxygen (> 2 ata) such as those that are known to cause CNS toxicity.[18] The injury pattern, however, is different than that seen with the classic presentation of pulmonary toxicity, which can be induced by lower partial pressures of oxygen (1 ata). Studies in rats have shown that oxygen-induced seizures are associated with pulmonary changes consistent with hydrostatic injury.[19] Mechanisms proposed include an increased output of the sympathetic nervous system in response to increased oxygen tensions, resulting in hemodynamic changes that lead to pulmonary edema and protein leakage.

Several markers for monitoring pulmonary oxygen toxicity have been proposed, most notably, the reduction of lung VC. This effect can be estimated by the unit pulmonary toxicity dose (UPTD),[20,21] which is based on the experimental measurement of changes in VC in human subjects. One UPTD is the degree of pulmonary decrement produced by breathing 100% oxygen continuously at 1 ata for 1 minute. A common benchmark is 1,425 UPTDs, which produces a VC decrease of 10%. This concept assumes no decrement in lung function when breathing oxygen at a partial pressure of 0.5 ata or less. The results of such calculations should be considered as an estimate of pulmonary effects, because there are large individual differences, but it is useful as a tool to limit prolonged oxygen breathing at partial pressures higher than 0.5 atm. Likewise, actual measurements of VC can vary between subjects, and results must be interpreted in context. Recently some experts have recommended using the parameter of diffusing capacity for carbon monoxide as a marker of pulmonary oxygen toxicity.[17]

Although pulmonary oxygen toxicity is an uncommon problem in routine air diving operations, gas content must be carefully planned in saturation diving, where divers are exposed to a Po_2 in the range of 0.4 to 0.5 ata for days to weeks with intermittent exposures to higher levels for diving excursions (see Chapter 17, Military Diving Operations). Care must also be taken to monitor divers using 100% oxygen rebreathers during special operations missions (see Chapter 17, Military Diving Operations). Pulmonary oxygen toxicity can also occur in prolonged recompression therapy for DCS or AGE, as well as during hyperbaric oxygen therapy. Therapy will often be planned to give at least 1 to 2 days' rest for each 5 to 6 days of treatment. Shallower depths may also be used to limit the oxygen exposure. Tradeoffs are made between benefits of high partial pressure oxygen used to promote healing versus the pulmonary damage incurred.

Central Nervous System Oxygen Toxicity

CNS oxygen toxicity, discovered by Paul Bert in 1878, has been referred to as acute oxygen poisoning because of its rapid onset. CNS toxicity can occur while breathing oxygen under pressure at a Po_2 of 1.6 ata but typically requires pressures higher than 2.0 ata

Po_2. There can be significant individual variability in susceptibility, as discussed below.

The most significant and potentially troublesome symptom of CNS oxygen toxicity, particularly for the diver, is the tonic-clonic convulsive seizure. Other symptoms of CNS toxicity can include facial muscular twitching, nausea, vertigo, tunnel vision, hearing changes, anxiety, and behavioral changes, but these symptoms do not usually precede seizures. Occasionally patients have reported aura, but typically there is no warning prior to the onset of seizure. There is some evidence in rat models for increased respiratory rates preceding seizures,[22] but anecdotally, this has not been a reliable predictor. The treatment for CNS toxicity is to remove the patient from the high-level oxygen source and prevent self-injury. Rarely, a convulsion may occur within minutes after oxygen breathing stops. This is known as the "off effect" and has no accepted explanation, but it likely represents the culmination of a process that began during oxygen breathing.

Because oxygen toxicity is dose-related, oxygen can safely be used as long as time and depth are limited. This allows the use of oxygen at greater than 1 ata partial pressure to reduce inert gas absorption at depth, both to accelerate inert gas elimination during decompression and for recompression therapy. The latent period prior to the onset of symptoms can be extended by periodically breathing a gas with a reduced oxygen partial pressure. Therefore, treatment tables at 60 fsw (18 msw) utilize oxygen breathing periods of 20 minutes, separated by 5-minute air breaks (see Chapter 17, Military Diving Operations).

A number of factors affect sensitivity to oxygen toxicity, including extreme variation among individuals and within the same individual from day to day. There is no guarantee that someone who did not have an oxygen convulsion today will not have one tomorrow. Immersion and exercise decrease the latent period, which increases the risk for a working diver compared with that for a diver at rest in a dry hyperbaric chamber.[23] Increased inspired or arterial carbon dioxide decreases the latency,[24] and individuals prone to retain carbon dioxide may be at greater risk. Modern underwater breathing apparatuses have less breathing resistance and dead space than earlier equipment, but heavy work at greater depth may cause carbon dioxide retention and potentiate oxygen toxicity. Increased gas density also decreases ventilation and can lead to retention of carbon dioxide.

The mechanism by which oxygen causes these pulmonary and CNS derangements is still not completely understood. The most accepted explanation is that oxidative stress mediated by reactive oxygen species, such as superoxide and hydrogen peroxide, act through intermediate reactions, and may interfere with cellular metabolism and signaling. Nitric oxide is of particular interest as an intermediate reactant; it can form peroxynitrite from its reaction with superoxide.[25] Plasma membrane is a significant site of free radical interaction, causing disruption by lipid peroxidation, amino acid oxidation, and protein crosslinking.[26]

Other well-known effects of oxygen include vasoconstriction-induced reduction in peripheral blood flow, decreased carbon dioxide carrying capacity of hemoglobin, and increased red blood cell fragility, but these subtle changes do not generally cause concern. Some evidence suggests that high oxygen tensions can also cause DNA strand breaks, but this damage is short lived as repair mechanisms are activated, and no mutagenic effects have been seen.[27]

Ocular Oxygen Toxicity

Though less common, the deleterious effects of oxygen on the eye tend to be cumulative and occur over a prolonged period, similar to pulmonary oxygen toxicity. This can occur in hospitalized patients who receive 100% oxygen for prolonged periods or in divers. Additionally, the condition known as retrolental fibroplasia, a disease of the growing eyes of a premature newborn exposed to elevated levels of inspired oxygen, causes a disruption of normal retinal growth patterns. In adults the common ocular manifestations are decreased peripheral vision and myopia. Both conditions tend to resolve after cessation of high dose oxygen.[28]

Carbon Dioxide Toxicity

Carbon dioxide is a naturally occurring gas produced by cellular respiration. It can have significant effects on divers, if not adequately controlled. These effects include hyperventilation, dyspnea, tachycardia, headache, lightheadedness, and dizziness, progressing to mental confusion and unconsciousness (Table 18-2). Carbon dioxide was once used as an inhalation anesthetic (beginning in 1824), but was surpassed by newer medications with fewer side effects.[29] As with other gases, the effects are dependent on the partial pressure of the gas, not the percentage. Increased carbon dioxide levels and toxicity can be caused by the following conditions:

- increased carbon dioxide in the breathing gas due to compression of contaminated gas in an open-circuit breathing apparatus or failure of the carbon dioxide absorbent in a closed-circuit apparatus;

TABLE 18-2

ACUTE EFFECTS OF INCREASED INSPIRED CARBON DIOXIDE

Partial Pressure of CO_2 (ata)	Effect
0.0003	None, normal atmospheric partial pressure of CO_2 at sea level
0.0003–0.04	None, exposures less than 8 hours
0.04–0.06	Mild hyperventilation and confusion, headache
0.06–0.10	Moderate dyspnea and confusion
>0.10	Unconsciousness, muscle twitching

Data source: Lambertsen CJ. *Carbon Dioxide Tolerance and Toxicity.* Philadelphia, PA; University of Philadelphia Institute for Environmental Medicine; 1971.

- increased respiratory dead space owing to poor equipment design or inadequate ventilation of the chamber or helmet;
- voluntary hypoventilation (when divers hold their breath after inhalation or "skip-breathe" to increase the duration of the open-circuit gas supply);
- increased partial pressure of oxygen, which decreases the ventilatory response to carbon dioxide; or
- increased breathing resistance, either intrinsic (in a diver's lungs) or extrinsic (due to breathing equipment); the latter is more common than the former. Added breathing resistance decreases ventilatory response to elevated carbon dioxide.

Carbon Monoxide Toxicity

Carbon monoxide sources in the environment are largely due to incomplete combustion of hydrocarbons (fuels) or other carbon compounds. Contamination of compressed air is rare but dangerous when it occurs. The most common source of divers' gas contamination is from an air compressor whose intake is near the exhaust of an internal combustion engine, even the compressor's own power source. Compressors lubricated with oil are also a potential source of carbon monoxide if the oil does not meet specifications or the compressor is operating at elevated temperatures. Physiologically, a small amount of carbon monoxide is produced during the metabolism of hemoglobin, and habitual smokers exhale even larger quantities for up to 24 hours after their last cigarette.[30] Any amount of contamination is concerning at increased ambient pressures, particularly when divers are exposed to prolonged bottom times such as in saturation diving.

Contamination of breathing air with carbon monoxide from any source prevents normal saturation of hemoglobin with oxygen and, through a cascade of events, can cause long-term neurologic sequelae. Exhibit 18-1 lists common symptoms of acute carbon monoxide toxicity. Many texts contain a table that correlates symptoms to measured levels of carboxyhemoglobin, but this table has been shown to be an outdated tool in the management of poisoned patients due to the poor correlation of carboxyhemoglobin levels with actual patient presentations.[31] The more important finding is to link the triad of exposure, symptoms, and abnormal elevation of carboxyhemoglobin to make decisions about treatment.

The acute toxic effects of carbon monoxide at depth depend on its partial pressure, and the symptoms are the same as at sea level. Because carbon monoxide binds to myoglobin as well as to hemoglobin, carboxyhemoglobin concentrations do not always correlate with symptomatology. The length of exposure, inspired carbon monoxide partial pressure, and physical activity are also important. A short exposure of a resting person to a high level of carbon monoxide may produce a lower blood level than a long exposure of a working person to a relatively low inspired carbon monoxide concentration.

Because carbon monoxide and oxygen compete for binding sites on hemoglobin, oxygen breathing, particularly at increased partial pressure, is considered to be an effective treatment for carbon monoxide poisoning. It has long been thought that breathing 100% oxygen at sea level (Po_2 of 1 ata) decreases the

EXHIBIT 18-1

COMMON SYMPTOMS OF ACUTE CARBON MONOXIDE TOXICITY

- headache
- dizziness
- nausea/vomiting
- confusion
- fatigue/weakness
- vision changes
- chest pain
- shortness of breath
- loss of consciousness
- death

morbidity and mortality associated with carbon monoxide poisoning. This led to the idea that hyperbaric treatment with oxygen breathing (Po_2 > 1 ata) might further improve outcomes. Several studies (of varying quality) have been conducted, some showing benefit, others showing no difference, in breathing 100% oxygen at sea level versus hyperbaric oxygen therapy. A recent review concluded that there is insufficient evidence to routinely recommend hyperbaric oxygen therapy for the treatment of carbon monoxide poisoning.[32] Some experts, however, while acknowledging the limitations of previous studies, still recommend hyperbaric oxygen therapy for poisoned patients with loss of consciousness, ischemic cardiac changes, neurological deficits, significant metabolic acidosis, or carboxyhemoglobin over 25%.[33]

Nitrogen Narcosis

Nitrogen narcosis, "the rapture of the deep," is the progressive intoxication that develops as a diver descends and the partial pressure of nitrogen increases. Depending on individual sensitivity, common lore states that each 1- to 2-ata increase in air pressure is equivalent to one martini, the so-called "martini law." While perhaps not absolutely correct, this analogy does reflect the subtle changes that begin at 50 to 100 fsw (15–30 msw) while breathing compressed air that have been described as feeling similar to alcohol intoxication.[34] Most commonly there is a general decrease of cognitive function and memory accompanied by motor impairment that worsens as depth increases. This continues to about 350 fsw (105 msw), where most divers lose consciousness.[35]

Factors that exacerbate nitrogen narcosis are inexperience, anxiety, alcohol, fatigue, and increased inspired carbon dioxide.[36] Ameliorating factors are suspected to be experience, mental discipline, and fixation on a task, but these are anecdotal at best. The evidence for adaptation to narcosis with frequent exposure is lacking and controversial, with some studies showing the contrary.[36] Because narcosis is a threat to diver safety, most agencies limit air diving with a scuba to 130 fsw (40 msw), and with hard-hat surface-supplied air diving to near 165 to 180 fsw (50–55 msw).

The mechanism of nitrogen narcosis, which is thought to be similar to anesthesia, is still under study. Anesthetic potency shows some relationship to lipid solubility, which was formerly thought to cause cell membranes to swell, thereby influencing ion transport. This is consistent with the lack of narcosis from helium, with its low solubility and low surface tension. More recent trends under consideration, supported by advanced laboratory techniques, have focused on a membrane protein binding theory, which may better explain discrepancies in the lipid solubility model.[37] Overall consensus is that there are likely multiple mechanisms that contribute to anesthesia.

Helium

Much experimentation has been done on breathing gas alternatives since the dawn of diving. Finding gases that benefit the diver while incurring the least amount of side effects has been the goal. Although the noble gases (group 18 in the periodic table of elements) are chemically inert, they can be physiologically active.

Substitution of helium for nitrogen in breathing gas has four major effects. The first is the effect on the voice due to decreased density of helium gas passing by the vocal cords. The second is the absence of narcosis. The third is decreased work of breathing, owing to the lower density. This, and the lack of narcosis, make helium particularly useful at depths greater than 150 fsw (45 msw). The fourth effect is loss of heat through the skin and the lungs, owing to helium's high heat capacity and conductivity. Both the skin and the breathing gas must be heated during prolonged helium–oxygen diving to prevent hypothermia. Hot water is usually used for this purpose. Hypothermia can also occur in a dry, helium–oxygen filled chamber if the temperature is not raised above the normal comfort level for air. The required temperature increases with increasing gas density. A full discussion of these effects can be found in the section on saturation diving in Chapter 17, Military Diving Operations.

Although helium is the gas of choice for deep dives, a deleterious effect called high-pressure nervous syndrome (HPNS) begins at depths typically greater than 600 fsw (180 msw). HPNS is characterized by CNS hyperexcitability, including tremors, poor sleep (with microsleep), loss of appetite, decreased manual dexterity, and impaired but stable cognitive function.[38] Limited accounts of psychotic symptoms have been reported at depths greater than 2,000 fsw (600 msw).[39] Initially, HPNS was believed to be caused by helium ("helium tremors"), but it is unclear if pressure is more likely the responsible agent.

Several studies in the 1970s involving deep dives showed that the onset of HPNS was dependent on rate of compression and overall depth. Protocols that slowed descent proved beneficial. Additionally, because the excitatory effects of pressure and the narcotic effects of nitrogen are partially counteractive, combinations were used to ameliorate each effect. This phenomenon, known as the pressure reversal of anesthesia, was first discovered in tadpole experiments

with ethanol as the narcotic. It is achieved by adding nitrogen to helium–oxygen mixtures when diving to greater depths.[40]

Hydrogen

Interest in hydrogen diving has been ongoing since engineering advances in handling hydrogen–oxygen mixtures reduced the danger of explosion. These techniques take advantage of the fact that hydrogen cannot ignite if mixed with less than 5.5% oxygen. Hydrogen is inexpensive, readily available, and less narcotic and less dense than nitrogen. The decompression properties of hydrogen appear to be between those of helium and nitrogen. Up to 20 ata of hydrogen have been used to reduce both HPNS and breathing resistance at depths greater than 2,000 fsw (610 msw), but hydrogen narcosis, perhaps exacerbated by HPNS, appears to be a limiting factor.[41]

Other Inert Gases

Of the other noble gases, neon has been used in deep diving experiments because of its lack of narcotic effect. However, it is not used for diving operations because it is expensive and has a higher density than helium (or hydrogen), which causes greater respiratory work. Argon, xenon, and krypton are narcotic at 1 ata and therefore not appropriate for diving. Because of its high insulating properties, argon is sometimes used to inflate dry diving suits.

DECOMPRESSION SICKNESS

DCS refers to the overt illness that follows a reduction in environmental pressure with the development of endogenous gas bubbles. The condition is distinguished from AGE, in which bubbles originate from the lungs or external sources and enter the vasculature through disruptions in the pulmonary membranes. DCS occurs in diving, caisson work, rapid ascent to high altitude, and following hyperbaric chamber work. DCS can occur at any time following the start of decompression and can be subtle or catastrophic. The initiating cause is the formation of bubbles of the inert gases dissolved in the tissues. Diagnosis can be difficult because the signs and symptoms of DCS resemble not only non-diving diseases but also AGE. Symptoms and signs may disappear spontaneously but return unpredictably hours later. DCS should be the top-most diagnosis in the physician's mind when a patient presents with complaints following any dive.

DCS is traditionally classified into two types: Type I, which is minor, includes only limb or joint pain, itch, skin rash, or localized swelling; and Type II, which is serious, includes presentations with neurological and pulmonary symptoms or signs (see also Chapter 16, Aerospace Medicine). The term "decompression illness" is used for any diving accident involving pressure reduction, including Type I DCS, Type II DCS, and AGE. In certain cases, it may be impossible to distinguish between AGE and DCS with signs of cerebral dysfunction, or a combination of both. Table 18-3 provides some guidance, but differentiation is often not possible. Clinically, this is not a significant issue; the *US Navy Diving Manual* specifies the same symptom-based therapy, including saturation therapy, for both AGE and severe neurological DCS: recompression with oxygen to 60 ft (18 m), or to deeper depths with air or a breathing gas with an increased oxygen

TABLE 18-3

COMPARISON OF DECOMPRESSION SICKNESS AND ARTERIAL GAS EMBOLISM

Factor	Decompression Sickness (DCS)	Arterial Gas Embolism (AGE)
Occurrence	A dive of sufficient depth and duration to cause significant inert gas absorption	Any dive
Onset	Immediate or delayed	Immediate
Cause	Inert gas bubbles in tissue, veins, or arteries	Bubbles in arteries only as a result of pulmonary barotrauma
Bubbles	From dissolved gas	From alveolar gas
Usual first symptom	Localized pain or numbness	Unconsciousness or paralysis
Neurological signs	Usually bilateral	Usually unilateral
Prognosis	Mild to serious	Serious

content (eg, 50% oxygen–50% nitrogen).[4] The depth and duration of therapy are determined by the clinical progression.

For treatment purposes, the most important point is how these symptoms respond to therapy, not the initial classification. In fact, as therapy progresses, the diagnosis may change. For example, a diver with severe pain may not notice local weakness or sensory loss until recompression relieves the pain. The pain may also mask the signs from an examiner. Immediate recompression of patients with serious signs and symptoms should take precedence over a detailed physical examination that may reveal less obvious signs. Additional information on the treatment of DCS and AGE for flight or diving environments can be found in Chapter 16, Aerospace Medicine, and Chapter 17, Military Diving Operations.

Inert Gas Exchange

When an inert gas is breathed at elevated pressure, its partial pressure in the lungs initially exceeds that in the tissues. As the time at pressure lengthens, the inert gas is progressively absorbed by the tissues until its partial pressure there equals that in the lungs. The principal factors governing the rate of gas absorption by tissue are perfusion and solubility. Lipid tissues, such as fat, with high inert gas solubility and poor perfusion, absorb (and eliminate) inert gas much more slowly than low solubility aqueous tissues, such as muscles, that are well perfused.[42] Diffusion is not as important as perfusion, but it can influence gas exchange any time areas of tissue have different inert gas tensions. For example, diffusion shunts can occur between adjacent arterial and venous vessels, resulting in slower inert gas exchange in a tissue than would be expected on the basis of perfusion alone.

Bubble Formation

Bubble formation that follows decompression isolates inert gas from the circulation and reduces the effectiveness of perfusion in eliminating the inert gas in the vicinity of the bubble. An effective method for accelerating the elimination of an inert gas from tissues or bubbles is to increase the partial pressure of oxygen in the inspired gas, which increases the difference between inert gas in the lungs and in the tissue.[42]

In experiments with animals performed during the 1870s, Paul Bert[43] of France demonstrated that the most severe forms of DCS are caused by bubbles in the blood and tissues. Bubbles are less obvious in the milder forms of DCS, which led to the suggestion of other etiologies, but none of these theories has been sustained. The presence of bubbles, after even very shallow or short dives, can be detected using ultrasound devices.[44]

In both living and nonliving systems, a primary factor that determines whether a bubble will appear is the level of supersaturation, or the sum of the partial pressures of all vapors and dissolved gases, minus the local absolute pressure. The level of supersaturation that leads to bubble formation is a clue to how bubbles form. Supersaturation of gases on the order of 100 to 1,000 atm in nonliving systems is evidence for de novo nucleation (ie, the formation of bubbles where no gas phase previously existed).[42,45] During physiological decompression, however, supersaturation rarely exceeds several atmospheres, and the lowest supersaturation at which DCS occurs is about 0.5 atm.[13,46] Under these conditions, bubbles probably expand from preexisting gaseous micronuclei, or gas nuclei, which exist in all aqueous fluids. It is difficult to understand the origin of gas nuclei in closed living systems, but some are probably mechanically generated by the de novo nucleation resulting from both viscous adhesion in tissue and shear forces during the relative motion of articular surfaces.[42] Such motion causes the local pressure to transiently decrease to hundreds of negative atmospheres and creates vaporous bubbles that make audible sounds as they collapse ("cracking" joints).[47] Stable gas bubbles also form as result of this process. The population of some gas nuclei in the body appears to normally be in a state of dynamic equilibrium, wherein their creation by mechanically induced nucleation is balanced by their elimination due to surface tension.

Pathophysiology of Decompression Sickness

DCS results from a reduction in ambient pressure with the subsequent formation of stable bubbles. Despite the common occurrence of bubbles in the circulatory system, bubbles probably do not originate in blood but form extravascularly and seed the microcirculation as they expand. These bubbles grow by the inward diffusion of nitrogen as they are carried in the venous blood to the heart and lungs. The pulmonary circulation is usually effective in filtering these bubbles. However, if the volume of gas becomes overwhelming, as can occur in accidental ascent from great depth, pulmonary edema and cardiac failure can result, leading to death by asphyxia.[48,49]

Venous bubbles have the potential for reaching the arterial circulation by passing through the pulmonary circulation or bypassing the lungs altogether through

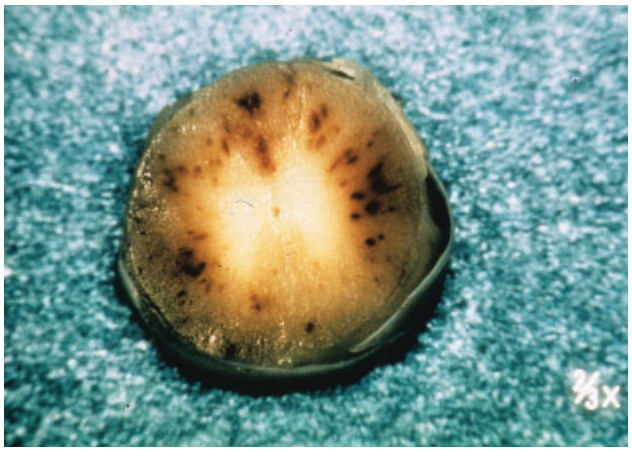

Figure 18-4. This gross cross-section of a spinal cord shows hemorrhages in the white matter that are typical of decompression sickness.

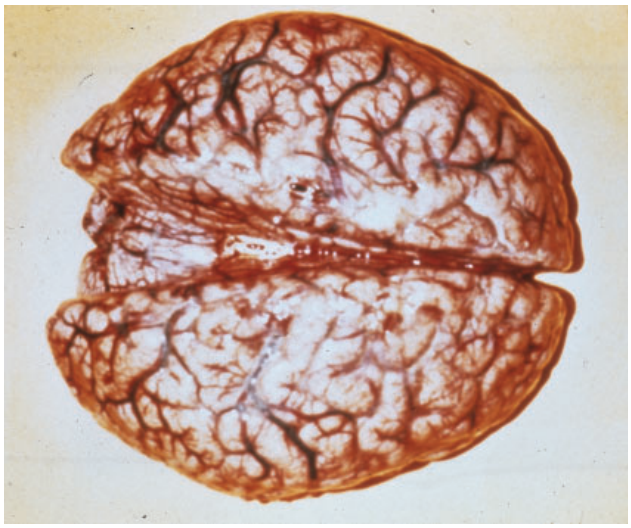

Figure 18-5. Brain from a patient who died of severe decompression sickness, showing bubbles in the surface vasculature.

arteriovenous shunts or possibly a patent foramen ovale (PFO). Large numbers of emboli in the arterial circulation can arise due to pulmonary barotrauma of ascent. While this gas can have serious consequences if carried to the brain, the situation is worse if the barotrauma occurs at the end of a dive when the tissues contain excess inert gas. Bubbles that enter the arterial circulation can expand if they reach supersaturated tissue. This may explain a devastating form of DCS that involves both the brain and spinal cord after relatively shallow dives that end with pulmonary barotrauma (Figures 18-4 and 18-5).

Bubbles have both mechanical and biochemical effects, which may be extravascular or intravascular (arterial and venous). Extravascular bubbles can compress or stretch tissue and nerves. Intravascularly, they can cause embolic obstruction, platelet aggregation, ischemia, edema, hemorrhage, and tissue death (Figure 18-6).[50,51] Endothelial cell membranes are stripped by the passage of bubbles. Bubbles may block lymphatic channels, causing tissue edema. The most serious of these events are rare, except in cases of severe DCS or AGE, and mild cases are the rule when modern dive procedures are used. The human body appears to tolerate a certain volume of bubbles with no symptoms or only mild symptoms.[44]

The biochemical effects of bubbles relate to their surface active properties, which cause enzyme activation and protein denaturation leading to thrombosis and complement activation.[50,52] Together, the mechanical and biochemical effects of bubbles can increase blood viscosity, postcapillary resistance, transcapillary fluid loss, and hemoconcentration.[51,53] These interactions can produce reperfusion injury, which occurs when toxic oxygen species are generated after circulation to an ischemic area of the brain has been reestablished.[54] Reperfusion injury may explain a poor response to hyperbaric therapy or relapse after initially successful treatment of DCS or AGE. Relapse may also occur due to the aggregation of leukocytes on damaged endothelium.

Clinical Presentation

Whether provoked by diving or by rapid ascent to altitude, DCS is a protean disease that can present with subtle symptoms and no signs. These may or may not worsen over minutes to hours. DCS may also present as a sudden catastrophic event with multiple symptoms, signs, and multiple organ involvement. Table 18-4 lists the signs and symptoms reported by Vann and colleagues in 2,346 cases of DCS in recreational divers reported to the Divers Alert Network from 1998 to 2004.[13] The five most frequent signs and symptoms reported were pain (68.0%); numbness or paresthesia (63.4%); constitutional symptoms including headache, lightheadedness, fatigue, malaise, nausea, vomiting, or anorexia (40.8%); dizziness or vertigo (19.4%); and motor weakness (18.7%).[13] In an older study by Rivera of 933 military and civilian divers with DCS, the five most frequent signs or symptoms were pain (91.8%), numbness or paresthesia (21.2%), motor weakness (20.6%), rash (14.9%), and dizziness or vertigo (8.5%).[55]

Comparison of the two studies indicates that the number of DCS cases involving pain was less in the Vann series, and that the number of cases with neuro-

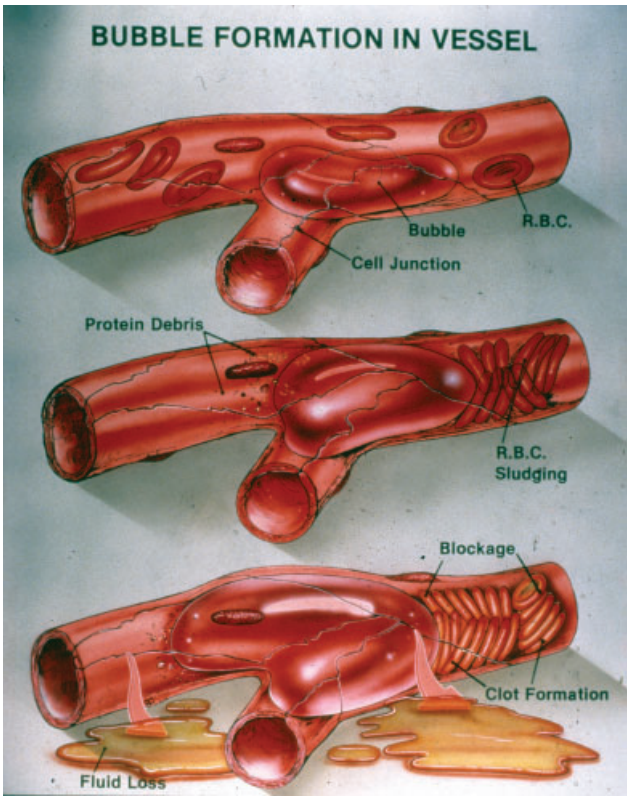

Figure 18-6. Bubble formation in a blood vessel. (**a**) The lodging of a bubble at a vascular junction, with (**b**) sludging of the red blood cells (RBCs) and formation of protein debris, and (**c**) blood clot and extravascular fluid loss. Bubbles in blood vessels are typically not spherical.

logical symptoms (numbness/paresthesia, dizziness/vertigo, and motor weakness) was greater. The reasons for these differences are not clear. In Rivera's series, most of the divers were military and perhaps reported their symptoms (and were subsequently treated) earlier, thereby preventing more serious symptoms.[55] Military divers are also under stricter control regarding adherence to decompression schedules and may be more conservative than recreational divers in selecting decompression schedules. Recreational divers also tend to make more repetitive dives, which could predispose them to more serious problems.

A minor DCS symptom is pruritus, known as "skin bends," which is most common in dry chamber dives. Itching may be accompanied by an urticarial rash and is usually localized in well-perfused, exposed skin such as the ears, although it can occur anywhere on the body. Skin bends is generally transient and does not require treatment, but the diver should be observed for at least an hour for the onset of more serious symptoms. A severe form of rash called "marbling" or "mottling" (cutis marmorata) appears as a pale area

TABLE 18-4

FREQUENCY OF SIGNS AND SYMPTOMS OF DECOMPRESSION ILLNESS

Sign or Symptom	Percentage of Patients with a Given Sign or Symptom (n=2,346)
Pain	68.0
Numbness/paresthesia	63.4
Constitutional*	40.8
Dizziness/vertigo	19.4
Motor weakness	18.7
Cutaneous	9.5
Altered mental status	7.9
Decreased coordination	7.9
Dyspnea or cough	5.6
Bladder or bowel incontinence	2.8
Decreased consciousness	1.8
Lymphatic	1.8

*Constitutional symptoms included headache, lightheadedness, inappropriate fatigue, malaise, nausea, vomiting, or anorexia.
Data source: Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. Lancet. 2010;377:153–164.

with cyanotic mottling. The area may enlarge, become hyperemic, and show swelling. While uncommon, cutis marmorata is often a harbinger of more severe DCS and warrants immediate treatment.

Lymphatic obstruction appears as painless local edea, usually on the trunk. If it is severe the skin may have a "pigskin" appearance. Unilateral breast swelling and swelling of specific muscles may occur. In these cases, recompression may help alleviate pain. Obstruction of the lymphatics usually disappears in a few days with or without treatment.

Joint pain and numbness are the most common DCS symptoms. The onset of pain may be gradual or abrupt, and its nature mild, severe, paroxysmal, aching, or boring. Severe cases may be associated with a cold sweat. Classically the pain is described as a deep, dull ache isolated to a particular joint. It is present at rest and usually not affected by movement. Occasionally, the pain can be reduced by the application of local pressure with a sphygmomanometer cuff. A painful joint will sometimes have an associated area of numbness or altered sensation that may reflect a peripheral nerve lesion, but this is difficult to diagnose.

Divers with neurological symptoms should be recompressed as soon as possible to achieve the most complete relief and to forestall the onset of more

severe problems. Abdominal or thoracic pain occurring in a circumferential pattern (girdle pain) signals the onset of spinal cord DCS. The pain may disappear after a short time but be followed within an hour by severe symptoms, usually paralysis of the lower extremities.

In diving, DCS occurs in the upper extremities two to three times more often than in the lower extremities. The opposite is true for caisson workers, saturation divers, and workers in ground-based altitude exposures simulating astronaut extravehicular activity. The reason for this difference may be due to the weight-bearing stresses of gravity in the latter cases. In divers, these stresses are relieved by immersion in water, which could reduce the formation and expansion of bubbles in the legs. CNS symptoms vary with the site of the tissue insult and are similar to those found in CNS disease from other causes. There is a wide range of symptoms (see Table 18-4). As with AGE and neurological symptoms, any CNS symptom following diving is serious, and the patient should be recompressed as soon as possible for best results.

Symptoms of DCS generally appear shortly after the diver surfaces. In a series of several thousand air dives compiled by the US Navy, 42% of divers with DCS developed symptoms within the first hour after surfacing, 63% within 3 hours, 83% within 8 hours, and 98% within 24 hours.[4] Occasionally, the onset time is longer, but few symptoms appear after more than 24 hours. Even if a symptom does not occur until 24 hours or longer after diving, the patient should be presumed to have DCS until it is ruled out. Symptoms with very long onset times sometimes respond to recompression and should not simply be dismissed as not diving related.

Rarely, symptoms of DCS begin in the water during decompression, and usually only after long, deep dives. In water DCS is most likely to occur at the shallower decompression stops near the surface. The primary symptom will usually be joint pain, but more serious neurologic symptoms may develop. Treatment of in-water DCS depends on the type of diving equipment used. Detailed information on the treatment of DCS can be found later in this chapter, as well as in the *US Navy Diving Manual*.

Aseptic Bone Necrosis

Aseptic bone necrosis, also known as dysbaric osteonecrosis, is a delayed consequence of chronic exposure to a hyperbaric environment that may not be evident until years after exposure (Figure 18-7). The exact mechanism of this condition is unknown, but incidence appears related to the length of the diver's career and magnitude of hyperbaric exposure.[56] Studies of commercial divers indicate the prevalence of bone necrosis is less than 5%.[57,58] Lesions usually occur at the proximal ends of the humerus, femur, and tibia, adjacent to a joint or midshaft of the humerus and femur.[56] Often the lesions never cause symptoms and are identified only by radiography. No treatment is required for lesions, such as shaft lesions, that do not cause symptoms. The most serious lesions are juxtaarticular. If these areas become necrotic and collapse, it may be necessary to replace the joint with a prosthesis.

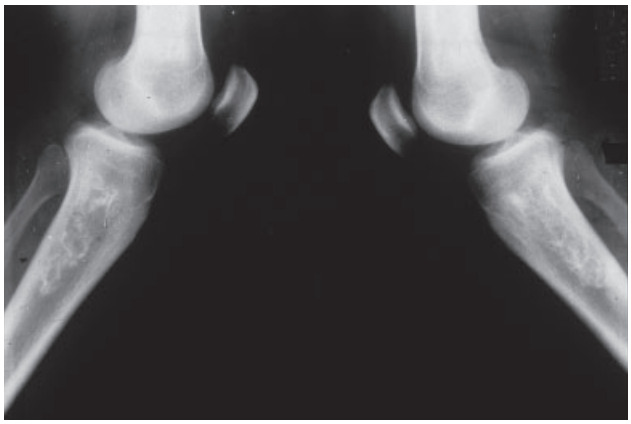

Figure 18-7. A composite radiograph shows an abnormal pattern of calcification in both proximal tibias as a result of aseptic bone necrosis, a sequelae of decompression sickness.

Diving at Altitude and Altitude Exposure after Diving

There is increased danger of DCS whenever the barometric pressure is reduced soon after, or in association with, diving. Circumstances can include diving at altitude, mountain travel after diving, and flying after diving. DCS can occur independently of diving during altitude exposures above 18,000 ft (5,486 m; a barometric pressure of 0.5 ata).[42] Nitrogen dissolved in the tissues at sea level has a tension of about 0.79 atm and leaves solution to form bubbles at altitude. Flying after diving increases the risk of DCS because additional nitrogen remains in the tissues after a dive. To reduce the DCS risk from flying too soon after diving, divers are advised to wait long enough at sea level until nitrogen dissolved in their tissues is eliminated harmlessly through the lungs. Table 9-6 of the *US Navy Diving Manual* lists preflight surface intervals required before flying is considered safe.[4] These surface intervals range from 0 to 21 hours for a commercial flight, depending on the severity of the previous diving exposure. The US Air Force requires a 24-hour wait after any diving before flight.

Diving at altitude also increases the risk of DCS. There are two main factors. First, because the bodies of sea-level residents are in equilibrium with the 0.79 atm of nitrogen in atmospheric air, rapid ascent to an altitude of 18,000 ft (5,486 m), where the atmospheric nitrogen is only 0.4 atm, causes a supersaturation of some 0.39 atm. Supersaturated nitrogen dissipates over about 24 hours and must be accounted for before then. Dives conducted within 12 hours of initial ascent to altitude are considered repetitive dives.[4] Table 9-5 of the *US Navy Diving Manual* lists the repetitive group designator associated with altitudes up to 10,000 ft (3,048 m).[4]

Second, after equilibration is complete, the no-decompression times are still shorter than normal because bubbles grow larger at reduced barometric pressure than at sea level. Therefore, a dive conducted at an altitude of 5,000 ft (1,524 m) to a depth of 60 fsw (18 msw) is equivalent to an 80 fsw (24 msw) dive at sea level.[4] Table 9-4 of the *US Navy Diving Manual* contains equivalent sea level depths for diving at altitudes up to 10,000 ft (3,048 m).[4]

Factors Affecting Individual Susceptibility

Several factors are known or suspected to affect susceptibility to DCS, among them inadequate decompression; exercise and body temperature as they affect perfusion; and individual characteristics such as age, obesity, and dehydration.[59] The most common cause of DCS is inadequate decompression, which can result from ignorance or misapplication of the decompression schedules, panic, equipment failure, and other causes. Omitted decompression does not necessarily result in DCS, and DCS may occur even if the prescribed decompression is followed exactly. DCS has also occurred after dives that ordinarily do not require decompression stops. The severity of DCS does not correlate well with the amount of omitted decompression. Neurological DCS is more common after deeper dives than long, shallow dives.[59] Repetitive diving (more than one dive in 12–24 h) may increase the risk of DCS, presumably due to the increased total load of absorbed nitrogen and errors in computing the necessary decompression schedule.

Any physiological or environmental factor that changes local perfusion (eg, exercise, body temperature) also changes regional inert gas exchange. A change in perfusion may have a different effect on DCS risk depending on the phase of the dive in which it occurs (at pressure, during decompression, or after decompression). For example, a factor that increases perfusion while a diver is at depth will increase inert gas uptake, but this same factor will increase its elimination if present during decompression. Factors that increase peripheral perfusion include exercise, immersion, and heat, while cold and dehydration decrease perfusion. Immersion and exercise at pressure have been demonstrated to increase gas uptake and DCS risk.[59] When multiple factors exist that change during a dive, the results may be difficult to anticipate. Mild exercise during decompression appears beneficial, but DCS may be confounded by exercise at pressure. The most disadvantageous situation probably occurs during a decompression dive, wherein the diver works at pressure and rests during decompression. In this instance, work at pressure increases the perfusion and warms the diver, while rest during decompression decreases perfusion and causes vasoconstriction from cooling.

Temperature and exercise also have effects unrelated to perfusion. Inert gas solubility decreases as the temperature rises. As a diver warms on the surface after a dive, the inert gas solubility falls and the dissolved gas tension and supersaturation rise. This fact may explain the anecdotal observations that hot showers precipitate DCS.

Exercise may mechanically aggravate the formation and expansion of bubbles by reducing the local absolute pressure in tissue and thus increasing the local supersaturation, which expands existing bubbles or micronuclei and may cause new nuclei to form. Human experimental evidence indicates that exercise after decompression from diving or ascent to altitude increases DCS incidence.[60] Note the distinction between exercise during and exercise after decompression. Exercise during decompression increases the elimination of inert gas by raising perfusion, while exercise after decompression, when bubbles are already present, can initiate or accelerate bubble expansion.

Increasing age increases the risk of DCS.[44,59] The age effect may be related to decreased inert gas exchange efficacy and increased formation of gas nuclei. Obesity may increase the risk of DCS, presumably because inert gas is more soluble in fatty than in aqueous tissue; however, this relationship has not been proven.[61] Similarly, there is no proven difference in DCS susceptibility between genders.[13,62]

Dehydration reduces perfusion and inert gas elimination. Divers with DCS are often found to have elevated hematocrit values, perhaps from dehydration but perhaps also from increased capillary permeability resulting from DCS.[13] Typical causes of dehydration in diving are alcohol consumption, low fluid intake, sweating in hot climates or diving suits, sea sickness, and cold- or immersion-induced diuresis.

Experience with caisson workers shows that the incidence of DCS is greatest during the first few days of repeated exposure to increased pressure.[63] This may result from a depletion of the gas nuclei that appear to be the origin of bubbles. Acclimatization to prevent DCS has been better proven in helium–oxygen diving than in air diving.[59]

Patent Foramen Ovale as a Risk Factor

Recently there has been much interest in the effect a PFO has on the incidence of DCS. A PFO is an atrial septal defect that results from the failure of the fetal foramen ovale to close after birth. Theoretically, venous bubbles may cross a PFO and enter the arterial circulation. However, in most persons the flow across a PFO is from left to right (high pressure to low pressure). In order for bubbles to enter the arterial circulation the shunt must be reversed. Factors thought to increase this flow are straining, lifting, coughing, or any other process that increases right atrial pressure. Immersion itself does not appear to increase right-to-left shunting across a PFO.[51]

The presence of a PFO has been shown to statistically increase the incidence of DCS with neurologic and cutaneous manifestations.[13] The estimated relative risk for serious neurologic DCS due to a PFO ranges from 2.5 to 6.6.[64] However, given that the actual risk of serious neurologic DCS is less than 0.02%, the presence of a PFO imparts a very small absolute risk.[65] Furthermore, a PFO is present in approximately 27% of healthy individuals.[66] Given the vast number of dives that are completed without incident, it seems the overall effect of a PFO is minor.

Due to its prevalence and the small absolute risk of serious neurologic DCS it carries, routine screening for a PFO among divers or diver candidates is not recommended. Divers who do experience serious neurologic DCS may warrant screening. However, it is unclear how to best manage those found to have a PFO, especially because the risks of operatively closing a PFO may outweigh the potential benefit.

PREDICTING AND PREVENTING DECOMPRESSION SICKNESS

Decompression Tables

The most important factors in reducing the risk of DCS are (*a*) limiting the time at depth to reduce inert gas uptake and (*b*) allowing for slow ascent so that inert gas may be harmlessly eliminated through the lungs. Death or permanent disability were common among divers and caisson workers in the 19th century, before this was understood. By the end of the century it was recognized that some type of slow decompression was necessary to prevent injury. Not until after the turn of the 20th century, when J.S. Haldane began studying the problem systematically in England, were decompression schedules similar to those used today constructed.[42] Haldane realized that the manner of decompression used at the time, steady decompression at rates between 1.5 and 5 fsw per minute, was too slow at deeper depths and too rapid at shallow; this rate allowed more inert gas to be absorbed at depth and did not provide enough time for inert gas to be eliminated in shallow water. He devised the now familiar staged decompression schedules, which use progressively longer stops near the surface. Modifications of the Haldane calculation methods are still the basis of many modern decompression schedules.

A decompression table contains multiple decompression schedules for a particular dive depth and bottom time. Each schedule defines the depths and duration of the decompression stops, and tables are available for diving with different inert gases and oxygen concentrations. With current procedures, DCS is a rare event. The DCS incidence when using the US Navy diving tables, which all US military diving operations are required to use, is about 0.1%.[59] The incidence of DCS during recreational diving is about 0.3%.[67] Granted, many of these dives are conducted well within decompression limits and do not represent the rate of DCS using the maximum exposure limit; the incidence of DCS using US Navy diving tables during decompression dives or the maximum no-decompression limit is about 2%.[13] See also Chapter 17, Military Diving Operations.

Probability of Decompression Sickness and Acceptable Risk

Any dive profile might be called "safe" if an individual diver completes it without DCS. But is a dive safe if DCS occurs in 1 of 10 divers, or in 1 of 100? The answer depends on the definition of safety. Many human activities would be impossible if absolute safety—the complete absence of risk—were required. Safety is based on the level of risk a person is willing to accept. Thus, safety is defined as the acceptable risk. The US Navy diving tables assume a risk determined to be acceptable for military diving operations; however, it should be understood that even if the tables are followed correctly, there is still a small risk of DCS.

TREATMENT OF DECOMPRESSION SICKNESS AND ARTERIAL GAS EMBOLISM

As previously discussed, AGE is the result of pulmonary barotrauma that leads to air entering the arterial circulation, while DCS is due to the formation of bubbles in blood and tissues after a reduction in ambient pressure. The immediate goal of therapy is to reduce the volume of the offending bubbles. This may be possible for patients treated shortly after symptom onset, but long delays before treatment are common, and gas bubbles may have caused physical or biochemical damage that persists after the bubbles themselves have resolved. In such situations, therapy to oxygenate poorly perfused tissue or reduce edema can still be beneficial.

The definitive treatment for DCS and AGE is increased atmospheric pressure and 100% oxygen. The additional pressure serves to reduce the size of the bubbles, and oxygen accelerates their resolution by causing nitrogen to diffuse from bubble to blood. On reaching the lungs, excess nitrogen from the blood is exhaled. Pressure (or recompression) was first used in 1909 to treat DCS (then called "caisson disease"), but oxygen was not routinely used during recompression until the 1960s.[35]

The best first aid for AGE or DCS is 100% oxygen delivered by mask. Inspired oxygen percentages near 100% are essential for the greatest effect. Because injured divers are often dehydrated due to illness or post-immersion diuresis, oral rehydration or administration of isotonic, non-glucose-containing intravenous fluids may be considered.[13] However, cases of AGE may be complicated by cerebral edema, and care should be given not to fluid-overload the patient.[4] The patient should be positioned on their back or side on a horizontal plane. In the past, it was advised that the patient be placed in the Trendelenburg position to prevent more gas from entering the cerebral circulation, but this is no longer recommended because the head-down position increases central venous pressure and cerebral venous pressure, and can cause cerebral edema.[68]

There are no proven adjuvant therapies. Aspirin and other platelet inhibitors are no longer recommend due to concerns about possible hemorrhage into neural tissues.[69] The only exception is the use of low-molecular-weight heparin in patients with lower extremity paralysis to prevent deep vein thrombosis. If convulsions are present, benzodiazepines may be considered. While not commonly used, intravenous lidocaine may be helpful in the treatment of AGE with serious neurological symptoms. Lidocaine has been shown to increase cerebral vasodilation and decrease the rise in intracranial arterial pressure.[70] It may also be useful in the treatment of cardiac dysrhythmias; however, no studies have been conducted concerning its use in divers.[13] If used, intravenous lidocaine should be dosed to produce an antiarrhythmic effect.[4] The use of parenteral steroids, once thought to reduce cerebral edema, has been shown to worsen outcomes in animals and is no longer recommended.[4,69,71]

Because there is no definitive test for DCS or AGE, a differential diagnosis is important to rule out other conditions that can have similar signs and symptoms (eg, stroke, myocardial infarction, or musculoskeletal injury). One of the first questions the medical officer should ask is whether the patient has a recent history of diving or altitude exposure. Signs or symptoms with onsets later than 48 hours after diving or altitude exposure are probably unrelated to decompression.

Therapy According to US Navy Treatment Tables

The standard US Navy therapy for DCS or AGE is recompression to 60 fsw (2.8 ata, 18 msw) while the patient is breathing 100% oxygen. Typically, the course of treatment is determined by the initial signs/symptoms and the response to treatment. US Navy Treatment Table 5 (Figure 18-8) may be used if:

- the only symptoms are joint or limb pain, itching, rash (except cutis marmorata), or local swelling;
- the absence of neurological findings is verified by physical examination; and
- the symptoms are completely relieved within 10 minutes of oxygen breathing at 60 fsw.

Treatment Table 5 requires 135 minutes to administer, with two oxygen periods at 60 fsw and one oxygen period at 30 fsw (1.9 ata, 9 msw). The oxygen periods are 20 minutes long, followed by 5 minutes of air breathing to reduce the risk of CNS oxygen toxicity. Ascent from 60 to 30 fsw and 30 fsw to surface occurs at 1 fsw per minute.

US Navy Treatment Table 6 (Figure 18-9) is used if:

- a neurological exam has not been conducted;
- Treatment Table 5 fails to provide complete resolution of symptoms within 10 minutes; or
- any neurological or cardiopulmonary signs or symptoms are present.

Treatment Table 6 requires 285 minutes to administer, including three 25-minute cycles at 60 fsw and two 75-minute cycles (60 min oxygen, 15 min air) at 30 fsw. Treatment Table 6 can be extended by up to two cycles at 60 fsw and two cycles at 30 fsw.

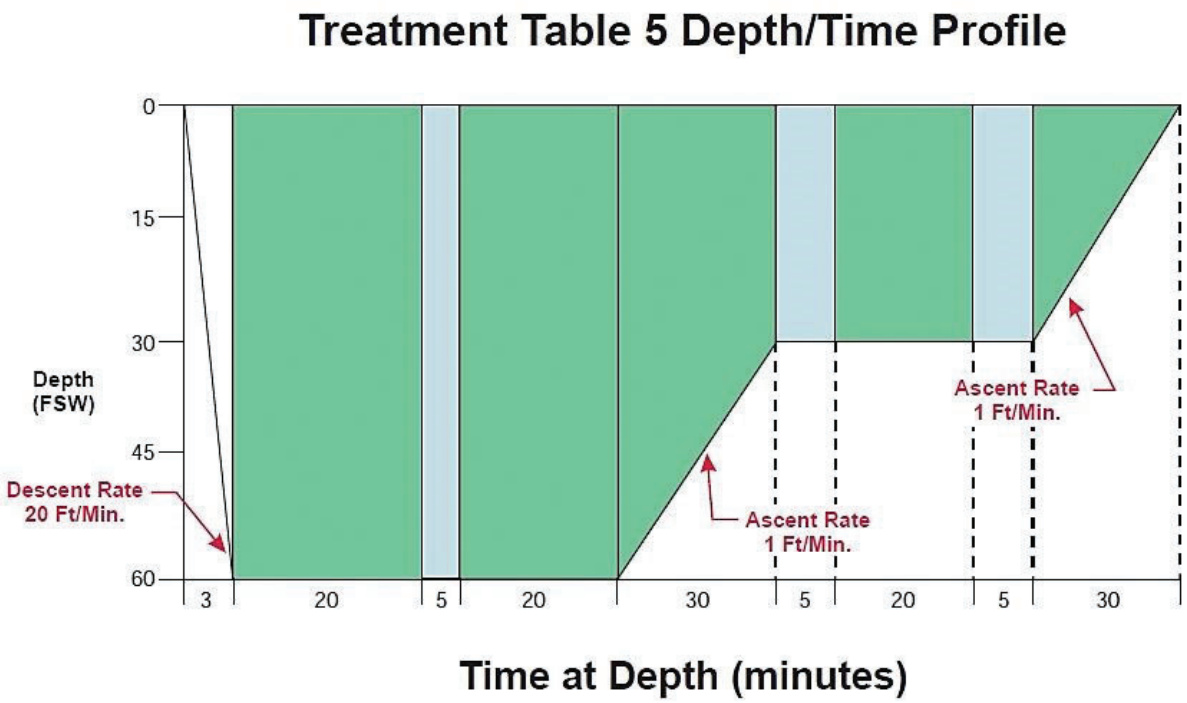

Figure 18-8. The US Navy Treatment Table 5 depth/time profile can be used only if the diving casualty has no neurological symptoms and if all symptoms are relieved within 10 minutes at 60 fsw. Consult the *US Navy Diving Manual* before using this or other treatment tables.
Reprinted from: US Department of the Navy. *US Navy Diving Manual*. Rev 6. Washington, DC: Naval Sea Systems Command; 2011: 20-40. NAVSEA 0994-LP-100-3199.

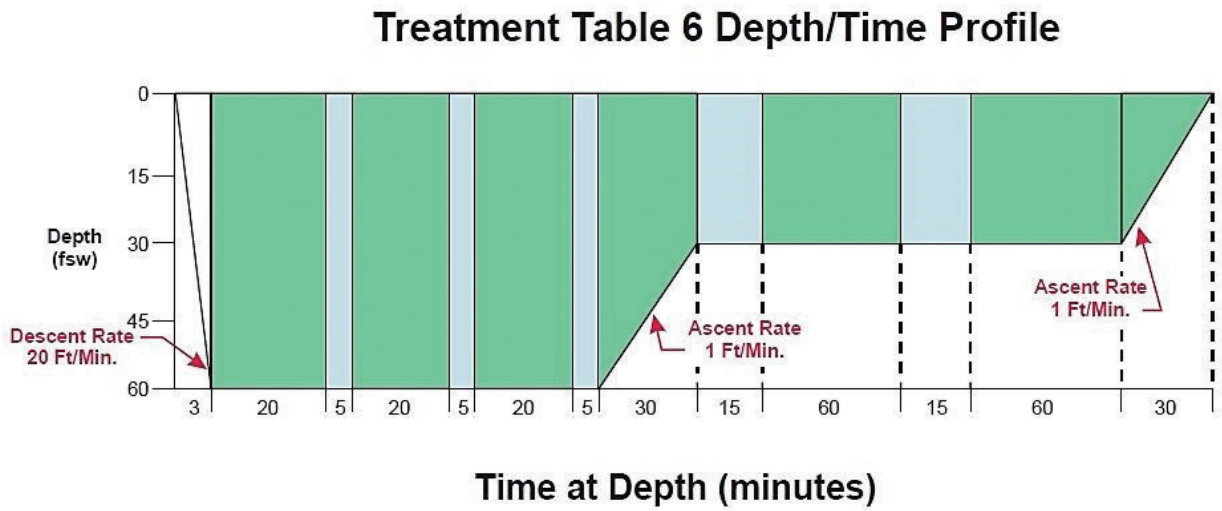

Figure 18-9. In the US Navy Treatment Table 6 depth/time profile, three 25-minute oxygen cycles at 60 fsw, and two 75-minute cycles (60 min oxygen, 15 min air) at 30 fsw, are standard. Treatment Table 6 can be extended by up to two cycles at 60 fsw and two cycles at 30 fsw. The decompression requirements of inside attendants must also be considered so that they do not themselves develop decompression sickness. Consult the *US Navy Diving Manual* before using this or other treatment tables.
Reprinted from: US Department of the Navy. *US Navy Diving Manual*. Rev 6. Washington, DC: Naval Sea Systems Command; 2011: 20-41. NAVSEA 0994-LP-100-3199.

For patients whose symptoms worsen or do not improve after 20 minutes of breathing 100% oxygen at 60 fsw, especially if the dive profile supports a depth greater than 60 fsw, the option is available for further compression to a depth of relief not to exceed 165 fsw (6 ata, 50 msw). Compression to 165 fsw is described in Treatment Table 6A (Figure 18-10). Due to the increased risk of CNS oxygen toxicity, treatment with this table requires switching from 100% oxygen to air or a mixture of 40% to 50% oxygen in nitrogen-helium. A high-oxygen mixture is preferable because it reduces nitrogen narcosis and limits the uptake of additional nitrogen. If symptoms completely resolve within 30 minutes at the depth of relief (not to exceed 165 fsw), the patient can be decompressed at 3 fsw per minute to 60 fsw. From there, the procedures for Treatment Table 6 should be followed.

If relief at depth (not to exceed 165 fsw) is unsatisfactory after 30 minutes, the time at that depth can be extended to 120 minutes, after which decompression is accomplished using US Navy Treatment Table 4 (Figure 18-11).

Patients with residual symptoms, or those who cannot tolerate further oxygen due to pulmonary toxicity, may be given repetitive treatments on subsequent days. (Mild pulmonary toxicity is reversible within hours to a day.) Typically, one to six additional treatments are given, which may be according to Treatment Tables 5, 6, or 9, or sometimes at 30 fsw for 60 to 90 minutes. The rule of thumb is that repetitive treatments should be discontinued when no clinical improvement is observed after two successive treatments.

Decompression Sickness in Saturation Diving

DCS that occurs during or after the slow decompression from saturation dives usually manifests as mild knee or leg pain. Neurological symptoms are rare. The onset of DCS is subtle and may begin with aching or a feeling in the anterior thighs similar to that after hard exercise. This is treated by recompression and administration of high oxygen partial pressures. For occurrences under pressure, oxygen partial pressures of 1.5 to 2.8 ata in helium-oxygen are administered, with a cycle of 25 minutes of high partial pressure oxygen (1.5–2.8 ata) and 5 minutes of low partial pressure oxygen (0.3–0.5 ata).

Recompression during a saturation dive occurs in 5-fsw stages until attaining the depth of relief, at which the diver is treated with oxygen at 1.5 to 2.8 ata for at least 2 hours. After treatment, the diver is maintained at that depth for an additional 2 to 12 hours, depending on the severity of the initial symptoms and the response to recompression therapy. The standard saturation decompression schedule is then resumed. For DCS that occurs after reaching the surface and breathing air, a diver may be treated according to Treatment Table 6.

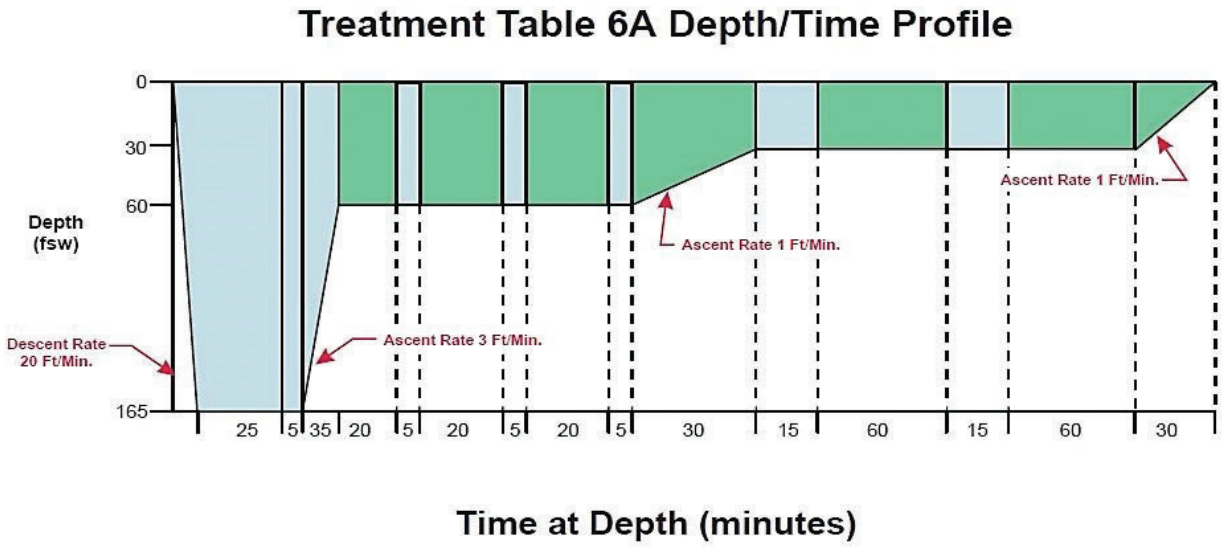

Figure 18-10. In the US Navy Treatment Table 6A depth/time profile, as long as 20 minutes may be spent at 60 fsw before making the decision to compress to a depth of relief not greater than 165 fsw. The advantage of compression to 165 fsw over 60 fsw is greater reduction in bubble volume. At 165 fsw, high-oxygen treatment gas may be utilized to reduce nitrogen uptake. Consult the *US Navy Diving Manual* before using this or other treatment tables.
Reprinted from: US Department of the Navy. *US Navy Diving Manual*. Rev 6. Washington, DC: Naval Sea Systems Command; 2011: 20-42. NAVSEA 0994-LP-100-3199.

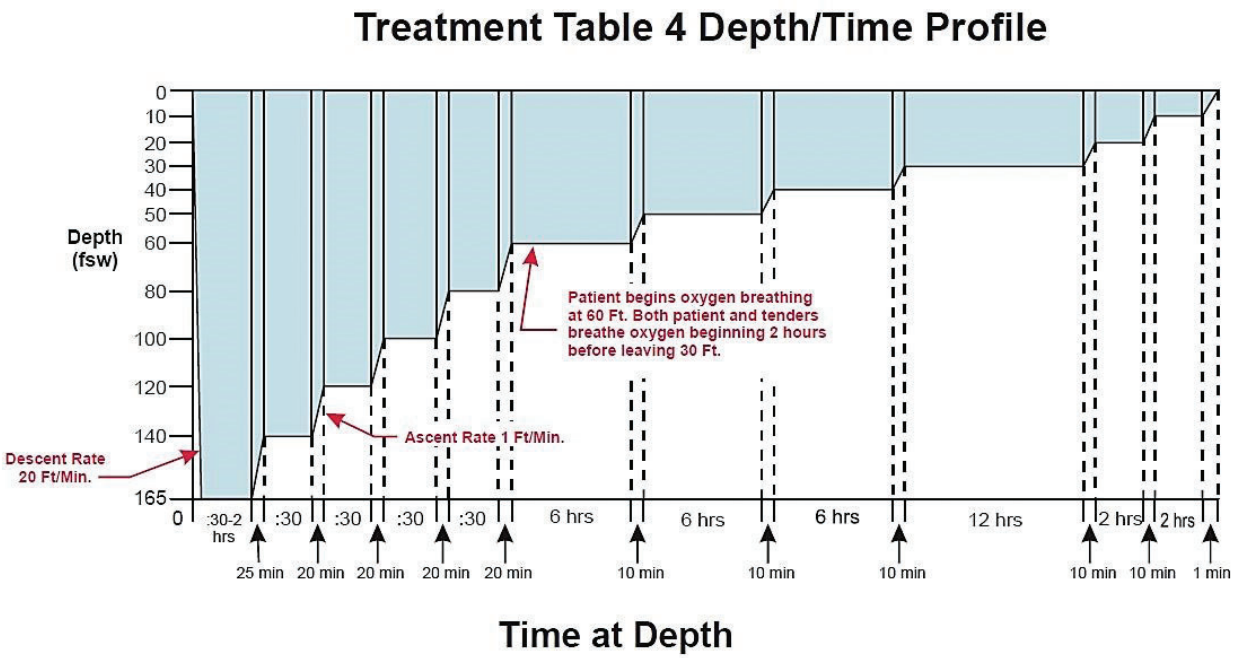

Figure 18-11. In the US Navy Treatment Table 4 depth/time profile, time at 165 fsw is from 30 minutes to 2 hours. At 165 fsw, high-oxygen treatment gas may be breathed to reduce oxygen uptake. Upon reaching 60 fsw, the diver begins breathing 100% oxygen in cycles of 25 minutes interrupted by 5 minutes of air breathing. Consult the *US Navy Diving Manual* before using this or other treatment tables.
Reprinted from: US Department of the Navy. *US Navy Diving Manual*. Rev 6. Washington, DC: Naval Sea Systems Command; 2011: 20-43. NAVSEA 0994-LP-100-3199.

DCS that occurs 60 minutes or more after an ascending excursion can be treated as described above, but DCS that occurs within 60 minutes of ascent from an excursion dive deeper than the saturation depth should be considered serious, even if pain is the only symptom. Neurological symptoms, particularly those related to the inner ear, are not uncommon after rapid ascent from excursions. Recompression to at least the excursion depth should be immediate, and high oxygen partial pressures should be administered for at least 2 hours as described above, after which the patient should be maintained at depth for 12 hours before resumption of standard saturation decompression.

BREATH-HOLD DIVING

Breath-hold diving, or free diving, requires little equipment and provides examples of the many physical and physiological constraints of the underwater environment. Free diving is the oldest form of diving and has long been performed by various cultures around the world for food gathering and the procurement of tradable goods. In modern times, the activity has become popular as a sport, either for competition or recreational spearfishing. Militaries have used free diving as a tactical option since the dawn of naval warfare. It was an essential operational technique in the Pacific during World War II and remains a fundamental skill for present-day combat swimmers.

When a free diver enters the water, he or she encounters several environmental challenges starting with the diving reflex in response to submersion. On descent, ambient pressure increases, which has the strongest effect on enclosed gas spaces within the body (lungs, sinuses). Additionally, the diver must contend with a limited supply of oxygen. Additional challenges occur with ascent, which is when divers most commonly lose consciousness in "shallow water blackout."

Diving Reflex

The diving reflex, or diving response, is a series of physiologic manifestations found in many animals that promotes the conservation of oxygen and the preservation of life while breath-holding and submersed in water. Primarily, they include bradycardia, decreased cardiac output, increased blood pressure, and peripheral vasoconstriction.

Long considered a vagally mediated process to conserve oxygen and redirect blood flow to the critical organs of the heart and brain, some of these effects, particularly bradycardia, can also be elicited by apnea with face submersion or facial exposure to cold.[72,73] Additionally, it appears that the increased ambient pressure of depth plays a role in producing a more robust response.[74] Increased vagal tone has been considered a cause of cardiac arrhythmias associated with breath-hold diving.

Transient splenic contraction, known to occur in diving animals, has also been documented in humans, and contributes to an increase in hematocrit. While this response may prolong the tolerance of the apneic period, it does not appear to be a learned skill as is tolerance to hypoxia and hypercapnea.[75]

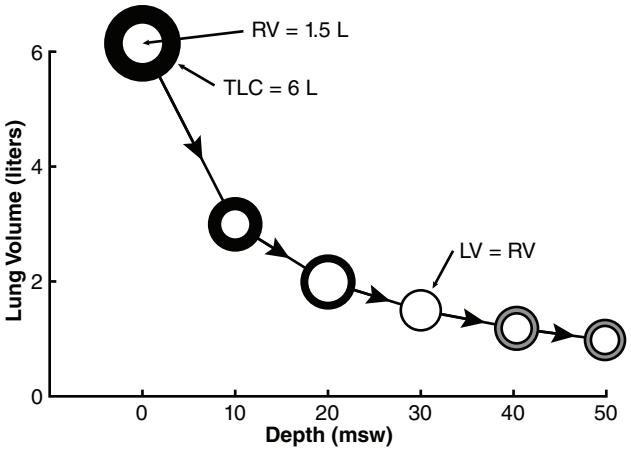

Figure 18-12. Compression of the lungs during descent on a breath-hold dive. The diver, with a residual lung volume (RV) of 1.5 L (white center of circles), begins on the surface with a 4.5-L vital capacity inhalation (black circles) to a total lung capacity (TLC) of 6 L. During descent, the lung volume (LV) decreases in inverse proportion to the absolute pressure, as described by Boyle's law. At 4 ata (30 msw; 99 fsw), the lung volume equals the residual volume. With additional descent, the elasticity of the thorax impedes further compression, leading to a relative vacuum in the lungs. Blood from the peripheral circulation shifts into the pulmonary capillaries (gray circles), which then reduces the residual lung volume (white centers) and allows further descent to 40 and 50 msw (132 and 165 fsw).

Compression of Descent

A free diver must anticipate and compensate for the compression of gas-containing spaces in the body that occurs during descent according to Boyle's law: the volume of a given mass of gas varies inversely with absolute pressure when temperature is held constant. The middle ear requires active inflation to avoid barotrauma (ear squeeze), which is the most common medical problem in diving, but the lungs are also affected, sometimes fatally, in breath-hold diving.

Figure 18-12 represents a breath-hold diver with a total lung capacity of 6 L and a residual lung volume of 1.5 L. If this diver performed a maximal inhalation on the surface (1 ata) and descended for a free dive, Boyle's law predicts that the lung volume would decrease to 3 L at 33 fsw (10 msw; 2 ata) and 2 L at 66 fsw (20 msw; 3 ata). On reaching 99 fsw (30 msw; 4 ata), the lung volume equals the 1.5 L residual volume. With greater descent, the elasticity of the chest wall resists further reduction in volume and the alveolar pressure becomes less than the absolute pressure, which is transmitted equally throughout all solid and liquid tissues in accordance with Pascal's law: pressure exerted at any point on a confined liquid is transmitted uniformly in all directions. This phenomenon causes a relative vacuum between alveolar gas and alveolar capillary blood and leads to engorgement of the alveolar capillaries as blood shifts from peripheral tissues into the thorax. Further descent is possible because these fluid shifts reduce the residual volume.

Consider the example of fluid shift for a diver with a 7.22-L total lung capacity and a 1.88-L residual volume who achieves a depth of 346 fsw (105 msw).[76] By Boyle's law, the ratio of these volumes specifies 93 fsw (28 msw) as the depth at which the lungs are compressed to residual volume. To reach 346 fsw, the residual volume would have to be reduced to 0.63 L, representing a 1.25-L shift of blood from peripheral vessels to pulmonary capillaries. The fluid shift effect is illustrated by record free dives to depths in excess of 660 fsw (200 msw). The current world record for no-limits free diving, which uses a weighted sled for descent and an inflatable lift bag for ascent, was 702 fsw (214 msw), accomplished by Herbert Nitsch in 2007.[77] There is indirect evidence that such blood shifts do occur, but reductions in residual volume may also result from elastic compression of the chest wall and upward shift of the abdominal contents and the diaphragm.[78]

Descent to too great a depth causes compression of the thorax beyond its elastic limit, and the resulting chest pain signals the diver to ascend. Failure or inability to heed this warning pain could cause lung squeeze, or thoracic squeeze, in which chest wall compression, intraalveolar vacuum, or both, damage the thorax and lungs. Thoracic squeeze is rare because the chest pain is a strong signal for the diver to ascend.

In an effort to further push physiological limits, free divers have developed a technique called "lung packing" or glossopharyngeal insufflation. The practice employs the use of the oral and pharyngeal muscles

to compress and force air into the lungs, thereby increasing the amount of available oxygen and overall gas volume. This process creates high transpulmonary pressures and mimics the conditions of pulmonary arterial hypertension.[79] Such practices could cause pulmonary barotrauma leading to hemoptysis[80] and increasing the risk for AGE.[81]

Hypoxia and Hypercapnia

There is an inherent risk of unconsciousness during diving or breath-hold swimming if time underwater is prolonged inadvertently, by hyperventilation, or by will power. This has been a causative factor in many cases of unconsciousness or drowning.[76] Hypercapnia and hypoxia are the principal causes of the ventilatory drive that causes dyspnea. In severe hypoxia, euphoria often precedes unconsciousness, and hypoxic seizures are not uncommon. In the early 1960s, Edward H. Lanphier, MD, and Hermann Rahn, PhD, conducted experiments in a hyperbaric chamber to demonstrate the changes that occur to gases in the lungs during breath-hold and breath-hold diving. This experiment demonstrated the progressive rise in end-tidal carbon dioxide partial pressure and the fall in oxygen partial pressure during a breath-hold experiment at sea level.[82] Gases were sampled from a bag into which the subject exhaled (and re-inhaled) every 10 seconds. Hypoxic and hypercapnic ventilatory drive caused a break in breath-hold at 60 seconds at well above the 20 to 30 mm Hg (0.04 atm) alveolar oxygen partial pressure at which there is a risk of unconsciousness.

In another experiment,[83] a subject made a breath-hold dive to 33 fsw (10 msw). The end-tidal oxygen and carbon dioxide partial pressures increased during descent, as described by Dalton's law of partial pressures: the total pressure exerted by a mixture of gases is equal to the sum of the pressures that each gas would exert if it alone occupied the container. While at depth, the oxygen partial pressure decreased owing to the subject's metabolism, but it remained above the 50 to 60 mm Hg level at which the hypoxic ventilatory drive begins. Carbon dioxide was actually diffused from the lungs into the blood because of its higher alveolar partial pressure. Hypercapnic ventilatory drive forced the subject to ascend after 40 seconds at 33 fsw (10 msw), and the oxygen and carbon dioxide partial pressures decreased, again due to Dalton's law.

In yet another experiment,[83] the subject dived to 33 fsw (10 msw) after hyperventilating to eliminate carbon dioxide and delay the onset of the hypercapnic ventilatory drive. This extended his dive time by 20 seconds, during which oxygen metabolism continued but without inducing hypoxic ventilatory drive. When hypercapnia finally caused the diver to ascend, his oxygen partial pressure fell to below 40 mm Hg, and he exhibited cyanosis, confusion, and loss of control. This is the most common scenario leading to shallow water blackout and subsequent drowning if the diver is not rescued in time.

Decompression Sickness of Free Diving

Free diving is not generally considered to put divers at risk for DCS due to the relatively short time spent at depth; however, several cases of DCS have been reported after free diving.[84] The most common associated risk factors include a high number of dives with short surface intervals. The assumption is that insufficient time is allowed during the surface interval to off-gas the nitrogen load that was accumulated during a single breath-hold dive. If this is multiplied over hours of free diving, a significant decompression obligation may occur that is not met, which could result in DCS.

Even more remote, but shown in case studies, is the occurrence of DCS after single breath-hold dives. These are more likely to be dives to extreme depths, not those performed by spear fishers or recreational free divers. Some modeling of the risk shows that it is negligible shallower than 300 fsw.[85]

SUMMARY

Since the earliest days of breath-hold diving, the underwater environment has posed unique risks due to the physiological effects of changes in pressure on the human body. The introduction of compressed air and artificial gas mixtures have extended diving range and duration but also created problems related to inert-gas effects and rapid ascent to the surface. A series of gas laws represented by simple equations describe the physical effects of pressure and temperature on gas volume and constituent partial pressures. Pressure change creates the potential for barotrauma in closed or functionally trapped gas pockets including the face mask, middle ear, inner ear, sinuses, and lungs, all of which are subject to problems on descent (compression or "squeeze") and ascent (expansion and possible rupture). Inhalation of compressed air or artificial gas mixtures at increased pressure readily leads to problems with oxygen toxicity, excess carbon dioxide, carbon monoxide poisoning, and narcosis due to nitrogen and other inert gases. At extreme depth, pressure itself produces effects collectively described as HPNS.

Early work with compressed air produced syndromes (called "caisson disease" or "the bends") that were eventually recognized as the result of relatively rapid decompression. Ascent to the surface reduces ambient partial pressure of nitrogen or other inert gases, resulting in the formation of bubbles in blood and tissues, which can cause clinical problems ranging from cutaneous mottling to joint pain and neurological dysfunction. In addition, leakage of expanding gas from the pulmonary system into the arterial circulation (AGE) can lead to circulatory collapse, severe cerebral damage, and death.

Fortunately, these problems can be avoided by correct use of diving equipment and implementation of staged decompression to allow gradual desaturation of tissues without excessive bubble formation. Treatment of decompression problems requires return to pressure in a hyperbaric chamber to shrink existing bubbles, followed by gradual decompression combined with inhalation of 100% oxygen to accelerate the elimination of inert gases from the body. All military physicians should be aware of the pathophysiology, prevention, and treatment of diving-related disorders.

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