# Chapter 30

# PHYSICS, PHYSIOLOGY, AND MEDICINE OF DIVING

JAMES VOROSMARTI, JR, MD* AND RICHARD D. VANN, PHD†

INTRODUCTION

PHYSICAL PRINCIPLES
- Gas Laws
- Buoyancy

UNDERWATER PERTURBATIONS OF THE SPECIAL SENSES
- Vision
- Hearing

PATHOPHYSIOLOGICAL EFFECTS OF PRESSURE: BAROTRAUMA
- Middle Ear Barotrauma (Descent)
- Middle Ear Barotrauma (Ascent)
- Inner Ear Barotrauma
- Sinus Barotrauma
- Pulmonary Barotrauma of Descent
- Pulmonary Barotrauma of Ascent

PATHOPHYSIOLOGICAL EFFECTS OF COMMON DIVING GASES
- Oxygen Toxicity
- Carbon Dioxide Toxicity
- Carbon Monoxide Toxicity
- Nitrogen Narcosis
- Helium and Other Inert Gases
- Hydrogen
- Physiological Effects of Pressure: High-Pressure Nervous Syndrome

DECOMPRESSION SICKNESS
- Inert Gas Exchange
- Bubble Formation
- Pathophysiology of Decompression Sickness
- Diving at Altitude and Altitude Exposure After Diving
- Factors Affecting Individual Susceptibility
- Predicting and Preventing Decompression Sickness

ADDITIONAL THREATS TO DIVERS
- Thermal Stress
- Underwater Blast Injury
- Drowning and Near-Drowning

SUMMARY

*Captain, Medical Corps, US Navy (Ret); Consultant in Occupational, Environmental, and Undersea Medicine, 16 Orchard Way South, Rockville, Maryland 20854
†Captain, US Navy Reserve (Ret); FG Hall Hypo/Hyperbaric Center, Box 3823, Duke University Medical Center, Durham, North Carolina 27710

## INTRODUCTION

Diving as a military activity is sufficiently common that any military physician has a probability—and those in Special Operations Forces have a high probability—of involvement in diving operations or in treating or examining divers during his or her career and certainly should have some familiarity with diving medicine. 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 BC, when Scyllus[1] and his daughter Cyane saved the fleet of Xerxes by freeing the ships' anchors and allowing them to get underway rapidly when threatened by a sudden storm. The Syracusans[2] are said to have trained divers to swim under water and damage enemy ships. Divers of Tyre[3] were employed to cut the anchor ropes of Alexander the Great's ships during the siege of Tyre

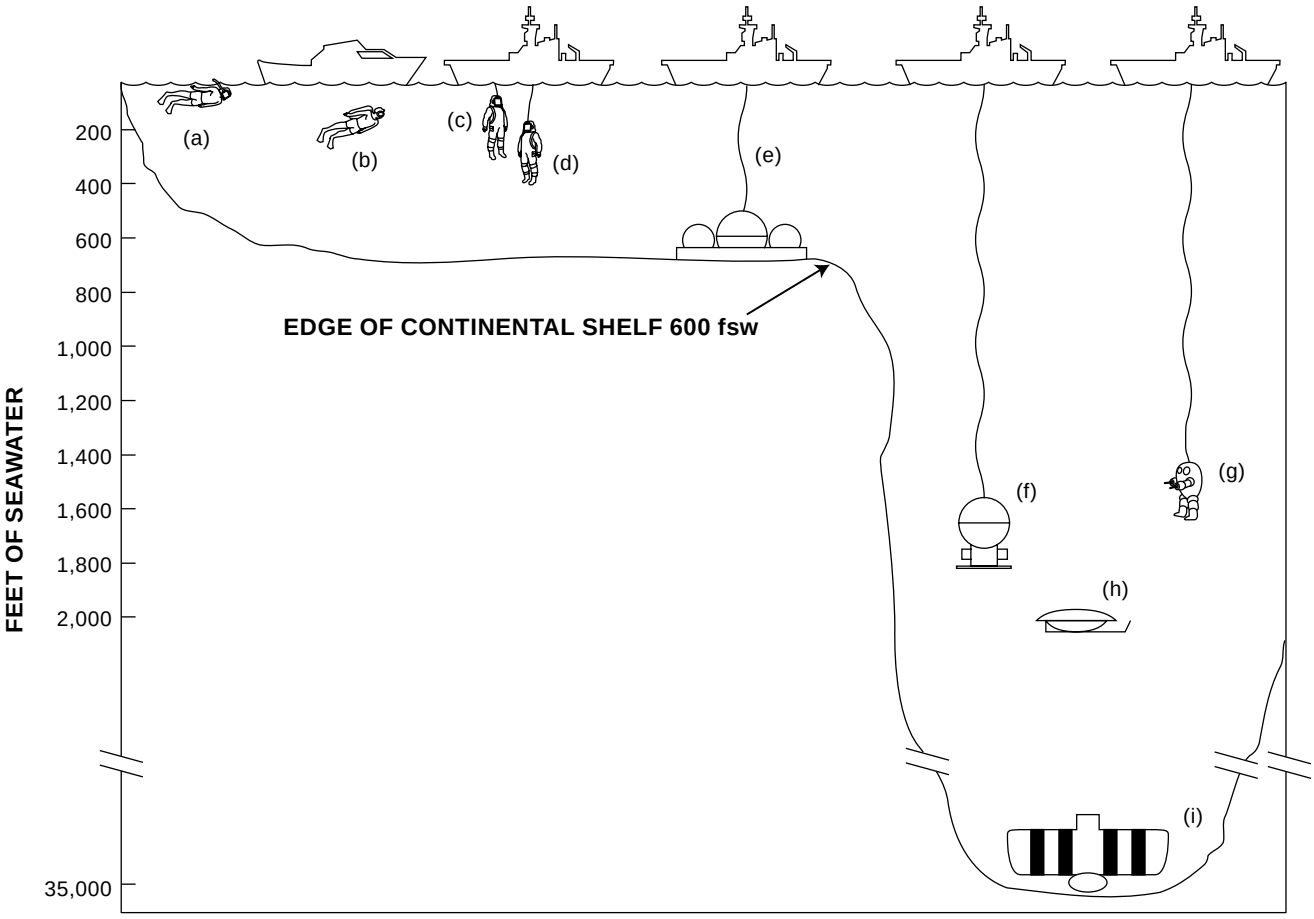


**Fig. 30-1.** Various forms of diving and other submarine activities in relation to ocean depth. (**a**) Breath-hold diving is usually not deeper than 45 fsw, although some divers can go deeper. (**b**) Self-contained underwater breathing apparatus (scuba) diving on air is usually limited to 130 fsw. (**c**) Surface-supported air diving is usually limited to 180 fsw, US Navy, or for commercial diving in the United States, 220 fsw. (**d**) Surface-supported helium–oxygen diving without a bell is usually to a depth of about 300 fsw, because of the problems of long decompressions; (**e**) the deepest habitat operation, 520 fsw; (**f**) the deepest saturation diving at sea operation, 1,510 fsw; (**g**) the deepest dive in an armored 1-atm suit, 1,440 fsw. (**h**) Research submersibles can operate to depths of about 2,000 fsw; (**i**) the bathyscaphe *Trieste* has been to the deepest-known depth of the ocean, 35,800 fsw, in the Marianas 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.

in 332 BC. However, not until the 19th century, after many technological advances, did diving became 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.

Figure 30-1 shows the relationship of the depth of the oceans to the attempts that man has 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 equipment required, increased breathing resistance, and other effects of pressure such as nitrogen narcosis. The diver is prone to diseases due to pressure changes, which are much greater underwater than those experienced by the aviator.

## PHYSICAL PRINCIPLES

Certain fundamental physical principles need to be understood before the deleterious effects specific to diving can be appreciated. Foremost among these physical principles are the descriptions of the behaviors of gases under pressure. Buoyancy is also a consideration.

Barometric pressure is the force per unit 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 Earth; its value was initially measured against a column of mercury and was given a value of 760 mm Hg at sea level under standard conditions.

The primary unit of pressure in the International System of Units (SI units, Système International d'Unités) is the pascal (Pa, a unit derived from newtons per square meter), where 1 atmosphere (atm) equals 101.3 kPa. In addition, other units are sometimes used (Exhibit 30-1).

Many pressure gauges read zero at ambient (surface) pressure, thereby measuring only differential pressure (eg, a blood pressure gauge). 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 meters of depth in sea water (msw) or 33.08 feet of depth in sea water (fsw). In fresh water, however, 1 atm = 10.38 msw, or 33.83 fsw.

The following formula may be used when convert-

**EXHIBIT 30-1**

**PRESSURE UNITS AND EQUIVALENTS**

| | | |
|---|---|---|
| 1 atm | = | 1.013250 bar |
| 1 atm | = | 101.3250 kPa |
| 1 atm | = | 760.0 torr* |
| 1 atm | = | 14.6959 psi |
| 1 atm | = | 33.08 fsw |
| 1 atm | = | 10.13 msw |
| 1 bar | = | 100.00 kPa* |
| 1 bar | = | 32.646 fsw†,§ |
| 1 bar | = | 10.00 msw |
| 1 msw | = | 10.000 kPa‡,§ |

*Signifies a primary definition from which other equalities were derived.

†Primary definition for feet of seawater (fsw); assumes a density for seawater of 1.02480 at 4°C, which is the value often used for calibration of a depth gauge.

‡Primary definition for meters of seawater (msw); assumes a density for seawater of 1.01972 at 4°C.

§These primary definitions for fsw and msw are arbitrary because the pressure below a column of seawater depends on the density of the water, which varies from point to point in the ocean. These two definitions are consistent with each other if the appropriate density correction is applied.

Adapted with permission from the pressure conversion table published on the last page of each issue of *Undersea & Hyperbaric Medicine,* the journal of the Undersea and Hyperbaric Medical Society, 10531 Metropolitan Avenue, Kensington, Md.

ing from diving depth to atmospheres (Equation 1):

$$(1) \qquad \text{absolute pressure} = ( D + De ) / De$$

where *D* represents the depth in units of length and *De* represents the number of those units equivalent to 1 ata. NOTE: this equation assumes a pressure of 1 atm at the surface of the water and requires correction for high-altitude diving.

**Gas Laws**

Changes in barometric pressure and 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 the gas laws and related physical principles is therefore prerequisite to understanding the physiological effects of the acute 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 (Equations 2a and 2b),

$$(2a) \qquad P_1 / P_2 = V_2 / V_1$$

or the formula can be rewritten:

$$(2b) \qquad P_1 \bullet V_1 = P_2 \bullet V_2$$

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 (Equation 3):

$$(3) \qquad (P_1 - P_{H_2O}) / (P_2 - P_{H_2O}) = V_2 / V_1$$

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 (Equations 4a and 4b):

$$(4a) \qquad P_1 / P_2 = T_1 / T_2$$

or the formula can be rewritten:

$$(4b) \qquad P_1 \bullet T_2 = P_2 \bullet T_1$$

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 (Equation 5):

$$(5) \qquad (P_1 \bullet V_1) / T_1 = (P_2 \bullet V_2) / T_2$$

***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 (Equation 6):

$$(6) \qquad P_t = P_1 + P_2 + P_3 + P_n$$

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 may be calculated from the following relationship (Equation 7):

$$(7) \qquad P_x = F_x \bullet P_t$$

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.

### *Related Physical Principles*

**Gas-Filled Compartments.** The gas laws are discussed above in terms of a flexible container that fully accommodates alterations in volume due to changes in pressure and temperature. For gas in a rigid container, however, increasing ambient pressure will eventually cause structural collapse of the container, while decreasing pressure will force the walls to burst. For this reason, the inability to ventilate semirigid, 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.

**Transmission of Pressure in a Liquid (Pascal's Law).** Pressure exerted at any point on a confined liquid is transmitted uniformly in all directions. Because solid organs are mostly liquid, pressure exerted on them is distributed equally throughout the body and does not change tissue volume.

**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, assuming that temperature remains constant and 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.

### Buoyancy

Buoyancy is important to a diver, as 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 may occur. As stated in Archimedes' 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—being 10 lb heavier than the water displaced—therefore, sink. If the amount of water displaced is equal in weight to the object, its depth remains constant, as it is neutrally buoyant.

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, he might swim too deep and develop oxygen toxicity; if positively buoyant, he 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 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 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. Buoyancy increases as the gas in diver-worn tanks is used.

## UNDERWATER PERTURBATIONS OF THE SPECIAL SENSES

### 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 (Figure 30-2). 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 annoying visual distortion. Stereoacuity or depth perception, the ability to determine the relative distance between objects, is also adversely affected. This is especially noticeable in clear, well-illuminated water and worsens with decreasing illumination and increasing turbidity. Decreased contrast underwater, even in clear water, is thought to contribute to this phenomenon.

Absorption of light affects underwater vision by decreasing available illumination. In clear (nonturbid) water, only about 20% of incident light penetrates to 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), and 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

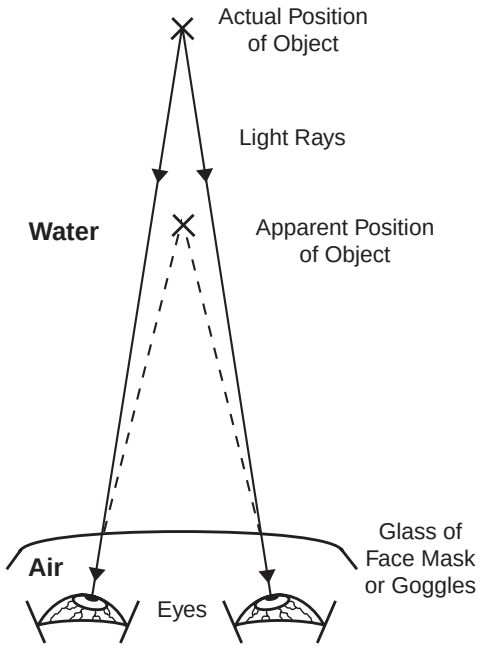


**Fig. 30-2.** When seen underwater, images are displaced toward the diver. The closer apparent position is caused by the refraction of light rays as they pass through the water, the material of the face mask, and the gas inside the mask.

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 will appear blue–green. For example, at a depth of about 10 ft (3 m) blood will appear 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 31, Military Diving Operations and Medical Support) 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. It reverses with decreasing pressure. This is true of 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. Without this difference, localization can often be impossible.

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, because helium shifts the resonance of vocalizing structures and gas-filled cavities and thus drastically changes 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 the "hard hat" (the classic rigid diving helmet) may still have this problem, the newer diving equipment is quieter and hearing loss is not common. If a decompression 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 (*so*und *n*avigation *a*nd *r*anging) 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 NAVSEA Instruction Series 3150.2. 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 sensorineural deafness. Whether the 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 can stay clear of the danger zone.

Divers exposed to sonar have reported feeling vibrations (in parts of 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 those symptoms seen in 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.

## PATHOPHYSIOLOGICAL EFFECTS OF PRESSURE: 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. 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 will expand during ascent causing abdominal distress, cramping, flatus, or eructation. No therapy is usually required as the gas will be expelled over a short time, but several cases of gastric rupture after decompression have required emergency attention.[4]

Less-obvious body cavities are restored dental caries and gingivitis, which can also be gas-filled. Often referred to as aerodontalgia, dental barotrauma—as the result of poor or eroded fillings or gum infection—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 drysuit 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 ecchymoses 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, 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. The consequences of nonequilibration 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 (Figure 30-3). 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. Should cold water then enter 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 in-

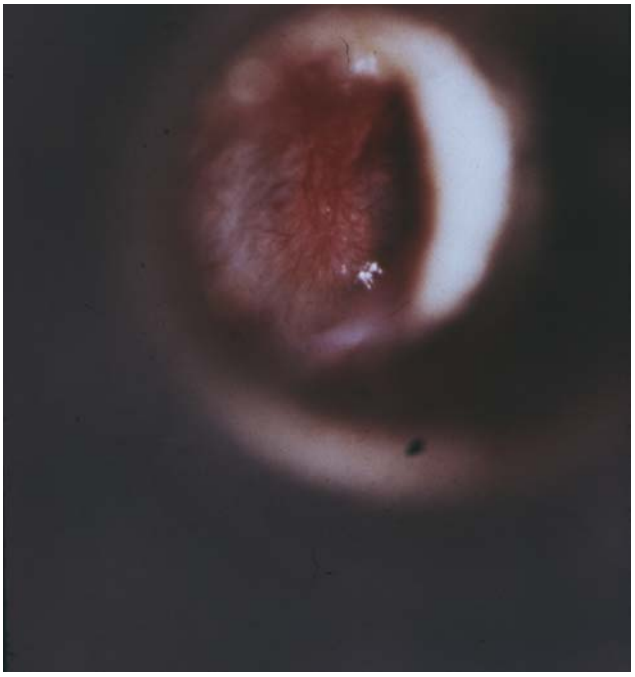

**Fig. 30-3.** Photograph showing moderate barotrauma of the middle ear. Note the hemorrhaging around the handle of the malleus and surrounding tympanic membrane, and the slight retraction and dullness of the tympanic membrane.

crease 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. For unknown reasons, clearing the ears in a head-up position is easier than in a head-down position.

Prevention of middle ear barotrauma includes not diving when any condition exists that prevents proper eustachian tube function. Some divers often have, and most sometimes have, "sticky ears," a condition in which eustachian tube function is present but equilibration is difficult to achieve. Oral, nonsedative decongestants can be helpful but may not be effective in certain cases. Topical decongestants should be used with care, as 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.

**Middle Ear Barotrauma (Ascent)**

Middle ear barotrauma of ascent is infrequent, as the middle ear can normally vent passively through the eustachian tube when its pressure reaches about 50 cm $H_2O$ 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 or pain, tinnitus, vertigo, or hearing loss. Vertigo, which is due to a pressure differential between the left and right middle ears (alternobaric vertigo), is a particular danger, as it can lead to disorientation and the inability to locate the surface. Pressure equalization during ascent, if necessary, uses the same maneuvers—Valsalva and Frenzel—as during descent. Descending a few feet before attempting equalization may be helpful. 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. If deafness is instantaneous and present in all frequencies, it is probably due to severe round-window rupture. If it is mild and de-

velops over several days, a round-window fistula may be present. If air leaks into the perilymph, the deafness may change with head position. If the deafness is limited to high frequencies (≥ 4,000 Hz) and does not change over several days, it is probably due to hemorrhage or trauma in the cochlea. Immediate referral to an otolaryngologist is recommended to prevent permanent hearing loss. Bed rest with the head elevated 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 until 1 week after improvement plateaus. Air 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 not to ascend above 200 ft (61 m). If deafness, vertigo, or tinnitus persist despite all therapy, the casualty should be advised against any future diving. Inner ear barotrauma and inner ear decompression sickness (DCS; discussed below) can have similar symptoms, 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. Sometimes the fluid, blood, or gas can be felt escaping from the sinuses. Divers who are unfamiliar with the problem can become panicky and extremely worried should this occur. 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. Fracture of the sinus walls, allowing gas or fluid to enter the soft tissues, is a rare event. Treatment is the application of nasal decongestants.

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

**Pulmonary Barotrauma of 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; and
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, as chest pain alerts the diver of excessive depth. For most people the maximum breath-hold depth is about 100 fsw (30 msw), although a few individuals have exceeded 300 fsw (90 msw). One factor believed responsible for these deeper dives in certain individuals is their bodies' unusual ability to pool blood in the veins of the thoracic cavity (up to 1 L), which compensates for the inability to further decrease the volume of gas below residual volume. (Also see Chapter 31, Military Diving Operations and Medical Support.)

**Pulmonary Barotrauma of Ascent**

Pulmonary barotrauma of ascent is potentially the most severe of all types of barotrauma and can occur in all types of diving and in submarine escape (as well as in rapid decompression to high altitudes; see Chapter 32, Pressure Changes and Hypoxia in Aviation). 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[5] in ascent from a depth as shallow as 6 ft (1.8 m) of water. During naval submarine escape training, the incidence of pulmonary barotrauma is about 1 in 2,300, with death occurring in about 1 in 53,000 ascents.[6] These ascents are done under close supervision after medical screening, with emergency medical care immediately available. Predisposing conditions for pul-

monary barotrauma are breath-holding during ascent, previous spontaneous pneumothorax, asthma, cysts, tumors, pleural adhesions, infections, pulmonary fibrosis, or any other disease that weakens the lung tissue or interferes with the free passage of gas.

Pulmonary barotrauma may result in local or wide damage to lung tissue, characterized by disruption of the alveolar–capillary membrane and consequent passage of alveolar gas (*a*) into the mediastinum, causing mediastinal emphysema; (*b*) into the pleural space, causing pneumothorax; and (*c*) into the capillaries, with probable resultant cerebral arterial gas embolism (CAGE). These traumata can occur alone or in combination. Patients with pulmonary barotrauma but no signs or symptoms of CAGE 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, should emerge.

### *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 near-drowning or acute respiratory distress syndrome. If pulmonary barotrauma is suspected, positive-pressure breathing is not recommended unless it is absolutely necessary for survival, to avoid the possibility of further lung damage or producing other complications of pulmonary barotrauma. Recompression is not needed unless symptoms or signs of CAGE 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. Symptoms include a fullness in the throat, retrosternal discomfort, voice changes, dyspnea, dysphagia, syncope, shock, and unconsciousness. Signs include crepitus under the skin, faint heart sounds, paralysis of the recurrent laryngeal nerve, tachycardia, hypotension, cyanosis, and coma. Crepitus that is heard over the precordium and is related to heart sounds is called Hamman's sign. Gas that has dissected into the pericardial sac or between the pleura and pericardium may cause cardiac tamponade. A chest roentgenogram will confirm the diagnosis, or it may be the only evidence of mediastinal emphysema. In our experience, mediastinal emphysema and the variants listed above are not very common. This is a diving problem that is generally not life-threatening and usually requires no treatment. Breathing 100% oxygen will accelerate absorption of the gas in mild cases and is required in severe cases for life support. Severe cases may benefit from recompression to reduce the volume of gas in the tissues. An oxygen treatment table for shallow depths, such as US Navy Treatment Table 5,[7] (see Figure 31-23 in Chapter 31, Military Diving Operations and Medical Support) should suffice.

### *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, should they occur, 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 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 CAGE or DCS. Treatment at sea level does not differ from that of a pneumothorax resulting from other causes. If recompression is required for therapy of CAGE or

DCS, a chest tube should be inserted before recompression. For emergency recompression, a needle with a nonreturn valve should be inserted and later converted to the usual chest tube with suction or underwater drainage. If recompression is needed and a chest tube with drainage is in place, care is required to prevent sucking water and gas 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. Because of the possibility of infection, we recommend that chest tubes not be used in a recompression chamber unless absolutely necessary.

### *Cerebral Arterial Gas Embolism*

CAGE can be life-threatening or result in permanent injury, and requires immediate recompression. CAGE 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. The bubbles block small blood vessels, with concomitant serious results. 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 cardiovascular accident. The onset of signs and symptoms is sudden and dramatic, appearing during the ascent or always within a few minutes of surfacing. The most common signs are semiconsciousness, 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.

Arterial gas emboli, whether in the brain or elsewhere, cause ischemia in the surrounding tissue and induce coagulopathies, hemorrhages, and endothelial damage. Protein and cells are deposited on the bubble surface. If treatment—recompression—is long delayed, reperfusion injury may occur once the circulation has been reestablished.

Studies may show abnormal electrocardiographic, electroencephalographic, and brain scan findings, but if CAGE 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. Surveys show that delay of recompression for more than a few hours results in a 50% decrease in the cure rate.[8(p107)] In severe cases, even immediate and aggressive therapy may not produce a good result, and death may follow. 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. There is no way to predict which case will resolve and which will fail. Therefore, all cases of suspected CAGE should be recompressed as soon as possible.

Iatrogenic CAGE has occurred during open heart surgery, brain surgery, and kidney dialysis. Treatment in these cases is no different from that in cases resulting from ambient pressure changes.

The treatment for CAGE is immediate recompression. There is some controversy about whether recompression should be to low pressure, 60 fsw (18 msw, or 2.8 ata) (in accordance with US Navy Treatment Table 6),[7] or to high pressure, 165 fsw (50 msw, or 6 ata) (in accordance with US Navy Treatment Table 6A).[7] (See Figures 31-24 and 31-25 in Chapter 31, Military Diving Operations and Medical Support.) At the present time the *US Navy Diving Manual*[7] recommends initial recompression to 60 fsw 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 165 fsw (50 msw) is an option in an effort to make remaining bubbles smaller. If immediate recompression is not available, 100% oxygen breathing by mask and intravenous fluid administration should be initiated. The patient should be positioned on his 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. This is no longer recommended, as it has been determined[9] that the head-down position increases central venous and cerebral venous pressure and cerebral edema. Increased venous pressure may also make it more difficult for bubbles to pass into the venous circulation.

If convulsions are present, Valium (diazepam; manufactured by Roche Products Inc, Manati, Puerto Rico) may be useful. Lidocaine hydrochloride has been shown[10] to increase cerebral vasodilation and decrease the rise in intracranial arterial pressure. It is also a useful drug in the treatment of cardiac dysrhythmias. The use of parenteral steroids, once thought to reduce cerebral edema, is controversial. Their utility has not been verified as

of this writing, and some claim[8(p109)] that they actually are detrimental. In any case, CAGE causes immediate damage and steroids take hours before an effect is evident.

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 no higher than 800 ft (242 m), and preferably in an aircraft that can be pressurized to sea level. Other supportive therapy should be used as required.

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

## PATHOPHYSIOLOGICAL EFFECTS OF COMMON DIVING GASES

Gases used in diving should be free of contaminants. A small fraction of contaminants cause increased toxicity at high barometric pressure because the partial pressures increase with depth, as described by Dalton's law (see above). The current US Navy standards for the purity of air, oxygen, nitrogen, and helium are found in the *US Navy Diving Manual*.[11] Breathing gases that do not meet these standards are not to be used for diving. This section deals with the pathological condition associated with commonly used breathing gases and some common contaminants ($CO_2$, CO).

### Oxygen Toxicity

The pathophysiology of all metabolically active gases ($O_2$, $CO_2$, CO) depend on the partial pressure, not on the percentage of that gas in the breathing mixture. For example, at 1 atm, a mixture containing 5% oxygen is not compatible with life but at 5 atm is perfectly adequate. Breathing high partial pressures of oxygen can cause two types of toxicity: pulmonary and cerebral.

#### *Pulmonary Oxygen Toxicity*

Pulmonary oxygen toxicity was first described by Lorrain-Smith[12] in 1899 as the result of breathing pure oxygen at sea level for a prolonged time (usually 2–3 d). We now know that this can occur at inspired partial pressures of oxygen ($P_{IO_2}$) as low as 0.5 to 0.6 atm, either at 1 atm or at higher pressures. Clinically, patients first report the symptoms of tracheobronchitis, such as tracheal burning and cough following deep inspiration. If oxygen administration continues, actual breathlessness will occur. A measurable sign of pulmonary oxygen toxicity is a decrease in vital capacity, with increasing loss of inspiratory capacity and increasing residual volume. Atelectasis occurs and increases as the pathology worsens. The pathophysiology includes endothelial thickening, proliferation of cells, loss of surfactant, exudate, hemorrhage, and consolidation. Even though the patient may be breathing pure oxygen, the cause of death is asphyxia.

Although pulmonary oxygen toxicity is an uncommon problem, gas content must be carefully planned in saturation diving, where divers are exposed to a $P_{IO_2}$ in the range of 0.4 to 0.5 atm for days with intermittent exposures to higher levels for diving excursions (see Chapter 31, Military Diving Operations and Medical Support, for a discussion of saturation diving). Pulmonary oxygen toxicity can also occur in prolonged recompression therapy for DCS or CAGE. When this occurs, the $P_{IO_2}$ must be reduced to at least 0.5 atm to allow recovery.

The effect of oxygen exposure on the vital capacity can be estimated by the unit pulmonary toxicity dose (UPTD),[13] which is based on the experimental measurement of changes in vital capacity in human subjects. This concept assumes no decrement in lung function when breathing oxygen at a partial pressure of 0.5 atm or less. The results of such calculations should be considered to be no more than a rough guide to pulmonary effects, as there are large individual differences, but it is useful as a guide to limiting prolonged oxygen breathing at partial pressures higher than 0.5 atm.

#### *Cerebral Oxygen Toxicity*

Cerebral oxygen toxicity was discovered by Paul Bert[14] in 1878 and is referred to as acute oxygen poisoning because of its rapid onset. Acute toxicity occurs only while breathing oxygen under pressure at $P_{IO_2}$ greater than 1.3 atm. The lowest documented $P_{IO_2}$ at which convulsions have occurred during diving with 100% oxygen is 1.74 atm, as opposed to 1.6 atm during mixed-gas diving. The first sign of trouble is often a grand mal convulsion. Other symptoms of central nervous system (CNS) oxygen toxicity include muscular twitching around the mouth or of the abdominal wall, nausea, dizziness, tunnel vision, and anxiety, but these are rare and cannot be relied on to precede seizures. The treatments for CNS oxygen toxicity are to remove the patient from the high-level oxygen source and to prevent self-injury. Rarely, a convulsion may occur within minutes after stopping oxygen breathing.

This is known as the "off effect" and has no accepted explanation, but it may represent the culmination of a process that began during oxygen breathing.

Because oxygen toxicity is dose-related, oxygen can safely be used so long as time and depth are limited. This allows the use of oxygen at greater than 1 atm 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 to 25 minutes, separated by 5-minute air breaks (see Chapter 31, Military Diving Operations and Medical Support, for a discussion of treatment tables).

A number of factors affect sensitivity to oxygen toxicity. These include the 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, and this increases the risk of a working diver compared with that of a diver at rest in a dry hyperbaric chamber. Increased inspired or arterial carbon dioxide decreases the latency, and individuals prone to retain carbon dioxide may be at greater risk. Modern underwater breathing apparatuses (UBAs) have less breathing resistance and dead space than earlier UBAs, but heavy work at great depth appears to cause carbon dioxide retention and to 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 cerebral derangements is still not understood. The most accepted explanation is that reactive oxygen species, such as superoxide, hydroxyl radicals, and hydrogen peroxide, are generated and may interfere with cellular metabolism and electrical activity. Oxygen can inactivate many enzymes and metabolic pathways, with enzymes containing a sulfhydryl group being especially sensitive. It is puzzling that the time required to effect these changes in vitro is much longer than the time required to produce convulsions in intact animals, although the partial pressure of oxygen in the in vivo tissue is much lower than the $P_{IO_2}$. Another possibility is lipid peroxidation and depression of the prostaglandin $I_2$ system, leaving the thromboxane $A_2$ system intact. Lipid peroxidation can affect cell membrane function causing decreased glutamate uptake, increased potassium retention, decreased active sodium transport, and inactivation of the sodium–potassium adenosine triphosphatase pump. In lung tissue, decreases occur in serotonin and norepinephrine uptake; pulmonary capillary endothelium function; and prostaglandin $E_2$, bradykinin, and angiotensin metabolism. Other nonbiochemical effects of oxygen include vasoconstriction-induced reduction in peripheral blood flow, decreased carbon dioxide carrying capacity by hemoglobin, and increased red blood cell fragility, but these subtle changes do not generally cause concern.

The probability of clinical oxygen toxicity is reasonably low if the time–depth limits in the *US Navy Diving Manual*[11] are observed (Table 30-1). In routine air diving the oxygen exposures are not high enough to produce either CNS or pulmonary toxicity, but the recent use of nitrogen–oxygen mixes with more than 21% oxygen (nitrox, enriched air) by recreational divers has led to a number of CNS oxygen-toxicity episodes and some fatalities.[15]

The *US Navy Diving Manual*[11] also allows making one excursion as deep as 50 fsw (15.1 msw) during dives on 100% oxygen, but only for 5 minutes and only under the following conditions:

- The maximum dive time cannot exceed 240 minutes.
- Only one excursion is allowed.
- The diver must return to 20 fsw (6.1 msw) or less by the end of the excursion.
- The excursion must not exceed 15 minutes at 21 to 40 fsw (6.4–12.1 msw) or 5 minutes at 41 to 50 fsw (12.4–15.1 msw).

**TABLE 30-1**

**SINGLE-DEPTH OXYGEN EXPOSURE LIMITS**

| Depth (fsw) | Maximum Oxygen Time (min) |
|---|---|
| 20 | 240 |
| 30 | 80 |
| 35 | 25 |
| 40 | 15 |
| 50 | 10 |

Reproduced from US Department of the Navy. *US Navy Diving Manual.* Vol 2. Washington, DC: DN; 1991. NAVSEA 0994-LP-001-9020. Rev 3.

The US Navy closed-circuit mixed gas UBA (the Mk 16 UBA) is designed to control the oxygen partial pressure to 0.7 atm. CNS oxygen toxicity is not a problem at this level (various other types of UBAs are discussed in Chapter 31, Military Diving Operations and Medical Support). During saturation diving, the US Navy maintains the oxygen partial pressure in the chamber at 0.40 and 0.45 atm; for diving excursions, the allowable partial pressure is 0.40 to 1.2 atm.

NOTE: The partial pressure and depth–time limits given here are subject to change. Readers should refer to the latest appropriate standards for the diving operation at hand. In addition, if the diving is conducted under other than US Navy authority (ie, another government agency or a foreign government), different limits may be used. American divers may be prohibited from diving under procedures other than those specified by the US Navy.

### Carbon Dioxide Toxicity

The effects of increased carbon dioxide include hyperventilation, dyspnea, tachycardia, headache, lightheadedness, and dizziness progressing to mental confusion and unconsciousness (Table 30-2). 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 open-circuit breathing apparatus or failure of the carbon dioxide absorbent in a closed-circuit apparatus;
- increased respiratory dead space owing to poor equipment design, or inadequate ventilation of chamber or helmet;
- voluntary hypoventilation (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; and
- increased breathing resistance, either intrinsic (diver's lungs) or extrinsic (breathing equipment); the latter is more common than the former. Added breathing resistance decreases ventilatory response to elevated carbon dioxide.

**TABLE 30-2**

**ACUTE EFFECTS OF INCREASED INSPIRED CARBON DIOXIDE**

| Carbon Dioxide (%,SLE) | Effects |
|---|---|
| 0–3 | No adverse effects |
| ~ 5 | Mild hyperventilation |
| 5–10 | Shortness of breath, panting, confusion, drowsiness |
| 10–20 | Extreme respiratory distress, unconsciousness, muscle twitching and spasms, convulsions, death |

*SLE: sea-level equivalent; increasing pressure causes increasing partial pressure and therefore the physiological effect of gas, although the percentage remains constant

### Carbon Monoxide Toxicity

The acute toxic effects of carbon monoxide at depth depend on its partial pressure and are the same at depth as at sea level. Absolute pressure has no effect on the binding of carbon monoxide to hemoglobin or on symptoms, but the increased partial pressure of oxygen at depth decreases carbon monoxide's binding to hemoglobin, somewhat lessening its effects.

Carbon monoxide contamination of compressed air is rare but dangerous when it occurs. The commonest source of contamination is an air compressor whose intake is near the exhaust of an internal combustion engine, perhaps the compressor's motor. Compressors lubricated with oil are also a potential source of carbon monoxide. A small amount of carbon monoxide is produced during the metabolism of hemoglobin, and smokers exhale even larger quantities for the first 24 hours that they are confined in a chamber. These amounts of carbon monoxide can accumulate in a saturation diving complex. Contamination of breathing air with carbon monoxide from any source prevents normal saturation of hemoglobin with oxygen. Table 30-3 lists the ranges of carboxyhemoglobin levels commonly associated with symptoms.

As 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

**TABLE 30-3**

**ACUTE EFFECTS OF INCREASED INSPIRED CARBON MONOXIDE**

| COHb* (%) | Effects |
|---|---|
| < 10 | None obvious; heavy smokers often reach this level. Subtle changes in vision and cognition have been detected by sophisticated testing. |
| 10–20 | Mild headache may be present. Skin flushing sometimes occurs. |
| 20–30 | Definite, often throbbing, headache. |
| 30–40 | Weakness, nausea, vomiting, drowsiness, dizziness, sweating, blurred vision. |
| > 40 | Unconsciousness, Cheyne-Stokes respiration, convulsions, coma, death. |

*COHb: hemoglobin in the form of carboxyhemoglobin in the arterial blood

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 pressure, is an effective treatment for carbon monoxide poisoning. The half-life of carbon monoxide is 4 to 5 hours breathing room air at rest, but this is reduced to 40 to 80 minutes breathing 100% oxygen at sea level, and to 20 minutes breathing 100% oxygen at 3 ata. The rates at which carbon monoxide is eliminated from myoglobin and cellular compartments are unknown.

### Nitrogen Narcosis

Nitrogen narcosis (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, each 1 to 2 atm increase in air pressure is said to be equivalent to one gin 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 and include increased reaction time, decreased manual dexterity, and mild impairment in reasoning. At 100 to 150 fsw (30–45 msw), most divers will become light-headed and euphoric with loss of fine discrimination. Deeper than 150 fsw (45 msw), the symptoms progress to joviality, garrulousness, and dizziness with uncontrolled laughter, loss of concentration, and mistakes in simple, practical, and mental tasks. Additional symptoms and signs include peripheral numbness and tingling and poor attention to personal safety, which is particularly hazardous to divers in the water. Responses to signals and other stimuli are slow. At 300 fsw (90 msw), mental depression, loss of clear thinking, and impaired neuromuscular coordination occur. At 350 fsw (105 msw), many divers lose consciousness. Severe narcosis may result in amnesia lasting for several hours. Sleepiness after the dive is common.

Factors that exacerbate nitrogen narcosis are inexperience, anxiety, alcohol, fatigue, and increased inspired carbon dioxide. Ameliorating factors are experience, strong will, and fixation on a task. The evidence for adaptation to narcosis with frequent exposure is limited and controversial. If it does occur, the effect appears small.

As narcosis is a threat to diver safety, most agencies limit air diving with self-contained underwater breathing apparatus (scuba) to around 130 fsw (40 msw) and with hard-hat tethered air diving to around 165 to 180 fsw (50–55 msw).

The mechanism of nitrogen narcosis, which is similar to anesthesia, is still under study. Anesthetic potency shows some relationship to the ratio of the gas solubilities in oil and water and also to their effects on surface tension. These were 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 studies, on the other hand, have focused on neurotransmitter release at presynaptic or postsynaptic sites as the active sites for anesthetic action.

### Helium and Other Inert Gases

Although the noble gases (group zero in the periodic table of chemical elements) are chemically inert, they are physiologically active. Substitution of helium for nitrogen in breathing gas has four major effects. The first, described previously, is the effect on the voice. The second is the absence of narcosis. The third is decreased work of breathing, owing to the lower density of helium. This, and the lack of narcosis, make helium particularly useful at depths greater than 150 fsw (45 msw). The fourth effect is the 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 31, Military Diving Operations and Medical Support.

Of the other noble gases, neon has been used in deep diving experiments but is not used for diving operations, as it is expensive and has a higher density than helium (or than 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.

### Hydrogen

Interest in hydrogen diving has been renewed because engineering advances in handling hydrogen–oxygen mixtures have 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 of lower density than nitrogen. The decompression properties of hydrogen appear to be between those of helium and nitrogen.

### Physiological Effects of Pressure: High-Pressure Nervous Syndrome

Because helium does not cause narcosis, it is the gas of choice for dives deeper than 150 to 180 fsw (45–54 msw). However, an effect called the high-pressure nervous syndrome (HPNS) begins at depths greater than 600 fsw (180 msw). HPNS is characterized by hyperexcitability, including tremors, poor sleep, loss of appetite, and psychosis at depths greater than 2,000 fsw (600 msw). Initially, HPNS was believed to be caused by helium per se ("helium tremors"), but subsequent experiments with liquid-breathing animals indicated that pressure is the responsible agent. Indeed, the excitatory effects of pressure and the narcotic effects of nitrogen are partially counteractive, and 5% nitrogen in a helium–oxygen breathing gas is sometimes used to ameliorate HPNS effects, which can be debilitating deeper than 1,000 fsw (300 msw).

The use of nitrogen in breathing gases for very deep diving is disadvantageous because its high density imposes ventilatory resistance and therefore reduces exercise capacity. Up to 20 bars of hydrogen have been used[16] to reduce both HPNS and breathing resistance at a record depth of 2,343 fsw (710 msw), but hydrogen narcosis, perhaps exacerbated by HPNS, appears to be a limiting factor. HPNS and low exercise capacity, particularly with UBAs, limit the maximum practical working depth to somewhere in the range of 1,500 to 2,000 fsw (450–600 msw).

## DECOMPRESSION SICKNESS

Decompression sickness (DCS) refers to the overt illness that follows a reduction in environmental pressure with the development of endogenous gas bubbles. This condition is distinguished from cerebral arterial gas embolism (CAGE), 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, as the signs and symptoms of DCS resemble not only nondiving diseases but also CAGE. Symptoms and signs may disappear spontaneously but return unpredictably hours later. DCS should be the topmost 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.

The term *decompression illness* has been suggested to be used for any diving accident involving pressure reduction, including Type I DCS, Type II DCS, and CAGE, and does not differentiate among these entities. There is a good deal of controversy over whether this terminology is advantageous.

In certain cases, it may be impossible to distinguish between CAGE and DCS with signs of cerebral dysfunction, or a combination of both, as Exhibit 30-2 illustrates. Table 30-4 provides some guidance, but differentiation is often not possible. Clinically, this is not a significant issue, as the current *US Navy Diving Manual*[7] specifies the same symptom-based therapy, including saturation therapy, for

both CAGE 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 content (eg, 50% oxygen–50% nitrogen). The depth and duration of therapy are determined by the clinical progression. Another term, Type III DCS, has also been suggested[17] as an appropriate designation to describe these very serious cases: when CAGE is suspected and both cerebral and spinal signs and symptoms of DCS are present.

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 to 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.

### 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. Diffusion is not as important as per-

**EXHIBIT 30-2**

**DECOMPRESSION SICKNESS, ARTERIAL GAS EMBOLISM, OR BOTH?**

An adult male diver was decompressing at 10 to 15 fsw after a 25-minute dive to 110 ft. A swell running had made it difficult to hold his depth, and near the end of the decompression stop (length of time unknown), he noted numbness and weakness of his right arm. After surfacing, the weakness increased, his right leg became weak, and both hands became numb. He was also dysphasic and had intermittent loss of consciousness. He was immediately given 100% oxygen and taken ashore, where he became disoriented with twitching of the muscles of the arms and left leg. On arrival at the nearest emergency room the disorientation had cleared but the other symptoms and signs remained. He was transported to a hospital with a recompression chamber. This took about 4 hours, during which oxygen and intravenous fluids were administered. On reaching the chamber, he was oriented, had mild weakness in his right arm and marked weakness in his right thigh, and now noted mild weakness in his left leg. He was initially treated in accordance with an extended US Navy Treatment Table 6[1] (see Figure 31-29 in Chapter 31, Military Diving Operations and Medical Support), near the end of which he developed bilateral loss of sensation from T-2–T-3 down, weakness of the left upper extremity, and weakness of both lower extremities. He was unable to urinate and required a Foley catheter. Over the next 2 weeks, he improved in all areas during subsequent recompression therapies, but still had mild weakness of all four limbs and required a Foley catheter for bladder drainage. Sensation at this time was normal. He was transferred for physical rehabilitation as there was no further improvement from hyperbaric oxygen treatments. The final outcome of all therapy is unknown.

The early onset of cerebral symptoms in this case is compatible with a cerebral arterial gas embolism (CAGE), which possibly occurred because of pulmonary barotrauma due to a sudden decrease in pressure from the heavy swell. The improvement with time and oxygen administration without recompression is not unusual in cases of mild CAGE. The bilateral numbness in the hands probably signaled the onset of spinal cord decompression sickness (Type II DCS), which developed into bilateral loss of sensation, quadriplegia, and loss of bladder function near the end of the first recompression. A number of factors may have contributed to the clinical presentation: the lack of adequate decompression time; the long delay to recompression, which led to further tissue damage; inadequate initial recompression therapy; and bubbles in the spinal circulation as a result of venous or arterial gas emboli from the pulmonary barotrauma or bubbles arising from elsewhere in the body, causing overwhelming DCS. The mechanisms are uncertain but appear to have involved both DCS and CAGE

(1) US Department of the Navy. *US Navy Diving Manual*. Vol 1. Washington, DC: DN; 1993. NAVSEA 0994-LP-001-9110. Rev 3.

**TABLE 30-4**

**COMPARISON OF DECOMPRESSION SICKNESS AND CEREBRAL ARTERIAL GAS EMBOLISM**

| Factor | Decompression Sickness (DCS) | Cerebral Arterial Gas Embolism (CAGE) |
|---|---|---|
| Occurrence | A dive of sufficient depth and duration to cause absorption of significant inert gas | 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 |

fusion, but it can influence gas exchange any time that 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. Diffusion between adjacent sections of tissue may lead to apparently anomalous results, as when the absorption of a tracer gas continues in one region of tissue as a result of diffusion from another region, when the tracer gas is no longer present in the inspired gas. Such effects and variations in perfusion rate make inert gas exchange a complex and unpredictable phenomenon.

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.

### Bubble Formation

In experiments with animals performed during the 1870s, Paul Bert[14] 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 and this has led to the suggestion of other etiologies, but none of these theories has been sustained. The presence of bubbles, after even very mild dives, can be detected using ultrasonic detectors.

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. Supersaturations 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). During physiological decompression, however, supersaturation rarely exceeds several atmospheres, and the lowest supersaturation at which DCS occurs is about 0.5 atm. 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 de novo nucleation that results from both viscous adhesion in tissue and shear forces during the relative motion of articular surfaces. 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). Stable gas bubbles also form as result of this process. The population of some

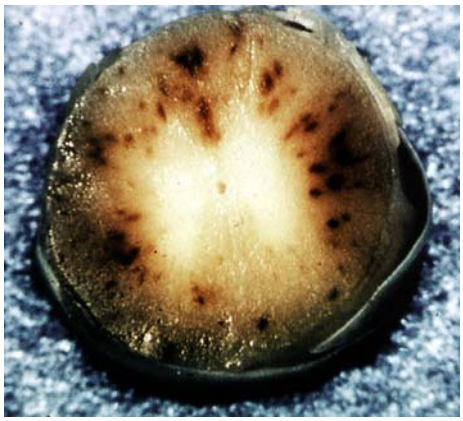

**Fig. 30-4.** This gross cross-section of a spinal cord shows the hemorrhages in the white matter that are typical of decompression sickness.

gas nuclei in the body appears to be normally 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 lungs filter small quantities of bubbles, but if the volume of gas becomes overwhelming, as can occur in accidental ascent from great depth, blood can be displaced from the heart, leading to death by asphyxia.

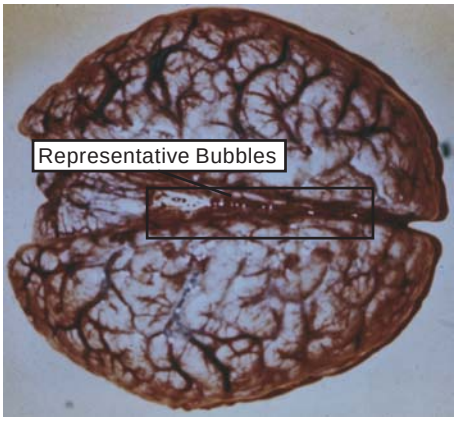


**Fig. 30-5.** Brain from a patient who died of severe decompression sickness, showing bubbles in the surface vasculature.

Venous bubbles have the potential for reaching the arterial circulation by passing through the pulmonary circulation or by-passing the lungs altogether through arteriovenous shunts or a patent foramen ovale. Venous bubbles can also pass to the arterial circulation if the number or volume of bubbles exceeds the filtering capacity of the lungs.

Large numbers of emboli in the arterial circulation can arise, of course, owing to pulmonary barotrauma, should a diver hold his breath during ascent. While this gas can have serious consequences if carried to the brain, the situation is worse if the barotrauma should occur 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 (Type III DCS), which involves both the brain and spinal cord after relatively mild dives that end with pulmonary barotrauma (Figures 30-4 and 30-5).

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, stasis, ischemia, hypoxia, edema, hemorrhage, and tissue death (Figure 30-6). En-

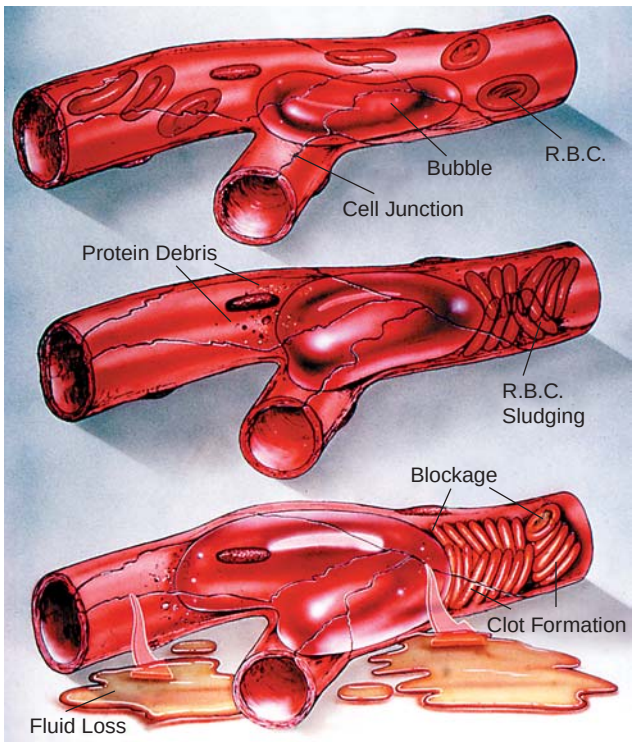


**Fig. 30-6.** Bubble formation in a blood vessel. (**a**) The lodging of a bubble at a vascular junction, with (**b**) sludging of the red blood cells (RBCs) and formation of protein debris, and (**c**) blood clot and extravascular fluid loss. Bubbles in blood vessels are typically not spherical.

dothelial cell membranes are stripped by the passage of bubbles. Bubbles may block lymph channels, causing tissue edema. The most serious of these events are rare, except in cases of severe DCS or CAGE, 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.

The biochemical effects of bubbles relate to their surface active properties, which cause enzyme activation and protein denaturation leading to thrombosis and complement activation. Together, the mechanical and biochemical effects of bubbles can increase blood viscosity, postcapillary resistance, transcapillary fluid loss, and hematocrit. These interactions can produce reperfusion injury, which occurs when toxic oxygen species such as superoxide, hydrogen peroxide, and hydroxyl radicals are generated after circulation has been re-established. Reperfusion injury may explain a poor response to hyperbaric therapy or relapse after initially successful treatment of DCS or CAGE. Relapse may 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 and signs and multiple organ involvement. Table 30-5 lists the signs and symptoms reported in a series of naval and civilian cases of DCS.[18] The five most frequent signs or symptoms reported by Rivera[18] were pain (91.8%), numbness/paresthesia (21.2%), weakness (20.6%), rash (14.9%), and dizziness or vertigo (8.5%). A more recent study by Vann and colleagues[19] of 3,150 cases of DCS in recreational divers reported to the Divers Alert Network revealed the following five most frequent signs and symptoms: numbness (56.3%), limb pain (47.0%), weakness (24.9%), dizziness (22.9%), and fatigue (21.3%). Comparison of the two studies indicates that the number of DCS cases involving pain was less in the Vann series,[19] and that the number of cases with neurological symptoms (numbness/paresthesia, dizziness, and fatigue) was greater. The reasons for these differences are not clear. In Rivera's series,[18] most of the divers were military and probably reported their symptoms and were treated earlier, thereby preventing more serious symptoms. Military divers are also under more strict 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. Half of the divers in the Vann series[19] reported past medical diagnoses and 25% reported current medical disease. Whether these contributed to causing more serious DCS has not been established. Military divers are usually in excellent physical condition and are routinely screened for medical problems.

A minor symptom, itching skin (pruritus), known as "skin bends," 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 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 with cyanotic mottling. The area may enlarge, become hyperemic, and show swelling. Marbling does not require therapy but may be a harbinger of more-severe symptoms or signs that do require treatment.

Lymphatic obstruction appears as painless local edema, 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 or may not help. 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. Pain commonly increases with motion. 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 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 or so by severe symptoms, usually paralysis of the lower extremities (see Exhibit 30-2).

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 ground-based altitude expo-

sures 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 30-5). Any CNS symptom following diving is serious, and the patient should be recompressed as soon as possible for best results.

Only about 10% of symptoms begin during decompression, and usually only after longer, deeper dives. About 45% of symptoms occur within the first hour after surfacing, with an additional 12% (approximately) in the second hour. About 85% of symptoms appear within the first 6 hours. Occasionally the onset time is longer, but few symptoms appear after more than 24 hours Even though a symptom does not occur until 24 hours or longer after diving, the patient should be presumed to have DCS until this cause is ruled out. Symptoms with very long onset times sometimes respond to recompression and should not be dismissed as not dive-related, although this probability becomes small for symptoms that appear several days after surfacing.

### *Sequela: Aseptic Bone Necrosis*

Aseptic bone necrosis is a delayed consequence of DCS that may not be evident until years after exposure (Figure 30-7). The condition is related to the occurrence of DCS and to the length of the diving career. Bone necrosis is found in 1% to 4%[8(p199)] of divers who observe standard diving practice. In divers who do not, the incidence can be as high as 50%.[20] Most of the lesions are in the midshaft of the humerus and femur, never cause symptoms, and are only identified by radiography. No treatment is required for 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.

## 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.

Diving at altitude requires reduced time on the bottom (bottom times) for no-stop decompression dives, and more decompression time for dives requiring decompression stops. The US Navy tables are not

**TABLE 30-5**

**FREQUENCY OF SIGNS AND SYMPTOMS OF DECOMPRESSION SICKNESS**

| Sign or Sympton | Percentage of Patients With a Given Sign or Symptom (n=933) |
|---|---|
| Pain | 91.8 |
| Numbness/paresthesia | 21.2 |
| Weakness | 20.6 |
| Rash | 14.9 |
| Dizziness or vertigo | 8.5 |
| Nausea or vomiting | 7.9 |
| Visual distrubances | 6.8 |
| Paralysis | 6.1 |
| Headache | 3.9 |
| Unconsciousness | 2.7 |
| Urinary disturbance | 2.5 |
| Dyspnea or "chokes" | 2.0 |
| Personality change | 1.6 |
| Agitation, restlessness | 1.3 |
| Fatigue | 1.2 |
| Muscular twitching | 1.2 |
| Convulsions | 1.1 |
| Incoordination | 0.9 |
| Disturbance of equilibrium | 0.7 |
| Local edema | 0.5 |
| Intestinal disturbance | 0.4 |
| Auditory disturbance | 0.3 |
| Cranial nerve disturbance | 0.2 |
| Aphasia | 0.2 |
| Hemoptysis | 0.2 |
| Subcutaneous emphysema | 0.1 |

Adapted with permission from Rivera JC. Decompression sickness among divers: An analysis of 935 cases. *Military Medicine: International Journal of AMSUS.* 1964;129(4):320.

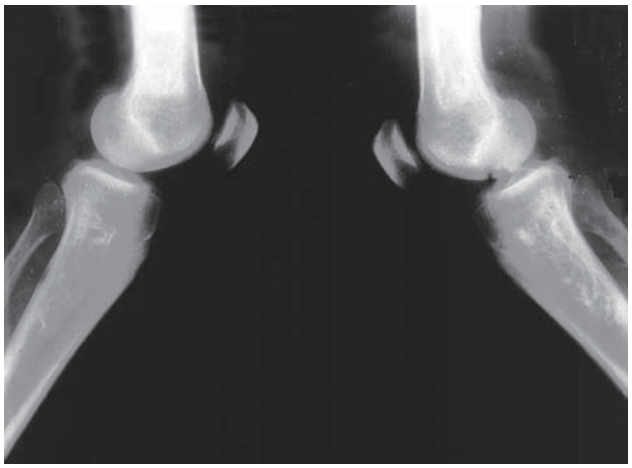

**Fig. 30-7.** A composite roentgenogram shows an abnormal pattern of calcification—shaft lesions—in both proximal tibias as a result of aseptic bone necrosis, a sequela of decompression sickness.

approved for use at altitudes higher than 2,300 ft (700 m). Because some depth gauges do not compensate for altitude, the depth of the dive should always be physically measured and the appropriate conversion made for fresh water (1 atm = 34 fsw). Decompression tables developed by Boni and colleagues[21] for use at altitude have received limited testing. No other tested tables exist. Ad hoc guidelines for adjusting sea level schedules for altitude have also been published in the recreational diving literature, but the adjustments simply increase the actual depth by a fraction to arrive at a corrected depth for table selection. There is no documentation of the efficacy of this method. Diving should be delayed for at least 12 hours after arriving at a high dive site to allow the nitrogen absorbed in the body to equilibrate to the reduced pressure. Without equilibration, the situation is similar to making a repetitive dive after decompression from a saturation dive at 1 ata.

Flying is common after recreational diving trips that may have lasted several days and involved many dives. During the subsequent flight home in a commercial aircraft, a diver may be exposed to a cabin altitude of as much as 8,000 ft (2,438 m), the maximum altitude permitted by the Federal Aviation Administration. To avoid DCS as a result of flying after diving, a diver must wait at sea level a sufficient time to allow excess nitrogen from diving to leave his body. A number of recommended preflight surface intervals have been published for single dives with no underwater stops for decompression; for example, 2 hours, US Navy; 12 hours, Divers Alert Network; and 24 hours, US Air Force.[22] The wide range of these guidelines indicates how few experimental data are available on which to base them. Recent experiments,[22] however, suggest that 2 hours is too short, while 12 hours may be satisfactory for some single dives and 24 hours conservative. Following multiple dives, on the other hand, a surface interval of at least 17 hours may be needed.

If air evacuation of a diving casualty is required, an aircraft capable of pressurization to 1 atm should be used. If such an aircraft is unavailable, and particularly for helicopter evacuation, the cabin altitude should not exceed 800 ft (242 m), and the diver should receive 100% oxygen during the flight. The administration of 100% oxygen is recommended during all types of transport of all casualties as a standard practice. The administration of 100% oxygen (not less) is particularly important as first aid for diving accidents.

### 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. 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 shallow dives. Repetitive diving (more than one dive in 12–24 h) may increase the risk of DCS.

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 (ie, 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. Immer-

sion and exercise at pressure have been demonstrated[23] to increase gas uptake and DCS risk. When multiple factors exist that change during a dive, the results may be difficult to anticipate. Mild exercise during decompression appears beneficial but 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[23] indicates that exercise after decompression from diving or to altitude increases the DCS incidence. Note the distinction between exercise during and after decompression (or while breathing oxygen before altitude 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.

There is no demonstrated relationship between physical fitness and DCS risk. A high level of physical fitness, however, is essential for the diver to be able to manage the rigors of the underwater environment and deal with occasional emergencies.

Increasing age increases the risk of DCS. This has been variously reported[23] to be 2- to 9-fold higher in older (> 45 y) individuals; the lower figure appears to us to be more reasonable. The age effect may be related to decreased inert gas exchange efficacy and increased formation of gas nuclei.

Early observations of humans and animals indicated that obesity increases the risk of DCS, presumably because inert gas is more soluble in fatty than in aqueous tissue. These observations have not held up in recent studies,[23] however, perhaps because modern diving exposures are less severe than earlier exposures.

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. 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[24] shows that the incidence of DCS is greatest during the first few days of repeated exposure to increased pressure. This may result from a depletion of the gas nuclei that appear to be the origin of bubbles. No acclimatization effect has been found in air diving, but it may be present in helium–oxygen diving.[25]

Several studies[26] suggest that women have a somewhat higher risk for DCS than men, both in aviation and during hyperbaric exposure. One study[27] showed that, at least in exposures to high altitudes, there was no difference between genders for DCS suseptibility. Whether the perceived differences are of clinical or operational significance is unknown at present, as are the causes of these differences, if they are real.

Other factors that have been proposed but not proven to increase the risk of DCS are previous injury (trauma or previous DCS), body position during decompression, increased carbon dioxide in the breathing gas, and fatigue.

## 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 had to be used to prevent injury. Not until after the turn of the 20th century in England, when J. S. Haldane[28] began studying the problem systematically, were decompression schedules as we know them today constructed. Haldane realized that the manner of decompression used at the time, steady decompression at rates between 1.5 and 5 fsw/min, was too slow at deeper depths and too rapid at shallow. This allowed more inert gas to be absorbed at depth and not 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. Most were derived from empirical data with little testing in water, and testing has usually been done with only physically fit young men as subjects. Some schedules or parts of schedules have been tested and the rest calculated mathematically to fill in the gaps. Different environmental conditions and individual physiology were not considered. Nevertheless, current procedures make DCS a rare event and diving a relatively safe activity. Estimates of DCS incidence during actual diving are uncertain because record keeping is difficult. The DCS incidence when using the US Navy diving tables,[7,11] which all US military diving operations are required to use, is about 0.1%. We have reviewed various reports and, depending on the types of diving, commercial diving companies have had a rate of 0.1% to 3% in the past. The current rate of DCS in commercial diving in the United States is about 0.1% or less.[29]

### *Dive Computers*

A dive computer measures the water pressure and automatically computes the diver's decompression requirement. Early pneumatic and analog dive computers were unreliable and were not widely accepted. Improvements in pressure transducer and digital technology coupled with the increasing popularity of recreational diving in the 1980s have made reliable mass-produced dive computers possible at reasonable cost. (Dive computers and computer models are also discussed and illustrated in Chapter 31, Military Diving Operations and Medical Support; see Figures 31-19 and 31–24.

The first commercially successful digital dive computer was tested without incident in 1983 during 110 chamber dives. Many mass-produced dive computers are now available, their programs all using variants of the Haldane decompression algorithm, but no further decompression trials of dive computers have been conducted. The decompression "safety" of computer model and table diving is debated vigorously, but accident reports do not offer compelling evidence for a higher DCS incidence with computers. Accident data[30] compiled by the Divers Alert Network indicate that DCS occurs after deeper dives and after more multilevel and decompression dives with computers, but such data are insufficient for quantifying DCS risk.

Dive computers minimize errors in table selection by accurately tracking actual depth–time profiles. Also, because table selection no longer depends on using the deepest depth of the dive for the entire dive time as when using the standard tables, time underwater can usually be increased without a significant increase in decompression time. Therefore, dive computer models often provide very different diving guidelines than the US Navy tables.

### *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.*

Establishing acceptable risk is a two-part process. The first part determines the probability and severity of an injury and is a scientific problem that diving researchers are only beginning to understand. The second part decides what level of risk is acceptable and is a political activity. An individual might choose an acceptable risk for himself or might rely on the judgment of an organization such as the US Navy or the National Oceanic and Atmospheric Agency.

## ADDITIONAL THREATS TO DIVERS

### Thermal Stress

Divers are exposed to both heat and cold, although cold is a far more common environmental stress, and diving is one of only a few situations in which healthy, active individuals may suffer serious hypothermia. This section discusses thermal stress only in divers. Immersion hypothermia in the wider population is dis-

cussed in detail in Chapter 18, Cold Water Immersion, in *Medical Aspects of Harsh Environments, Volume 1*.[31]

For a nude human at rest, the thermal comfort zone during immersion lies between 30°C and 35°C, depending on the individual's amount of subcutaneous fat. Therefore, diving in even tropical water (20°C–25°C) allows the body to lose heat faster than it can be generated. This is because the thermal conductivity and specific heat of water are, respectively, 25- and 1,000-fold greater than those of air. For the unprotected diver, it is cold rather than decompression that usually limits the dive time. Divers will be comfortable and require minimal protective clothing during several hours of moderate work at water temperatures of 23°C to 30°C. Thermal protection is required in water colder than 23°C.

The first indication of impending hypothermia is the feeling of being uncomfortably chilly. As hypothermia progresses, the hands become insensitive and manual dexterity decreases. With continuing exposure, the core temperature drops, the body increases heat production by shivering, the respiration rate rises, and gas consumption increases. Subsequently, coordination degenerates, thinking becomes difficult, and unconsciousness follows.

The most common thermal protection is passive, as with a wetsuit that traps a stagnant layer of warm water to prevent cold water from washing over the diver's skin. Wetsuits are made of closed air-filled cells that have the disadvantage of collapsing and providing less insulation as the water pressure increases. The drysuit is another passive system that allows no water to contact the diver's skin; it is commonly worn over underwear, which provides additional insulation. Wetsuits and drysuits are used by free-swimming divers. Tethered divers can have the advantage of active heating provided from the surface, and the hot-water wetsuit is common in military and commercial diving. A hot-water wetsuit fits the diver loosely, allowing hot (105°C) water to flow over the body. The hot water is exhausted through the wrist and ankle seals into the gloves and boots to keep the hands and feet warm. Close control of the water temperature is important to prevent skin burns.

Divers breathing helium–oxygen deeper than 300 fsw (91 msw) also lose heat through respiration even if the skin is comfortably warm. The breathing gas must be heated to avoid this problem.

Although the condition is much less common than hypothermia, divers can become hyperthermic while working in water warmer than 35°C, such as in the outflow of a power plant or the Red Sea. Under these conditions, the temperature difference between the water and the diver's body is insufficient to eliminate metabolic heat by conduction or convection. Hyperthermia can also occur in a suited diver or a recompression chamber in a hot environment. Two saturation divers died of hyperthermia in the late 1970s or early 1980s when their chamber overheated (—JV, Jr, personal information). Shading the chamber or spraying cool water on it can reduce this problem.

The treatment for hypothermia or hyperthermia following diving is no different than that used to treat these conditions arising from other circumstances.

**Underwater Blast Injury**

Even in peacetime, divers work with explosives and may inadvertently be exposed to underwater blast. Blast is more dangerous in water than in air because the greater density and incompressibility of water allow the shock waves to travel further and lose less energy. A shock wave is reflected by tissue in air but is transmitted underwater. If the transmitted wave reaches a gas-filled cavity in the human body, the wave's energy is dissipated at the interface and great injury can occur. Multiple factors affect the severity of injury, including the size and type of explosive, thermal profile of the water, bottom composition, underwater banks or shoals, a nearby ship or structure, reflection from the sea bottom and the surface, and location of the explosive in the water column relative to the position of diver (Figure 30-8).

The out-rushing gas from an underwater detonation produces a shock wave called the initial pulse, which is characterized by a sudden pressure rise with exponential collapse. The initial pulse is followed by second and third expansions called the subsidiary and the bubble pulses. These cause less injury than the initial pulse unless they are reinforced by reflection from the bottom, the surface, or nearby structures. They may also cancel each other out.

Calculation of peak shock wave pressures is controversial and requires knowledge of empirical constants for each explosive. There is general agreement that injury is unlikely for peak shock wave pressures lower than 50 psi (< 345 kPa). Injury is likely for peak pressures between 50 and 500 psi (345–3,447 kPa), whereas serious injury occurs at 500 to 2,000 psi (3,447–13,790 kPa), and death is certain at pressures higher than 2,000 psi (> 13,790 kPa).

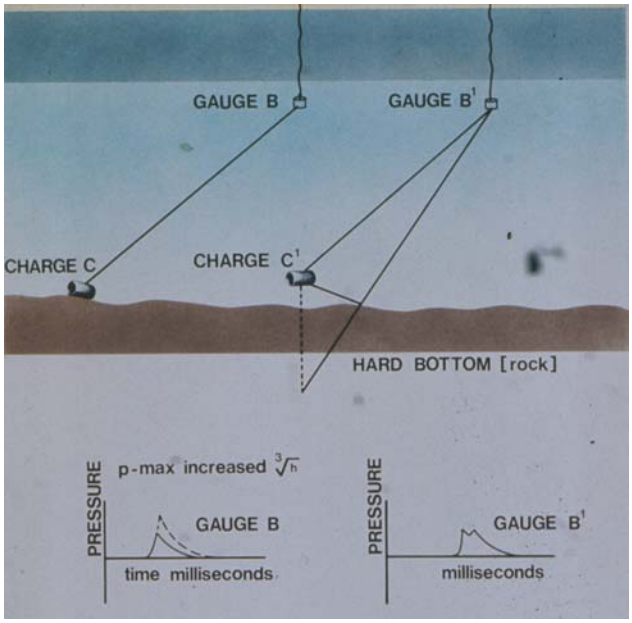


**Fig. 30-8.** The difference produced in a pressure waveform by moving an explosion from the hard, rock bottom of a waterway (charge C) to slightly above the bottom (charge $C^1$). Because of the reflection of the original pressure wave from the hard, rock bottom (waveform, bottom right), a diver located in the position of pressure gauge $B^1$ (top) would experience two pressure peaks from an explosion at charge $C^1$. Compare this waveform with the one that a diver at gauge B would receive from an explosion at charge C (waveform, bottom left). Adapted with permission from Wakeley CPG. Effect of underwater explosives on the human body. *Lancet*. 1945;June 9:715.

Injury occurs by tissue shear in air-filled organs and by motion in solid organs. A shock wave travels through water and tissue at the same speed but compresses on reaching a tissue–gas interface (eg, in the middle ear, lungs, intestines) and tears (or "spalls") the tissue. The sudden motion of tissue by abdominal compression may damage solid organs, but only rarely.

Blast injury to the lung results from transmission of the shock waves through the chest rather than through the airways, with alveolar disruption, lacerations, and pinpoint-to-massive hemorrhage. A blast casualty may exhibit dyspnea, chest pain, and slowed breathing. Complications of lung damage include pulmonary edema and pneumonia. Lung damage can affect the heart, circulation, and nervous system. Vagal reflex may cause a transient bradycardia or asystole. Myocardial damage may occur due to gas embolism of the coronary arteries. There may be hemothorax or pneumothorax, mediastinal emphysema, and contraction of the pulmonary capillary bed with an increase in pulmonary capillary pressure. Hemorrhages in the mucosa of the trachea and bronchial tree are common. Severe lung damage may lead to circulatory collapse. The CNS may be affected by transmission of the pressure to the cerebrospinal fluid, causing rupture of blood vessels in the brain. Other CNS damage may occur from gas embolism or hypoxia, owing to circulatory dysfunction. Transient paralysis, headaches and disorientation, delirium, amnesia and other symptoms of CNS damage can occur.

The gastrointestinal tract is thought to be more sensitive to blast injury than the lungs. The abdomen may be distended and silent. Intestinal vessels rupture, causing microhemorrhages in the gut wall and large bands of hemorrhage in the wall or massive hemorrhage into the lumen. Paralytic ileus can be present. The gastrointestinal tract can rupture and peritonitis ensue. Liver contusion probably occurs only in the most severe cases. Rectal bleeding may be the first sign of gastrointestinal problems.

Injury to the upper airway and the sinuses is not common, but there are records of tympanic membrane rupture and ossicle derangement, so the ear should not be overlooked as a site for damage.

A victim of underwater blast with no apparent injury should remain under observation, as late complications may occur. Treatment is the same as for acute total body trauma. All standard measures should be undertaken to combat shock and maintain respiration. Oxygen should be administered. Abdominal surgery must be considered if the clinical signs point to severe abdominal distress. Appropriate laboratory studies should be done as soon as possible. If cerebral or coronary gas embolism is suspected, recompression therapy must be considered, but this will require air evacuation to a medical center that can provide critical care in a recompression chamber.

**Drowning and Near-Drowning**

Drowning is defined as death from suffocation caused by immersion in a liquid, usually water. Near-drowning is defined as survival, at least temporarily, after suffocation by immersion. Three immersion suffocation syndromes are recognized: wet, dry, and salt-water aspiration. About 85% of drowning or near-drowning victims have aspirated water into their lungs (ie, the lungs are "wet"). The remaining 15% are "dry." In dry suffocation, the casualty becomes hypoxic because of laryngospasm

or breath-holding. Death from the respiratory distress syndrome after a near-drowning is known as *secondary* drowning and is the cause of death in 10% to 25% of cases. In the third syndrome, called the salt-water aspiration syndrome, the severity of the effects of immersion is not sufficient to classify the case as one of near-drowning. These three syndromes are part of a common process with gradations in severity that depend on the extent of the pathophysiological changes.

The early stages of drowning and near-drowning are the same. During initial submersion or loss of respirable gas, breath-holding continues until the increasing concentration of arterial carbon dioxide forces inspiration or causes unconsciousness. If aspiration with laryngospasm occurs, the lungs will remain dry. Water is often swallowed at this point and may induce vomiting with aspiration of water or vomitus. As hypoxemia progresses, unconsciousness, arrhythmia, and circulatory arrest follow. However, most casualties involuntarily inspire water and will die if not immediately removed from the water. Survival is possible with adequate treatment. Factors associated with a poor prognosis after rescue are the following:

- age less than 3 years,
- submersion longer than 5 minutes,
- resuscitation delayed for longer than 15 minutes after rescue,
- seizures with posturing and fixed or dilated pupils,
- cardiopulmonary resuscitation required on admission to the emergency department,
- arterial pH lower than 7.10, and
- persistent coma.

The pathophysiology of drowning and near-drowning can be complex. Bradycardia and peripheral vasoconstriction may occur due to the mammalian diving reflex. Recovery is usually rapid if no fluid is aspirated. If more than 22 mL of water per kilogram of body weight have been aspirated, electrolyte changes may occur. Near-drowning victims will usually have aspirated less than 4 mL/kg, so electrolytes are usually within the normal range. Aspirated fresh water is absorbed rapidly through the alveolar membranes and can cause transient hypervolemia. Fresh water decreases pulmonary surfactant, leading to poorly ventilated or unventilated alveoli. Salt-water aspiration causes increased osmolality, with rapid fluid movement into the alveoli and loss of surfactant. The result in either case is hypoxemia. With aspiration of as little as 1 to 3 mL of fluid per kilogram of body weight, the arterial oxygen tension can fall to 50% or less.

The clinical picture is largely a result of hypoxia or anoxia. The CNS may be undamaged, or the patient may show a decorticate or decerebrate response. Symptoms and signs of pulmonary pathology include dyspnea, retrosternal chest pain, frothy sputum (may be blood-tinged), tachypnea, cyanosis, crepitations, rales, and rhonchi. Chest roentgenograms may be normal or show patchy infiltrates or evidence of pulmonary edema. Adult respiratory distress syndrome, pneumonitis, bronchopneumonia, pulmonary abscess, and empyema may be secondary or late complications. Common cardiac problems are supraventricular rhythms or other dysrhythmias, hypotension, and shock. Cardiac output may be low, and failure of the right side of the heart may occur, owing to increased pulmonary vascular resistance. Other complications include decreased urinary output, renal failure, hemolysis with hemoglobinuria, and disseminated intravascular coagulation.

The single most important factor influencing long-term survival is immediate life support. Cardiopulmonary resuscitation, using 100% oxygen with positive pressure, should start as soon the casualty is removed from the water. If the patient is unconscious and appears to have cardiac arrest, intubation is recommended with administration of intravenous fluids. If hypothermia is present, further loss of body heat should be prevented until active rewarming can be done, if required by the degree of hypothermia. Drowning victims should be transferred to a medical treatment facility as soon as possible and placed on 100% oxygen even if they are in no apparent difficulty. Serious complications often arise up to 24 hours after near-drowning. As there are "miraculous" recoveries after 40 minutes of submersion, cardiopulmonary and other resuscitative measures should be continued until normal body temperature has been restored and death is without question. Factors that improve the chances of recovery from prolonged immersion include hypothermia, laryngospasm with gas in the lungs, and continuing pulmonary gas exchange despite fluid in the lungs. Hypothermia has a well-known protective effect against hypoxia, and when present, death cannot be assumed until the casualty has been rewarmed to normal body temperature.

Salt-water aspiration syndrome refers to events beginning with aspiration of small amounts of salt water, as may occur during buddy-breathing, breathing through a defective regulator, air-breathing on the

surface, or snorkeling. The initial symptom is coughing with or without sputum. A latent period of 1 to 15 hours (most commonly 1–2 h) follows, after which the cough returns with dyspnea, retrosternal pain on inspiration, a feverish feeling, malaise, headache, anorexia, tachycardia, and shivering. The shivering may sometimes be severe and may be alleviated by a hot shower. Generalized aches and pains may be present. Signs include a mildly increased temperature, elevated heart rate, and generalized or localized rhonchi in the chest. The chest roentgenogram may show patchy consolidation or increased pulmonary markings. During the first 6 hours, the forced expiratory volume in 1 second and vital capacity may be decreased. Blood gases may show a decreased arterial oxygen partial pressure with normal or low arterial carbon dioxide partial pressure while breathing air. These signs and symptoms rarely continue for more than 24 hours. Treatment consists of oxygen administration to alleviate the pulmonary signs and symptoms, rest, and warmth. The major difficulty with the salt-water aspiration syndrome is differentiating it from other causes, including a lung or other infection, DCS, pulmonary barotrauma, or the effects of immersion in cold water. The diagnosis can be made on the basis of the confirmation of aspiration by the diver, a latent period between initial coughing and later symptoms, a rapid response to oxygen administration without recompression, and delayed symptom onset that is not compatible with the rapid onset of pulmonary barotrauma or gas embolism.

Pulmonary edema without aspiration of water has been reported[32] and termed *cold-induced pulmonary edema,* because all of the original cases appeared to be associated with diving in water temperatures of less than 12°C. However, the same authors reported a case that occurred in 27°C water, and reported several cases in divers who were in cold water but fully protected with drysuits. These same authors also reported multiple episodes in some divers. The cause for this is unknown. In the divers studied, no cardiac disease was found and there was no history of severe exertion. Clinically, severe dyspnea occurs at depth and is associated with cough, weakness, chest discomfort, wheezing, hemoptysis, expectoration of froth, and other usual symptoms of pulmonary edema. Signs include rales and roentgenographic evidence of pulmonary edema. The episodes last from 24 to 48 hours and may require standard therapy for pulmonary edema, although cases have resolved without treatment.

## SUMMARY

Since the earliest days of breath-hold diving, the underwater environment has posed unique risks due to large pressure changes, hypoxia, and drowning. Immersion also distorts human vision and hearing. The introduction of compressed air and artificial gas mixtures extend diving range and duration but also create 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 high-pressure nervous syndrome.

Early work with compressed air produced syndromes (called "caisson disease" or "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, and the now-supersaturated tissues and blood tend to form bubbles, which underlie clinical problems ranging from cutaneous mottling to joint pain and a range of neurological problems. In addition, leakage of expanding gas from the pulmonary system into the circulation (arterial gas embolism) can lead to circulatory collapse and/or 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 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 wash out inert gases. All military physicians, and not just those practicing Special Operations medicine, should be aware of the pathophysiology, prevention, and, if necessary, treatment of the unique hazards found in the underwater environment.

## REFERENCES


1. Herodotus. *Urania*. Vol 3, Book 8. Beloe W, trans. New York, NY: Harper Bros; 1844. Cited by: Larson HE. *A History of Self-Contained Diving and Underwater Swimming.* Washington, DC: National Academy of Sciences, National Research Council: 1959: 5.

2. Beebe W. *Half Mile Down.* New York, NY: Duell, Sloane, and Pierce; 1934. Cited by: Larson HE. *A History of Self-Contained Diving and Underwater Swimming.* Washington, DC: National Academy of Sciences, National Research Council: 1959: 5.

3. Bachrach AJ. A short history of man in the sea. In: Bennett PB, Elliott DH, eds. *The Physiology of Diving and Compressed Air Work.* 2nd ed. Baltimore, Md: Williams & Wilkins; 1975: Chap 2.

4. Molenat FA, Boussages AH. Rupture of the stomach complicating diving accidents. *Undersea Hyperb Med*. 1995;22:87–96.

5. Waite CL. *Case Histories of Diving and Hyperbaric Accidents.* Bethesda, Md: The Undersea and Hyperbaric Medical Society; 1988: 52.

6. Greene KM. *Causes of Death in Submarine Escape Training Casualties: Analysis of Cases and Review of Literature.* Alverstoke, Gosport, England: Admiralty Marine Technology Establishment; 1978. AMTE(E) Report R78-402.

7. US Department of the Navy. *US Navy Diving Manual*. Vol 1. Washington, DC: DN; 1993. NAVSEA 0994-LP-001-9110. Rev 3.

8. Edmonds C, Lowry C, Pennefeather J. *Diving and Subaquatic Medicine*. 3rd ed. Oxford, England: Butterworth-Heinnemann; 1992.

9. Dutka AJ, Polychronides JE, Mink RB, Hallenbeck JM. *The Trendelenburg Position After Cerebral Air Embolism: Effects on the Somatosensory Evoked Response, Intracranial Pressure and Blood Brain Barrier.* Bethesda, Md: Naval Medical Research Institute; 1990. Report 90-16.

10. Evans DE, Catron PW, McDermott JJ, et al. Effect of lidocaine after experimental cerebral ischemia induced by air embolism. *J Neurosurg.* 1989;70:97–102.

11. US Department of the Navy. *US Navy Diving Manual.* Vol 2. Washington, DC: DN; 1991. NAVSEA 0994-LP-001-9020. Rev 3.

12. Smith JL. The pathological effects due to increase of oxygen tension in the air breathed. *Journal of Physiology, London.* 1899;24:19–35.

13. Wright WB. *Use of the University of Pennsylvania Institute for Environmental Medicine Procedures for Calculation of Pulmonary Oxygen Toxicity.* Panama City, Fla: US Navy Experimental Diving Unit; 1972. NEDU Report 2-72.

14. Bert P. *Barometric Pressure.* Hitchcock MA, Hitchcock FA, trans. Columbus, Ohio: College Book Co; 1943: 709–754, 859–890. Originally published in 1878.

15. Vann RD. Nitrox diving data review. In: Lang M, ed. *Diver's Alert Network Nitrox Workshop Proceedings.* Durham, NC: DAN; 2000: 19–29.

16. Lafay V, Barthelemy P, Comet B, Frances Y, Jammes Y. EDB changes during the experimental human dive HYDRA 10 (71 atm/7200 kPa). *Undersea Hyperb Med.* 1995;22:51–60.

17. Neuman TS, Bove AA. Combined arterial gas embolism and decompression sickness following no-stop diving. *Undersea Biomed Res.* 1990;17:429–436.

18. Rivera JC. Decompression sickness among divers: An analysis of 935 cases. *Mil Med.* 1964;129(4):314-334.

19. Vann RD, Bute BP, Uguccioni DM, Smith LR. Prognostic indicators in DCI in recreational divers. In: Moon R, Sheffield P, eds. *Treatment of Decompression Illness: 45th Undersea & Hyperbaric Medical Society Workshop.* Kensington, Md: The Undersea and Hyperbaric Medical Society; 1996: 352–363.

20. Oiwa H, Itoh A, Ikeda T, Sakurai S. Osteonecrosis of the long bones in diving fishermen. In: *Proceedings of the 9th International Symposium on Underwater and Hyperbaric Physiology.* Bethesda, Md: The Undersea and Hyperbaric Medical Society; 1987: 893–902.

21. Boni MM, Schmidt R, Nussberger P, Buhlmann AA. Diving at diminished atmospheric pressure: Air decompression tables for different altitudes. *Undersea Biomed Res*. 1976;3:189–204.

22. Vann RD, Gerth WA, Denoble PJ, Sitzes CR, Smith LR. A comparison of recent flying after diving experiments with published flying after guidelines. Proceedings of the Annual Scientific Meeting, The Undersea and Hyperbaric Medical Society, May 1996; Bethesda, Md. *Undersea Hyperb Med.* 1996;23(suppl):36.

23. Vann RD, Thalmann ED. Decompression physiology and practice. In: Bennett PB, Elliott DH, eds. *The Physiology and Medicine of Diving.* 4th ed. Philadelphia, Pa: WB Saunders; 1993; Chap 14.

24. Hempleman V. History of decompression procedures. In: Bennett PB, Elliott DH, eds. *The Physiology and Medicine of Diving*. 4th ed. Philadelphia, Pa: WB Saunders; 1993: Chap 13.

25. Eckenhoff RG, Hugher JS. Acclimatization to decompression stress. In: Bachrach AJ, Matzen MM, eds. *Proceedings of the 8th Symposium on Underwater Physiology.* Bethesda, Md: Undersea Medical Society; 1984: 93–100.

26. Taylor MB. Women and diving. In: Bove AA, Davis J, eds. *Diving Medicine.* 2nd ed. Philadelphia, Pa: WB Saunders; 1990: Chap 13: 157.

27. Webb JT, Pilmanis AA, Kraus KM, Kannen N. Gender and altitude-induced decompression sickness susceptibility. *Aviat Space Environ Med*. 1999;70:364.

28. Boycott AE, Damant GDD, Haldane JS. The prevention of compressed air illness. *J Hyg (Cambridge)*. 1908;8:342–443.

29. Reedy J. Past President, Association of Diving Contractors, Labadieville, La. Personal communication, March 2001.

30. Diver's Alert Network. *Report on Decompression Illness and Diving Fatalities.* Durham, NC: DAN; 1997.

31. Pandolf KS, Burr RE, Wenger CB, Pozos RS, eds. *Medical Aspects of Harsh Environments, Volume 1.* In: Zajtchuk R, Bellamy RF, eds. *Textbook of Military Medicine.* Washington, DC: Department of the Army, Office of The Surgeon General, and Borden Institute; 2001. In press.

32. Hampson NB, Dunford RG. Pulmonary edema of scuba divers. *Undersea Hyperb Med.* 1997;24:29–33.

## RECOMMENDED READING

Shilling CW, Carlston CB, Mathias RA. *The Physician's Guide to Diving Medicine.* New York, NY: Plenum Press; 1984.