Chapter 31

MILITARY DIVING OPERATIONS AND MEDICAL SUPPORT

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INTRODUCTION

Divers breathe gases and experience pressure changes that can cause different injuries from those encountered by most combatant or noncombatant military personnel. This chapter places diving hazards and the therapy of diving casualties in historical and operational context. There are no formal data from which accurate estimates of diving populations can be estimated, but recreational dive training statistics suggest there are currently some 2 to 4 million active divers in the world, most participating in sport or recreation. Those of concern in this chapter are the 4,000 to 5,000 divers of the US military, a number that appears to be in decline since the end of the Cold War. An informal survey suggests there are some 5,000 to 10,000 military divers in other nations of the world.

The US Navy has responsibility for all diving by US forces, and most US military divers are in the Navy. All diving operations by US forces must be conducted in accordance with the *US Navy Diving Manual*[1] and related directives. As of this writing, approximately 3,000 Navy divers typically conduct some 100,000 dives annually. About 1,500 divers are in diving and salvage and 1,500 are in Naval Special Warfare (NSW). Diving and salvage forces include four ARS-50 *Safeguard*-class salvage ships, Explosive Ordnance Disposal (EOD) units, two Mobile Diving and Salvage Units (MDSUs), two Underwater Construction Teams (UCTs), a Consolidated Diving Unit, and teams assigned to submarine tenders and shore-based ship repair facilities. The diving and salvage forces conduct salvage, search and recovery, underwater mine clearance, underwater construction, security inspections, and ships husbandry tasks such as hull cleaning and maintenance.

Salvage divers receive basic training at the Navy Diving and Salvage Training Center (NDSTC), Panama City, Florida, and some qualify in the Mark (Mk) 21 Mod 1 mixed-gas diving helmet (Figure 31-1) to a depth of 90 meters of seawater (msw; equivalent to 300 feet of seawater [fsw]). EOD divers receive basic training at the Explosive Ordnance Disposal School, Indian Head, Maryland, and later qualify in the Mk 16 Underwater Breathing Apparatus (UBA), which is used for mine clearance. About 100 US Navy divers specialize in saturation diving using Personnel Transfer Capsules (PTCs) and Deck Decompression Chambers (DDCs).

Of the 1,500 combat swimmer and divers in Naval Special Warfare, most are in six SEAL (*se*a, *a*ir, *l*and) teams and two SEAL delivery vehicle (SDV) teams. SEALs are trained for reconnaissance and direct action missions at rivers, harbors, shipping, and coastal facilities in restricted or denied waters. SEAL divers operate from surface craft, submerged submarines, and miniature wet submersibles (which are SDVs). Insertion by fixed or rotary wing aircraft is also possible. All SEALs qualify in open-circuit and closed-circuit oxygen scuba (self-contained underwater breathing apparatus), and those assigned to SDV teams receive additional training in the Mk 16. SEALs are part of the Special Operations Forces that include about 1,000 US Army, Air

Fig. 31-1. Both divers are wearing the US Navy Mark (Mk) 21 Mod 1 Underwater Breathing Apparatus for helium–oxygen diving to 90 msw (300 fsw). Reprinted from US Department of the Navy. *US Navy Diving Manual.* SS521-Ag-PRO-010. Washington, DC: Naval Sea Systems Command. Rev 4 (20 Jan 1999); Change A (1 Mar 2001): p13-12. NAVSEA 0994-LP-100-3199.

Force, and Marine Corps divers, who have narrower training and missions and dive less frequently. SEALs train at the Naval Special Warfare Center (NSWC), Coronado, California, while divers from other services train at NDSTC, Panama City, or at the US Army Underwater Swim School, Key West, Florida.

Other military divers include about 100 from the US Air Force for rescue, about 100 from the US Army for port facility maintenance, and fewer than 50 from the US Coast Guard for rescue and pollution response. The military also employs civil service divers and contracts with commercial diving companies for specific projects. Military personnel and dependents with military or civilian diving training are an unofficial military diving population who participate in recreational diving and can develop diving-related illnesses not in the line of duty.

Training in diving medicine for military physicians is conducted by the US Navy for all US forces and occasionally for foreign militaries. Instruction includes 6 weeks of practical diving training and 3 weeks of diving medicine in Panama City. Navy Undersea Medical Officers (UMOs) receive 12 weeks of additional training in submarine-specific issues at the Naval Undersea Medicine Institute (NUMI), New London, Connecticut. Training is followed by an operational tour with a submarine squadron, fleet diving unit, or NSW command. Operational tours are typically succeeded by assignment to administrative positions, graduate education, research at the Navy Experimental Diving Unit (NEDU), Panama City, or medical specialty residency training. There are fewer than 100 UMOs on active duty but others are in the reserves. Diving medicine training for noncommissioned officers is conducted by the Navy in Coronado and Panama City and by the Army in Key West.

The unique medical support requirements for diving are a result of the physiological, engineering, and environmental challenges present underwater. These challenges are nicely illustrated by examples from diving history. Breath-hold is the simplest form of diving and although uncommon in current military operations except for submarine escape training, its physics and physiology are a good basis for understanding many of the respiratory and circulatory adjustments to UBAs. A UBA extends the time at elevated pressure leading to the potential for hypothermia, nitrogen narcosis, oxygen toxicity, carbon dioxide poisoning, arterial gas embolism (AGE), and decompression sickness (DCS). Military diving physicians require broad training for the wide range of missions they may support, but military physicians may also consult concerning civilian diving casualties, as many military personnel or their dependents are recreational divers. In addition to these hazards, divers must cope with threats posed by various forms of marine life. Medical officers need to be mindful of all the problems, not the respiratory ones exclusively, that military divers and swimmers may encounter in the marine environment.

BREATH-HOLD DIVING

Breath-hold diving, or free diving, requires little equipment and provides examples of many physical and physiological constraints of the underwater environment. Free diving was an essential operational technique in the Pacific Theater of Operations during World War II (Exhibit 31-1) and remains a fundamental skill for present-day combat swimmers.

Figure 31-2 shows pressures to which a diver must adapt during descent. The pressure at sea level is 1 atmosphere absolute (1 ata; 14.7 pounds per square inch [psi] absolute [psia]). With each 10 msw (33 fsw) of descent, a diver is exposed to an additional atmosphere of pressure. The forward escape trunk of a submarine during lock-out and lock-in operations of combat swimmers (ie, entering and leaving a submarine via an escape trunk that can be flooded or drained and the pressure increased or decreased; discussed below in Submarine Rescue and Escape) is at about 9 msw (30 fsw; 1.9 ata [which refers to the absolute, or total, pressure; atm, on the other hand, refers to the partial pressure of gases]). The standard depth for treating DCS (see Chapter 30, Physics, Physiology, and Medicine of Diving) is 18 msw (60 fsw; 2.8 ata). For diving with scuba in the US Navy and in civilian recreational diving, the usual depth limit is 39 msw (130 fsw; 4.9 ata).

The breath-hold diver must anticipate and compensate for the compression of gas-containing spaces in the body that occurs during descent according to Boyle’s law: the volume of a given mass of gas varies inversely with absolute pressure when temperature is held constant (the gas laws are described in Chapter 30, Physics, Physiology, and Medicine of Diving). The middle ear requires active inflation to avoid otic or aural barotrauma (ear squeeze), which is the most common medical prob-

EXHIBIT 31-1

AMPHIBIOUS WARFARE IN THE PACIFIC

When the landing craft bottomed-out and the bow ramps dropped on the island of Tarawa on November 20, 1943, US Marines waded off a barrier reef and into a deep lagoon. Many drowned and more were killed or wounded during the long walk to the beach. This disaster gave rise to the Underwater Demolition Teams (UDT), whose mission was to chart off-shore waters and destroy natural or man-made obstacles that could impede an invasion force.[1]

Under cover of heavy naval gunfire on the morning before an invasion, UDT swimmers wearing facemasks and swim fins were dropped about half a mile off-shore and swam to and from the beach recording the water depths shallower than 3.5 fathoms (6.4 msw; 21 fsw) with lead-lines and slates while examining the bottom for obstacles. On returning seaward, they formed a pick-up line and were snared by a passing boat, one by one, with a rubber loop thrown over an outstretched arm. The rare swimmer who missed his pick-up swam to sea hoping for a passing destroyer.[1]

Information obtained from the hydrographic reconnaissance was converted into a beach chart for planning the amphibious landing. The swimmers returned the following morning and fixed demolition charges to preassigned obstacles during breath-hold dives. When all obstacles were connected by detonating cord, the swimmers retired to seaward while a pair of fuse-pullers attached and ignited firing assemblies with 10 to 15 minute delays. The fuse-pullers—usually the fastest swimmers—freestyled seaward. This was the only time that underwater recovery strokes were not required.[1]

In 1983, the Underwater Demolition Teams were converted to SEAL teams or SDV teams. Today's students at Basic Underwater Demolition/SEAL (BUD/S) training are still taught breath-hold diving, hydrographic reconnaissance, and combat demolition, but these techniques see only occasional use such as the 1983 combat hydrographic reconnaissance conducted in Grenada (*Operation Urgent Fury*) or the decoy combat demolition during the 1991 invasion of Kuwait in the Gulf War (*Operation Desert Storm*).

Source: (1) Fane FD. *The Naked Warriors*. Annapolis, Md: Naval Institute; 1995.

lem in diving, but the lungs are also affected, sometimes fatally, in breath-hold diving (Case Study 1).

Case Study 1: Fatal Lung Squeeze. A 28-year-old US Navy Underwater Demolition Team diver was free-diving in 24 msw (80 fsw) in Subic Bay, Philippines, in 1968. After a series of uneventful dives, he was found unconscious and face-up at 12 msw (40 fsw) while sinking slowly. On rescue, he was apneic and bleeding frothy, bright, red blood from the mouth. He became coherent 45 minutes later but developed progressive dyspnea and cyanosis and died 3 hours later. At autopsy, his lungs were congested and edematous with interstitial and intraalveolar hemorrhage.[2]

Figure 31-3 represents a breath-hold diver with a total lung capacity of 6 L and a residual lung volume of 1.5 L. If this diver performed a maximal inhalation on the surface (1 ata) and descended for a free dive, Boyle's law predicts that the lung volume would decrease to 3 L at 10 msw (33 fsw; 2 ata) and 2 L at 20 msw (66 fsw; 3 ata). On reaching 30 msw (99 fsw; 4 ata), the lung volume equals the 1.5-L residual volume.

With greater descent, the elasticity of the chest wall resists further reduction in volume and the alveolar pressure becomes less than the absolute pressure, which is transmitted equally throughout all solid and liquid tissues in accordance with Pascal's law: pressure exerted at any point on a confined liquid is transmitted uniformly in all directions. This phenomenon causes a relative vacuum between alveolar gas and alveolar capillary blood and leads to engorgement of the alveolar capillaries as blood shifts from peripheral tissues into the thorax. Further descent is possible because these fluid shifts reduce the residual volume.

The fluid-shift effect is illustrated by record free dives to depths in excess of 100 msw (330 fsw). Suppose, for example, a diver with a 7.22-L total lung capacity and a 1.88-L residual volume achieved a depth of 105 msw (346 fsw).[3] By Boyle's law, the ratio of these volumes specifies 28 msw (93 fsw) as the depth at which the lungs are compressed to residual volume. To reach 105 msw, the residual volume would have to be reduced to 0.63 L, representing a 1.25-L shift of blood from peripheral vessels

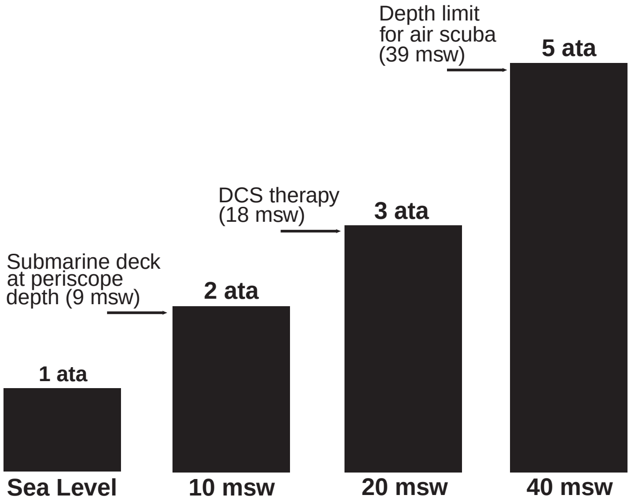


Fig. 31-2. Benchmark seawater pressures. The standard pressure at sea level is defined as 1 atmosphere absolute (1 ata, or 760 mm Hg). Each 10 meters of seawater (msw) (equivalent to 33 ft of seawater [fsw]) of descent adds an additional atmosphere of pressure. The deck of a submarine at periscope depth during lock-out and lock-in of combat swimmers is at about 9 msw (30 fsw). (Divers "lock-out" from and "lock-in" to a submarine when they enter and leave through an escape trunk that can be flooded or drained while the pressure is increased or decreased to allow transition from lower submarine pressure to higher sea pressure.) Decompression sickness (DCS) is usually treated at a pressure of 2.8 ata (18 msw; 60 fsw). The depth limit for open-circuit compressed air scuba is 39 msw (130 fsw).

to pulmonary capillaries. There is indirect evidence that such blood shifts do occur, but reductions in residual volume may also result from elastic compression of the chest wall and upward shift of the abdominal contents.

Descent to too great a depth causes compression of the thorax to beyond its elastic limit, and the resulting chest pain signals the diver to ascend, as in Case Study 2, below. Failure or inability to heed this warning pain could cause lung squeeze, thoracic squeeze, or chest squeeze, in which chest wall compression, intraalveolar vacuum, or both, damage the thorax and lungs. Thoracic squeeze is rare because of chest pain, but the diver in Case Study 1, above, appears to have lost consciousness, exhaled passively, and descended due to negative buoyancy according to Archimedes' principle: any object immersed in liquid will be buoyed up by a force equal to the weight of the water displaced. In such a circumstance, the warning chest pain would have gone unnoticed.

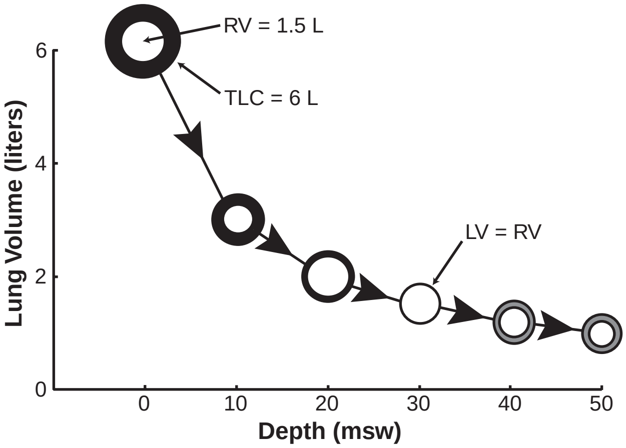


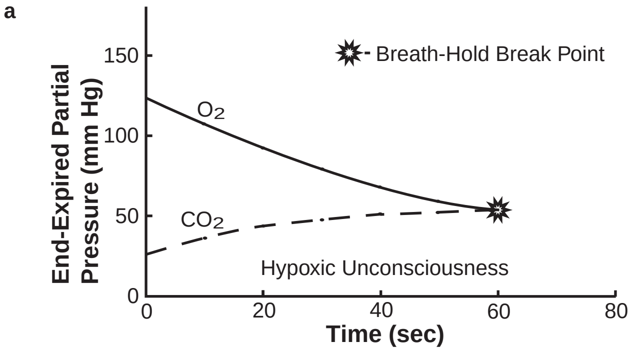
Fig. 31-3. Compression of the lungs during descent on a breath-hold dive. The diver with a residual lung volume (RV) of 1.5 L (white center of circles) begins on the surface with a 4.5 L vital capacity inhalation (black circles) to a total lung capacity (TLC) of 6 L. During descent, the lung volume (LV) decreases in inverse proportion to the absolute pressure, as described by Boyle's law. At 4 ata (30 msw; 99 fsw), the lung volume equals the residual volume. With additional descent, the elasticity of the thorax impedes further compression, leading to a relative vacuum in the lungs. Blood from the peripheral circulation shifts into the pulmonary capillaries (gray circles), which then reduces the residual lung volume (white centers) and allows descent to 40 and 50 msw (132 and 165 fsw).

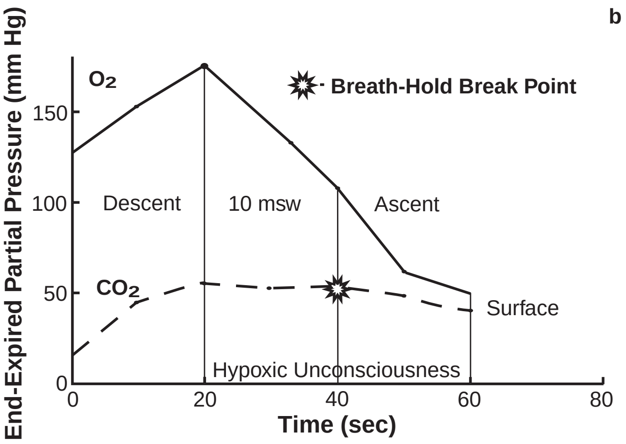
Case Study 2: Unconsciousness on Ascent. A 29-year-old Underwater Demolition Team diver making a breath-hold dive in the US Navy Submarine Escape Tower in Hawaii in 1971 noted chest pain at 27 msw (90 fsw) during descent, and he began his ascent. On reaching 12 msw (40 fsw), he experienced severe dyspnea followed by euphoria at 6 msw (20 fsw). Just before surfacing, he became unconsciousness and convulsed after being pulled from the water. Recovery was uneventful.

The symptoms reported in Case Study 2 are clues to the mechanisms responsible for the not-uncommon "breath-hold blackout" that occurs in breath-hold swimming and diving. (Breath-hold blackout is sometimes called "shallow-water blackout." The term "shallow-water blackout" originated during World War II and is discussed below in Closed-Circuit Oxygen Scuba.) There is an inherent risk of unconsciousness during diving or breath-hold swimming in shallow water if time underwater is prolonged inadvertently, by hyperventilation, or by will power. This has been a causative factor in many cases of unconsciousness or drowning.[3]

Hypercapnia and hypoxia are the principal causes of the ventilatory drive that is responsible for dyspnea. In severe hypoxia, euphoria often precedes unconsciousness, and hypoxic seizures are not uncommon (as in Case Study 2.) In the early 1960s, Edward H. Lanphier, MD, and Hermann Rahn, PhD, conducted experiments in a hyperbaric chamber to demonstrate the changes that occur to gases in the lungs during breath-hold and breath-hold diving. Figure 31-4a illustrates the progressive rise in end-tidal carbon dioxide partial pressure and the fall in oxygen partial pressure during a breath-hold experiment at sea level.[4] Gases were sampled from a bag into which the subject exhaled (and reinhaled) every 10 seconds. Hypoxic and hypercapnic ventilatory drive caused a break in breath-hold at 60 seconds at well above the 20 to 30 mm Hg (0.04 atm) alveolar oxygen partial pressure at which there is a risk of unconsciousness.

In another experiment (Figure 31-4b),[5] a subject made a breath-hold dive to 10 msw (33 fsw). The end-tidal oxygen and carbon dioxide partial pressures increased during descent, as described by Dalton's law of partial pressures: the total pressure exerted by a mixture of gases is equal to the sum of the pressures that each gas would exert if it alone occupied the container. While at depth, the oxygen partial pressure decreased owing to the subject's





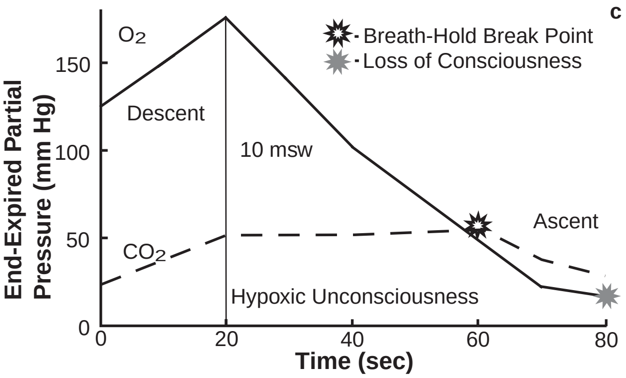


Fig. 31-4. (**a**) Effects of breath-hold at sea level on end-tidal oxygen and carbon dioxide. End-exhalation oxygen and carbon dioxide partial pressures were measured at 10-second intervals when the subject exhaled into a bag and then reinhaled. There was a progressive decrease in the oxygen partial pressure and an increase in the carbon dioxide partial pressure. Breath-hold breakpoint occurred when the sum of the hypoxic and hypercapnic ventilatory drives became too great to withstand. (**b**) Effects of breath-hold on end-tidal oxygen and carbon dioxide during a 10 msw (33 fsw) dive. The oxygen and carbon dioxide partial pressures increased on descent to 10 msw (33 fsw) according to Dalton's law of partial pressures: the total pressure exerted by a mixture of gases is equal to the sum of the pressures that each gas would exert if it alone occupied the container. While at depth, oxygen was continually removed from the lungs by the circulation to replenish metabolic consumption. Carbon dioxide remained relatively constant, as its alveolar partial pressure was greater than that in the arterial blood. Breath-hold breakpoint occurred entirely as a result of hypercapnic ventilatory drive, since the oxygen was well above the onset point of hypoxic drive. The oxygen and carbon dioxide partial pressures decreased on ascent, again according to Dalton's law. (**c**) Effects of breath-hold on end-tidal oxygen and carbon dioxide during a 10 msw (33 fsw) dive after hyperventilation. Hyperventilation eliminates carbon dioxide and delays the onset of hypercapnic ventilatory drive, which is responsible for the breath-hold breakpoint. Additional oxygen is consumed during the extended breath-hold, and on ascent, the oxygen partial pressure falls below the level needed to sustain consciousness. Graph a: Adapted with permission from Lanphier EH, Rahn H. Alveolar gas exchange during breath-hold diving. *J Appl Physiol*. 1963;18:471–477. Graphs b and c: Adapted with permission from Lanphier EH, Rahn H. Alveolar gas exchange during breath holding with air. *J Appl Physiol*. 1963;18:478–482.

metabolism, but it remained above the 50 to 60 mm Hg level at which the hypoxic ventilatory drive begins. Carbon dioxide actually diffused from the lungs into the blood because of its higher alveolar partial pressure. Hypercapnic ventilatory drive forced the subject to ascend after 40 seconds at 10 msw (33 fsw), and the oxygen and carbon dioxide partial pressures decreased, again due to Dalton's law.

In yet another experiment,[5] the subject dived to 10 msw (33 fsw) after hyperventilating to eliminate carbon dioxide and delay the onset of the hypercapnic ventilatory drive (Figure 31-4c). This extended his dive time by 20 seconds, during which oxygen metabolism continued but without inducing hypoxic ventilatory drive. When hypercapnia finally caused the diver to ascend, his oxygen partial pressure fell to below 40 mm Hg, and he exhibited cyanosis, confusion, and loss of control.

Lanphier and Rahn's experiments took place under close supervision in a dry, hyperbaric chamber. Underwater, the outcome might have been different. The diver in Case Study 2 could have drowned, for example, or met a fate similar to that described in Case Study 1, had he not been rescued.

CENTRAL NERVOUS SYSTEM OXYGEN TOXICITY IN COMBAT DIVERS

During World War II, Italian and British divers used oxygen diving effectively for hydrographic reconnaissance, ship attack, and with submersible craft, but fatalities were common due to loss of consciousness and subsequent drowning in both training and combat. Cerebral oxygen toxicity (gas toxicities are discussed in Chapter 30, Physics, Physiology, and Medicine of Diving) was one cause of these difficulties, and recommended maximum exposures of 120 minutes at 15 msw (50 fsw) or 30 minutes at 27 msw (90 fsw) provided inadequate protection. To better define safe limits for oxygen diving, the Royal Navy established a program that exposed human volunteers to various oxygen pressures and durations. The experience of one volunteer, quoted below, is instructive Case Study 3:

Case Study 3: Oxygen Convulsions During 30 Minutes at 50 fsw. I suddenly felt a violent twitching of my lips. … [M]y mouth was blown out like a balloon. … The twitching … increased, and I felt a terrific tingling sensation at the side of my mouth as if someone were touching it with a live wire. This … became a definite pain … . [M]y lips became so distorted … as if my mouth were stretched to my right ear. … Although my lips formed words, no sound came. … [B]lackness closed in on me—I was out.[6(pp60–61)]

A seizure is the most spectacular and objective sign of central nervous system (CNS) oxygen toxicity, but there is no evidence that it leads to permanent damage if the oxygen exposure is promptly discontinued (assuming, of course, that drowning and physical injury are avoided). Experimental oxygen exposures are often terminated by the subject when symptoms, including abnormal breathing, nausea, twitching, dizziness, incoordination, and visual or auditory disturbances, are noted. These symptoms also occur during operational exposures but do not necessarily precede convulsions.

Factors that elevate cerebral blood flow also augment oxygen delivery to the brain, which appears to increase susceptibility to oxygen toxicity. These factors include immersion, exercise, and carbon dioxide. Carbon dioxide may be present in the inspired gas or may be retained in the body, owing to inadequate ventilation caused by high gas density or external breathing resistance. Some people appear to have a lower than normal response to hypercarbia and are known as "carbon dioxide retainers." The primary treatment for all forms of oxygen toxicity is to reduce the partial pressure of inspired oxygen to a nontoxic level.

Oxygen exposure limits have been established to reduce the risk of convulsions for divers breathing pure oxygen or the oxygen in nitrogen–oxygen gas mixes; Figure 31-5 shows the exposure limits for pure oxygen[1] and for oxygen in mixed gas.[7] Oxygen exposure limits are based on very few data, and these data can be highly variable (compare Figures 31-5 and 31-18). Chamber trials and experience in open water indicate that convulsions occasionally occur near or within the accepted oxygen exposure limits. The incidents of convulsions or symptoms of CNS oxygen toxicity that have occurred in US Navy experiments are shown in Figure 31-5. Incidents with nitrogen–oxygen gas mixes were observed at lower oxygen partial pressures than incidents with pure oxygen. This is the reason that the oxygen exposure limits are more conservative for nitrogen–oxygen mixes than for pure oxygen. Nitrogen–oxygen mixes have higher gas densities than oxygen, as they are used at greater depths. The higher gas densities are believed to cause greater carbon dioxide retention within the body, which increases susceptibility to oxygen toxicity.

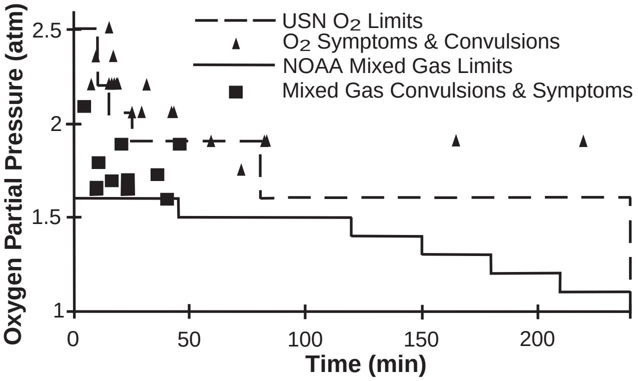


Fig. 31-5. Central nervous system (CNS) oxygen toxicity exposure limits for 100% oxygen and nitrogen–oxygen mixes. The dashed line describes the US Navy limits for single dives with 100% oxygen.[1] The solid line describes the National Oceanographic and Atmospheric Administration's (NOAA's) limits for exposure to oxygen in mixed gas.[2] The triangles represent CNS oxygen toxicity symptoms or convulsions during experiments with 100% oxygen that were conducted by the US Navy, and the squares represent symptoms or convulsions with nitrogen–oxygen mixes.[3–8] The mixed-gas limits are more conservative than the 100% limits because symptoms and convulsions occurred at lower oxygen partial pressures when nitrogen–oxygen mixes were breathed.

Data sources: (1) US Department of the Navy. *US Navy Diving Manual.* SS521-Ag-PRO-010. Washington, DC: Naval Sea Systems Command. Rev 4 (20 Jan 1999); Change A (1 Mar 2001): p18-14. NAVSEA 0994-LP-100-3199. (2) National Oceanographic and Atmospheric Administration. *NOAA Diving Manual*. 4th ed. Washington, DC: US Department of Commerce; 2001: 16-9. (3) Lanphier EH. *Nitrogen-Oxygen Mixture Physiology: Phases 1 and 2*. Panama City, Fla: Navy Experimental Diving Unit; 1955. NEDU Report 7-55. (4) Butler FK Jr, Thalmann ED. CNS oxygen toxicity in closed-circuit scuba divers. In: Bachrach AJ, Matzen MM, eds. *Underwater Physiology, Vol 8*. Bethesda, Md: Undersea Medical Society, Inc; 1984. (5) Butler FK Jr, Thalmann ED. CNS oxygen toxicity in closed-circuit scuba divers: II. *Undersea Biomed Res*. 1986;13(2):193–223. (6) Butler FK. *Central Nervous System Oxygen Toxicity in Closed-Circuit Scuba Divers: III.* Panama City, Fla: Navy Experimental Diving Unit; 1986. NEDU Report 5-86. (7) Piantadosi CA, Clinton RL, Thalmann ED. Prolonged oxygen exposures in immersed exercising divers at 25 fsw (1.76 ata). *Undersea Biomed Res.* 1979;6:347–356. (8) Schwartz HJC. *Manned Testing of Two Closed-Circuit Underwater Breathing Apparatus: US Navy Emerson Rig and Fenzy PO.68.* Panama City, Fla: Navy Experimental Diving Unit; 1984. NEDU Report 13-84.

UNDERWATER BREATHING APPARATUSES

A UBA provides the diver with a continuous and reliable supply of physiologically safe breathing gas. The use of such devices by the military became prominent in World War II, when they were used by the "human torpedoes" (see the Section IV frontispiece in this textbook) and by the divers who cleared the Normandy beaches of German countermeasures before the D day invasion (Exhibit 31-2). There are two broad categories of UBAs, surface supplied (or tethered) and self-contained, and each of these has a number of subcategories with advantages and disadvantages that lend to a particular operational utility.

Open-Circuit Self-Contained Underwater Breathing Apparatus: The Aqualung

The most familiar and common UBA in use today is the open-circuit, compressed-air self-contained underwater breathing apparatus (scuba), also known as the aqualung. This concept was first conceived and implemented in the 19th century[8] but did not become practical until Cousteau and Gagnan applied 1940s technology (Exhibit 31-3).[9] Improvements have been continuous since then, and compressed-air scuba is presently used by millions of divers throughout the world.

Open-circuit scuba consists of one or more tanks of gas compressed to a pressure of 137 to 341 ata (2,000–5,000 psi gauge [psig]). A first-stage pressure regulator attached to the tanks reduces their high pressure to an intermediate pressure of about 7 atm (100 psig) over the ambient water pressure. During inhalation, a second-stage regulator, held in the diver's mouth, reduces the intermediate pressure to ambient. The system is described as "demand" and "open-circuit" because the diver inhales on demand and exhales directly into the water.

The partial pressures of oxygen and nitrogen change with depth for divers breathing air or 100% oxygen (Figure 31-6). With pure oxygen, there is a significant risk of CNS oxygen toxicity at 10 msw (33 fsw), while with compressed air, which is only 21% oxygen, there is no oxygen CNS toxicity risk until depths of about 57 to 66 msw (187–218 fsw) are reached.

Surface-Supplied Diving

UBAs became possible with the invention of the air compressor in the 18th century during the industrial revolution, but the first practical equipment did not appear until about 1828, when the Deane brothers in England developed an open helmet that rested on the diver's shoulders.[10] Hand-driven pumps on the surface supplied the helmet with air through a hose, and excess air escaped around the shoulders, but the helmet would flood if the diver leaned over too far. When the men operating the hand-pumps on the surface became tired or when the diver was deeper than about 45 msw (150 fsw), work became impossible, as the air supply was insufficient to remove carbon dioxide.

Further developments by the Siebe-Gorman Div-

EXHIBIT 31-2

AMPHIBIOUS WARFARE IN NORMANDY: OPERATION OVERLORD

Hydrographic reconnaissance and obstacle clearance in the English Channel before the invasion of Europe on June 6, 1944, in Operation Overlord, were quite different than they were in the Pacific. The tidal zone off the Normandy beaches was 300 yd broad, with a tidal range of 25 ft and a speed of rise of 8 min/ft. Row after row of steel, concrete, rock, and wooden obstacles had been emplaced by German forces, in anticipation of an invasion.[1]

Hydrographic reconnaissance was conducted at night by divers (using oxygen rebreathers) who were launched from Royal Navy X-craft (dry midget submarines). The divers, who were from several nations, departed the surfaced submarines at about 1,500 yd off-shore, taking soundings and bottom samples as they approached the beach. On reaching the obstacles, they made detailed drawings of their location, arrangement, and associated mines.[2]

During clearance operations on D day, US Naval Combat Demolition Units (NCDU) at Omaha and Utah beaches loaded demolitions on their obstacles while wading in the rising tide. On Omaha Beach, direct exposure to hostile fire caused more than 50% casualties in NCDU personnel. On Sword and Gold beaches, the British used divers and had fewer than 5% casualties.[3]

The US landing force did not have an oxygen diving capability at the time of Operation Overlord. This technique was declined by the Navy in 1940 when offered it by Christian Lambertsen, who was a medical student at the time. Dr Lambertsen subsequently developed military oxygen diving teams for the Office of Strategic Services (OSS) and trained Navy Underwater Demolition Teams and Army engineers in oxygen diving after the war[4] (Exhibit Figure 1).

Exhibit Fig 1. An Office of Strategic Services (OSS) swimmer is pictured underwater using the Lambertsen Amphibious Respiratory Unit (LARU) Mark 10, also called the Lambertsen rebreather, on a training exercise in 1943. Photograph: Courtesy of C. J. Lambertsen, Philadelphia, Pa.

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1. Fane FD. *The Naked Warriors*. Annapolis, Md: Naval Institute; 1995.
2. Brou W-C. *Combat Beneath the Sea*. New York, NY: Thomas Y. Crowell; 1957.
3. Kelly O. *Brave Men—Dark Waters*. Novato, Calif: Presidio; 1992.
4. Larsen HE. *A History of Self-Contained Diving and Underwater Swimming*. Washington, DC: National Academy of Sciences—National Research Council; 1959. Publication 469.

EXHIBIT 31-3

JACQUES YVES COUSTEAU AND THE INVENTION OF THE AQUALUNG

Jacques Cousteau's first look through a pair of underwater goggles in 1936 changed his life, refocusing it on diving. He was not content with free diving, however, or diving with a manually controlled air supply, which he used in the 1930s. He found that a homemade oxygen rebreather, which gave him two seizures, and a continuous-flow surface air supply, which gave him a near chest-squeeze, did not meet his requirements, either. He wanted to swim freely with an apparatus that would automatically provide air when he breathed. In 1942, he found Emile Gagnan, a Parisian engineer who had built a demand valve to supply natural gas to automobile engines during World War II, when gasoline was in short supply. Within 6 months, this device was adapted for compressed air, and the aqualung was a reality. Today, it is used by millions of divers worldwide.

Source: Cousteau JY, Dumas F. *The Silent World*. New York, NY: Harper Brothers; 1953.

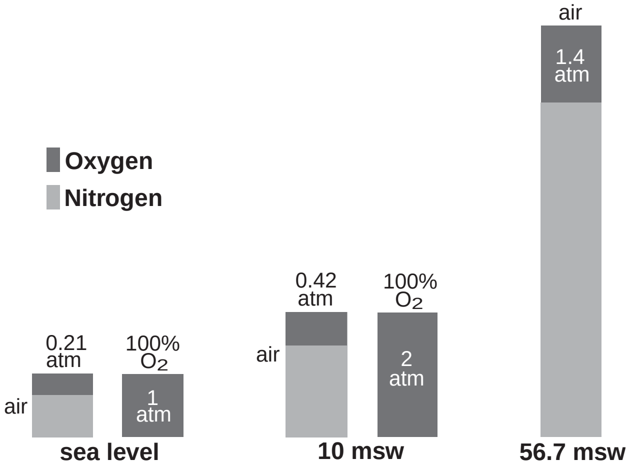

Fig. 31-6. Nitrogen and oxygen partial pressures for air and 100% oxygen. When diving with 100% oxygen, the threshold depth for central nervous system oxygen toxicity is quite shallow, about 6 to 7.6 msw (20–25 fsw; 1.6–1.76 atm). With air, the threshold is on the order of 56.7–66.2 msw (187–218 fsw), where the oxygen partial pressure is 1.4 to 1.6 atm. The "thresholds" are poorly defined owing to lack of data, and the threshold concept may not be entirely valid because of various factors that influence susceptibility, such as exercise, immersion, and inspired carbon dioxide.

Fig. 31-7. US Navy Mark (Mk) 12 (left) and Mk V (right) diving helmets. The Mk V was used until 1980, when it was replaced by the Mk 12. The Mk 21 replaced the Mk 12 in 1993. Reprinted from US Department of the Navy. *US Navy Diving Manual.* SS521-Ag-PRO-010. Washington, DC: Naval Sea Systems Command. Rev 4 (20 Jan 1999); Change A (1 Mar 2001): p1-9. NAVSEA 0994-LP-100-3199.

ing Company of England in the mid 19th century added a closed suit to the helmet, which prevented flooding and improved thermal protection. This became the traditional "hard-hat" deep-sea diving dress, which remained the primary equipment for military and commercial diving until the 1970s. The US Navy diving helmet, the Mk V (Figure 31-7), was introduced 1905 with improvements in 1916 and 1927 and was the system of choice until 1980, when it was replaced by the Mk 12. The Mk 12 was used until replaced by the Mk 21 in 1993 (see Figure 31-1). The Mk 21 incorporates a demand regulator from open-circuit scuba but instead of a mouthpiece, uses an oronasal mask that allows spoken communications. The oronasal mask in the Mk 21 has much less respiratory dead space than the Mk V and Mk 12 and permits lower gas-supply flow rates without carbon dioxide retention. This was an important improvement.

The Mk 21 Mod 1 helmet has a depth limit of 57 msw (190 fsw) when used for air diving. Its principal applications are search, salvage, inspection, ship's husbandry, and enclosed-space diving. The Mk 21 Mod 1 may also be used to a depth of 90 msw (300 fsw) with helium–oxygen mixtures (see Figure 31-1). Another tethered diving system in the US Navy inventory is the lightweight Mk 20 Mod 0 (Figure 31-8), which is used to a maximum depth of 18 msw (60 fsw) for diving in mud tanks or enclosed spaces. For saturation diving, the Navy uses

Fig. 31-8. The US Navy Mark 20 Mod 0 surface-supplied, open-circuit, lightweight system with full facemask is limited to a depth of 60 fsw for applications such as diving in mud tanks and enclosed spaces. Reprinted from US Department of the Navy. *US Navy Diving Manual.* SS521-Ag-PRO-010. Washington, DC: Naval Sea Systems Command. Rev 4 (20 Jan 1999); Change A (1 Mar 2001): p6-55. NAVSEA 0994-LP-100-3199.

Fig. 31-9. The US Navy Mark (Mk) 21 Mod 0 helmet with hot water suit (left) and Mk 22 Mod 0 band mask with hot water suit and shroud (right) for heating breathing gases. These are the primary underwater breathing apparatuses for saturation diving. Reprinted from US Department of the Navy. *US Navy Diving Manual.* SS521-Ag-PRO-010. Washington, DC: Naval Sea Systems Command. Rev 4 (20 Jan 1999); Change A (1 Mar 2001): p15-6. NAVSEA 0994-LP-100-3199.

the Mk 21 Mod 0 helmet and Mk 22 Mod 0 band mask (Figure 31-9) with hot water suit and hot water shroud for breathing-gas heating.

Closed-Circuit Oxygen Scuba

Closed-circuit oxygen scuba was invented by Henry Fleuss in 1878, when chemical agents that absorbed carbon dioxide were discovered.[11] This device allowed a diver to rebreathe expired gas after carbon dioxide had been removed and oxygen added. There were further developments by the Siebe-Gorman Diving Company in England, but the only apparent use of oxygen rebreathers before World War II was for clearing a flooded tunnel under the Severn River in 1882.[11] Closed-circuit oxygen diving became firmly established as an operational technique as a result of activity during World War II (Exhibit 31-4).

Closed-circuit oxygen scuba is relatively simple and produces no bubbles in the water to disclose the diver's location when used correctly, but its hazards have relegated it largely to military operations. During World War II, unconsciousness from mysterious causes was common during closed-circuit oxygen diving and was known as "shallow-water blackout."[12] Factors contributing to this problem included

- dilution hypoxia from failure to initially purge nitrogen from the lungs and the breathing loop;
- CNS oxygen toxicity at depths deeper than about 7.5 msw (25 fsw);
- carbon dioxide poisoning caused by the design, malfunction, or exhaustion of the CO_2 absorbent; and
- flooding of the breathing loop, leading to both loss of buoyancy and chemical burns from wet absorbent ("caustic cocktail").

The safety of closed-circuit oxygen diving equipment is determined, to large degree, by its design. Early units were pendulum rebreathers in which the diver inhaled and exhaled through a single hose into a CO_2 scrubber and "counterlung" (breathing bag). Figure 31-10 shows the pendulum unit used by X-craft divers to attack the *Tirpitz* and *Takeo*[6] (see Exhibit 31-4). Oxygen from a high-pressure cylinder was added to the breathing bag to make up for metabolic consumption. In this configuration, the mouthpiece and hose were a dead space in which carbon dioxide accumulated. The dead space volume increased as the

EXHIBIT 31-4

CLOSED-CIRCUIT OXYGEN DIVING IN COMBAT

The first extensive use of closed-circuit oxygen diving was by the Italian navy in the Mediterranean Sea during World War II. Attack swimmers from Italian "Gamma" units sank or disabled about 20 British merchantmen in Algiers, Algeria; Alexandretta, Turkey; and the Bay of Gibraltar, while piloted torpedolike submersibles known as "Maiale" (literally, "Sea Swine," in the Italian) disabled or sank about 15 ships in Alexandria, Egypt, including the British battleships HMS *Valiant* and HMS *Queen Elizabeth*.[1,2]

The British retaliated with their divers, known as Chariots or Human Torpedoes, who sank two Italian cruisers and several merchantmen moored near Palermo, Sicily. British divers also operated from midget submarines, known as X-craft, conducting successful operations in Norway, the Normandy region of France, and the Pacific Ocean. A dry-dock, a transport, and the German battleship *Tirpitz* were sunk or disabled in Norway. Divers also conducted hydrographic reconnaissance of the Normandy landing beaches prior to the invasion of Europe. They also disabled the Japanese cruiser *Takeo* in the Singapore harbor, and cut undersea telegraph cables to Hong Kong and Singapore.[1,2]

Since that time, public knowledge of closed-circuit oxygen diving in combat has been rare. A recent example was the sinking of a patrol craft by US Navy SEALs in the Panama Canal Zone. The US Navy also has applied the concept of Maiale and Chariots in developing the SEAL Delivery Vehicle (SDV) (see the Frontispiece of the Special Environments section of this textbook). An SDV is a wet submersible designed to carry combat swimmers on missions that include underwater mapping, recovery of lost objects, reconnaissance, and destruction of enemy harbor facilities or the naval order of battle. SDV divers use compressed air, closed-circuit oxygen, or closed-circuit mixed gas scuba. SDVs often operate off nuclear submarines that are equipped with Dry Deck Shelters (DDSs). A DDS has three pressure chambers and mates to an escape trunk or missile tube on the submarine. One chamber can be flooded to allow the submerged launch of an SDV, a second is a recompression chamber, and a third provides access to the submarine.[3]

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1. Brou W-C. *Combat Beneath the Sea*. New York, NY: Thomas Y. Crowell; 1957: 132–146.
2. Halberstadt H. *US Navy SEALs*. Osceola, Wis: Motorbooks; 1993.
3. Wood MP. Silent but deadly: The *USS Kamehameha* Dry Deck Shelter. *Full Mission Profile.* 1994;Spring:48-51.

CO_2 absorbent nearest the diver became depleted. Accumulating carbon dioxide gas led to carbon dioxide poisoning or potentiated CNS oxygen toxicity, both of which were causes of shallow-water blackout.[13]

The carbon dioxide retention problem due to dead space in the pendulum unit was corrected by the recirculating (ie, closed-circuit) rebreather, in which exhaled gas passes through the oxygen absorbent (O_2 scrubber) and into the breathing bag prior to reinhalation (Figure 31-11). This is the design of the Mark 25 UBA Draeger Lar V closed-circuit oxygen rebreather that is currently used by North Atlantic Treaty Organization (NATO) combat swimmers. About 10,000 Mark 25s have been produced. Another configuration placed the CO_2 scrubber between inhalation and exhalation breathing bags, which decreased the work of breathing by reducing the gas flow rate through the absorbent bed (Figure 31-12). This allowed more physical exertion with less carbon dioxide retention and was the design of the US Navy Emerson-Lambertsen oxygen rebreather that was used for about 20 years, from the 1960s through the 1980s.

The location of the breathing bag or bags relative to the lungs can cause carbon dioxide retention and reduce the diver's exercise capacity (Figure 31-13). A back-mounted bag is at a lower pressure than the lungs with the diver in a prone swimming position. This imposes a negative static lung load (as in breathing through a snorkel) and requires extra work during inspiration but less work during expiration. A chest-mounted bag imposes a positive static lung load, which assists inhalation but imposes extra work during exhalation. Of the two types of lung load, a small positive load causes less carbon dioxide retention and is preferable to a negative load.

Semiclosed Mixed-Gas Scuba

After the Normandy landings in World War II, European ports were found to be heavily mined and had to be cleared to allow war materiel to move for-

Fig. 31-10. (**a**) A diver wearing the pendulum closed-circuit oxygen rebreather used during World War II by Royal Navy X-craft divers. This rebreather was an adaptation of the Davis Submarine Escape Device (DSEA) manufactured in England by the Siebe-Gorman Diving Company. (**b**) Schematic diagram of a pendulum oxygen rebreather. The mouthpiece and hose, which connect the diver to the CO_2 scrubber, are a dead space in which carbon dioxide builds up during bidirectional ventilation. The diver exhales and inhales through a single hose connected to a CO_2 absorbent scrubber. The scrubber is built into a breathing bag (or counterlung) into which the diver exhales and from which he inhales. Fresh oxygen from the compressed gas supply below the breathing bag is added to compensate for metabolic consumption. The distal end of the scrubber is unused, while the proximal end adds to the dead space as absorbent is consumed. The rising concentration of carbon dioxide increases cerebral blood flow, thereby augmenting oxygen delivery to the brain and potentiating oxygen toxicity.

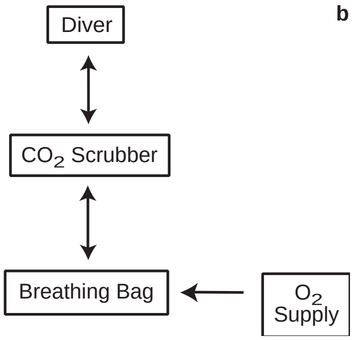
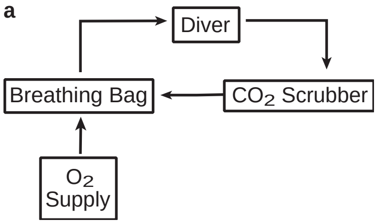
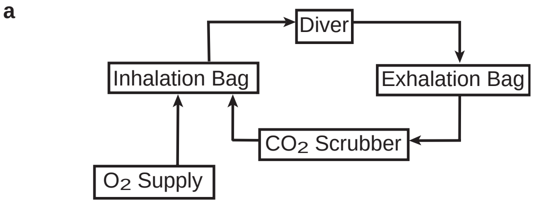


Fig. 31-11. (**a**) Schematic diagram of a closed-circuit oxygen rebreather. Equipment dead space is reduced by one-way valves, which ensure that gas flows in only one direction through the breathing hoses and the CO_2 absorbent (ie, the CO_2 scrubber). This eliminates all but a small dead space in the mouthpiece and results in more even use of the CO_2 scrubber. (**b**) The Mark (Mk) 25 UBA (Underwater Breathing Apparatus—Draeger Lar V) closed-circuit oxygen rebreather. This equipment has been the standard unit used by the North Atlantic Treaty Organization since the 1980s, and about 10,000 have been manufactured since 1981. Photograph b: US Navy.





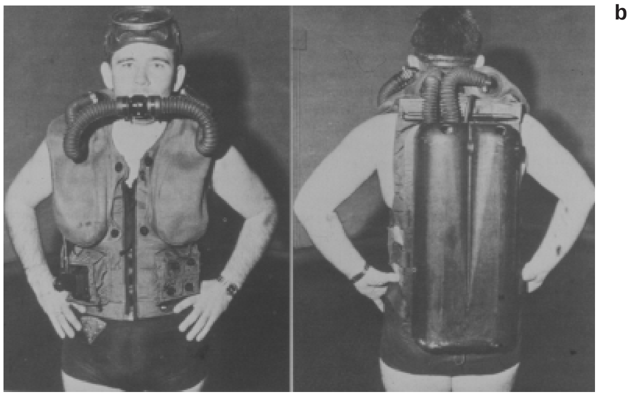


Fig. 31-12. (**a**) Schematic diagram of a split-bag closed-circuit oxygen rebreather. The split bag (ie, the inhalation bag is worn on the front; the exhalation, on the back) decreases peak gas flows through the CO_2 absorbent canister (the CO_2 scrubber). Separating the inhalation and exhalation bags reduces breathing resistance and prolongs gas residence time, which improves canister efficiency. (**b**) US Navy Emerson-Lambertsen closed-circuit oxygen rebreather. This is a split-bag, recirculating unit based on a design by Dr Christian Lambertsen that evolved from the Lambertsen Amphibious Respiratory Unit (LARU) Mark 10 (see Exhibit 31-2). The US Navy used the Emerson-Lambertsen unit from the 1960s through the 1980s. Photograph b: US Department of the Navy. *US Diving Manual*. Washington, DC: Navy Dept. March 1970: p573. NAVSHIPS 0994-001-9010.

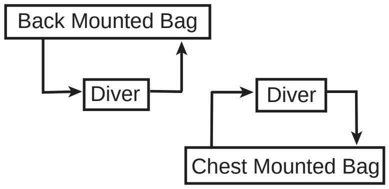


Fig. 31-13. Chest- and back-mounted breathing bags relative to a diver swimming in the prone position. The position of the breathing bag determines the static lung load, which significantly affects respiratory performance, particularly during exercise. A back-mounted bag imposes negative-pressure breathing, which is more deleterious to ventilatory function than the positive-pressure breathing imposed by a chest-mounted bag. The absolute magnitude of the static lung load is greater with the back-mounted bag, which floats off the diver's back instead of being pressed to the chest, as with the chest mount.

ward in support of the Allied advance. Closed-circuit oxygen scuba was unsatisfactory for this purpose, as oxygen toxicity limited its use to about 10 msw (33 fsw). To overcome the problem, the Royal Navy developed a breathing apparatus in which a mixture of nitrogen and oxygen (typically 40%–65% oxygen) rather than pure oxygen was added to the counterlung.[13] The apparatus was known as *semiclosed,* because some gas had to be exhausted to the sea as the diver did not metabolize the nitrogen. Thus, semiclosed scuba is not as bubble-free as oxygen scuba.

The same respiratory design constraints apply to semiclosed scuba as to closed-circuit oxygen scuba, and single or split breathing bags may be used with chest or back mounting. The chest-mounted, two-bag, semiclosed-circuit Mk VI unit that was employed by the US Navy from the 1960s through the 1980s and a schematic diagram of the Mk VI are shown in Figure 31-14. The inspired oxygen partial pressure of semiclosed scuba is a function of depth, the percentage of oxygen in the supplied gas, the gas injection rate, and diver's oxygen consumption. There is an optimal oxygen percentage for each depth to achieve a maximum gas-supply duration, and the diver must adhere to specific depth limits for each gas mixture to avoid CNS oxygen toxicity. If a diver works too hard, more oxygen may be consumed than is added, and the "dilution hypoxia" that results can cause unconsciousness. Dilution hypoxia may also occur if the supply gas is exhausted without the diver's knowledge.

Closed-Circuit, Mixed-Gas Scuba

Many of the difficulties of semiclosed-circuit scuba—oxygen toxicity, tell-tale bubbles, and gas-supply duration—are corrected by closed-circuit, mixed-gas scuba, which maintains a nearly constant oxygen partial pressure in the inspired gas. Figure

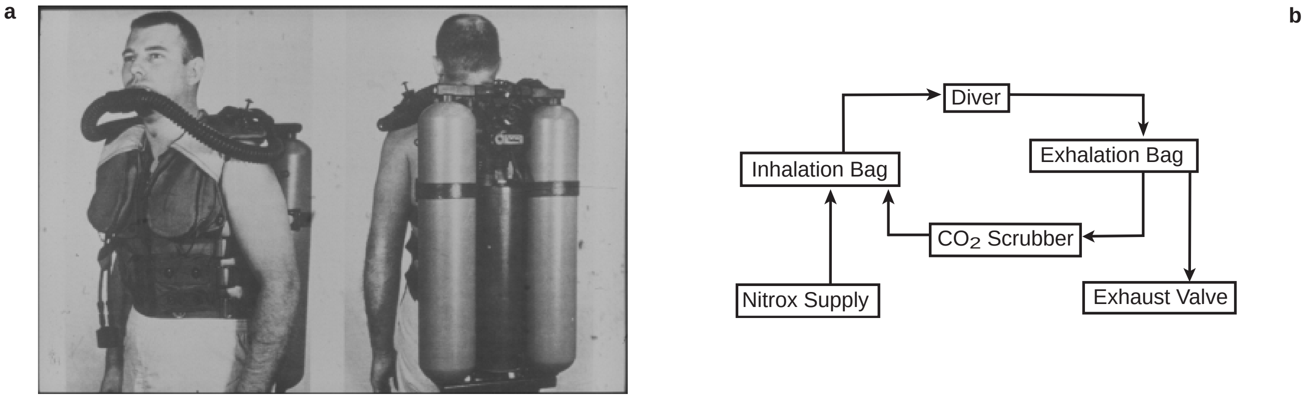


Fig. 31-14. (**a**) A Mark (Mk) 6 semiclosed-circuit, mixed-gas rebreather. This is a split-bag, recirculating unit, based on a Lambertsen design, which was used by the US Navy from the 1960s through the 1980s. (**b**) A schematic of the Mk 6 underwater breathing apparatus. Note the similarity of the Mk 6 shown here to the Emerson-Lambertsen closed-circuit oxygen rebreather (see Figure 31-12). The principal difference between the two designs is that the Mk 6 has a relief valve for venting excess gas because nitrogen from the supply gas is not metabolized and collects in the breathing circuit. Photograph a: US Department of the Navy. *US Diving Manual*. Washington, DC: DN. March 1970: 594. NAVSHIPS 0994-001-9010.

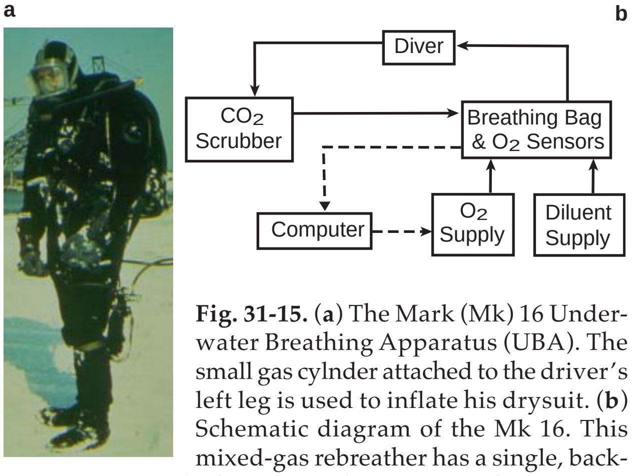


Fig. 31-15. (**a**) The Mark (Mk) 16 Underwater Breathing Apparatus (UBA). The small gas cylnder attached to the driver's left leg is used to inflate his drysuit. (**b**) Schematic diagram of the Mk 16. This mixed-gas rebreather has a single, back-mounted bag, which contains three sensors that measure the oxygen partial pressure. The dotted lines represent electrical signals to and from the computer. If the mean partial pressure falls below the set point (0.7 ata for the Mk 16), the computer adds oxygen to the breathing loop. The diluent supply is air or 16% oxygen in helium and serves to inflate the breathing bag during descent. Photograph a: US Navy.

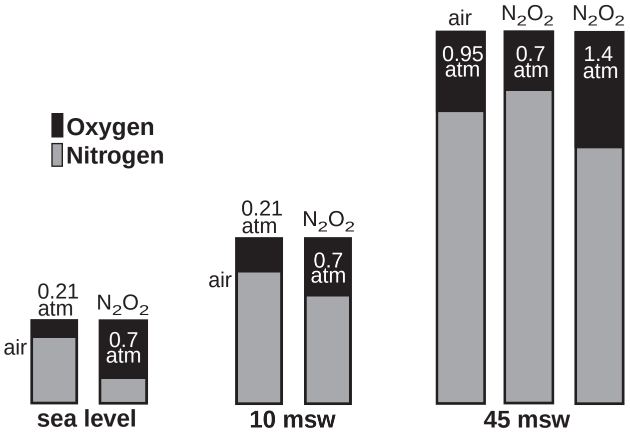


Fig. 31-16. Oxygen and nitrogen partial pressures when using a Mark (Mk) 16 mixed-gas rebreather. With an oxygen set point of 0.7 atm, the diver breathes 70% oxygen at sea level instead of 21% in air. The fraction of oxygen decreases with increasing depth for the Mk 16 and is 35% at 10 msw (33 fsw). At 23.3 msw (77 fsw), the gas in the Mk 16 is 21% oxygen. Deeper than 23.3 msw (77 fsw), the Mk 16 oxygen fraction becomes less than 21% (eg, 12.7% at 45 msw [150 fsw]). The Mk 16 is at a decompression disadvantage for dives deeper than 23.3 msw (77 fsw) and at an advantage shallower than 23.3 msw (77 fsw) or during shallow decompression stops. The disadvantage for deeper dives can be removed by raising the oxygen set point to 1.4 atm.

31-15 shows the US Navy Mk 16 UBA, which has a back-mounted breathing bag, and a schematic of the Mk 16. Closed-circuit, mixed-gas rebreathers have one or more oxygen sensors (the Mk 16 has three) that measure the inspired oxygen partial pressure. This partial pressure is compared to the desired oxygen set point (0.7 atm for the Mk 16) by an analog or digital computer, which adds oxygen to the breathing bag when the partial pressure falls below the set point.

Figure 31-16 shows how the oxygen and nitrogen (or other inert gas) partial pressures change with depth for an Mk 16 with an oxygen set point of 0.7 atm (compare Figure 31-6 with Figure 31-16.) Shallower than 77 fsw, a diver breathes a nitrogen–oxygen mixture that contains less nitrogen than air and, consequently, provides a decompression advantage (see below). Deeper than 77 fsw, the reverse is true, and the diver absorbs more nitrogen with the Mk 16. This disadvantage can be remedied by raising the set point to 1.3 or 1.4 atm.

Perhaps the greatest advantage of a closed-circuit mixed-gas rebreather is gas conservation. At a fixed depth, the gas consumption of a closed-circuit rebreather equals the diver's metabolic rate (between 0.5 L/min for rest and 3.0 L/min for work), which a small oxygen supply can support for hours. The purpose of the diluent supply (see Figure 31-15) is to fill the counterlung during descent, but the diluent can be quickly exhausted by multiple vertical excursions. The diluent is usually air for depths shallower than about 45 msw (150 fsw), and 12% oxygen in helium to prevent nitrogen narcosis to a depth of 90 msw (300 fsw). Both air and 12% oxygen (but not less) can be breathed in an open-circuit mode at the surface in the event of equipment malfunction. The use of rebreathers in the open-circuit mode is one of a number of emergency procedures that must be mastered during diver training.

Closed-circuit mixed-gas rebreathers are significantly more complex than open-circuit oxygen scuba, and malfunctions are more frequent. Among the disadvantages are a generally greater breathing resistance, additional training requirements for divers and maintenance personnel, and costs of initial purchase and subsequent maintenance. Closed-circuit mixed-gas scuba is still evolving and will always be a specialty that is most appropriate for divers who are highly trained, well funded, and willing to assume risks beyond those encountered with open-circuit scuba.

THE ROLE OF RESPIRATION IN DIVING INJURIES

Divers who make emergency ascents to the surface are at risk for arterial gas embolism (AGE) or decompression sickness (DCS), and those who lose consciousness underwater are at risk of drowning. Loss of consciousness when breathing air or nitrogen–oxygen has been called "deep-water blackout," as opposed to shallow-water blackout, which occurs with oxygen rebreathers. The causes of these events can be difficult to determine, but nonfatal occurrences and unplanned laboratory incidents indicate that respiration plays an important role, as Edward H. Lanphier, MD, describes in Case Study 4, quoted below. Knowledge of the underlying mechanisms is incomplete, as experimental investigations are understandably rare.

Case Study 4: Carbon Dioxide Retention and Dyspnea. We were testing a new bicycle ergometer at 7.8 ata (67 msw; 224 fsw) in the dry chamber. Nitrogen narcosis is very evident on air at that pressure, but we were doing OK until we started breathing on the measuring circuit that gave us only about half the air we needed. Herb stopped pedaling after about three minutes, out cold with his eyes rolled back. I took the bike. I knew I wasn't getting nearly enough air, but I was too narc'd to think straight and was determined to finish the test. I pedaled myself right into oblivion and coming around slowly afterwards with a horrible feeling of suffocation was the worst experience of my entire life. Both of us surely would have drowned if such a thing had happened when we were underwater.[14(pp67–69)]

Carbon Dioxide Retention and Dyspnea

Respiration is designed to maintain physiologically acceptable levels of oxygen and carbon dioxide in the blood and tissues, and healthy people breathing free air at sea level adjust their ventilation unconsciously to match their exertion. This is not always so during diving, where the effects of nonphysiological levels of oxygen, nitrogen, and carbon dioxide can interact and are exacerbated with increasing depth by work, breathing resistance, and gas density.[12]

Exercise capacity at sea level is limited by the cardiovascular system, whereas the respiratory system is usually the limiting factor during diving. Immersion shifts blood from the legs to the thorax, which reduces vital capacity and maximum ventilatory capacity. A regulator decreases the ventilatory capacity still further by increasing the work of breathing. Work of breathing is caused, in part, by resistance to gas flow in the airways and breathing apparatus. This resistance increases with depth as the gas density increases. Carbon dioxide is retained when ventilation is inadequate.

Carbon dioxide is the primary ventilatory stimulus in diving. The hypoxic ventilatory drive is generally absent, as most diving gases are hyperoxic. Blood is designed to carry oxygen and carbon dioxide at normoxic pressures, not at elevated oxygen partial pressures. At sea level pressure, where the venous oxygen is low, carbon dioxide is tightly bound to hemoglobin. At high oxygen partial pressures during diving, carbon dioxide is more loosely bound to hemoglobin, causing its tension in the blood and tissues to rise. This is known as the Haldane effect.

Dyspnea generally results if increased ventilation does not reduce the elevated carbon dioxide tension, but carbon dioxide can accumulate in the body without the increased ventilation that occurs at sea level. An interesting environment, frightening experience, or nitrogen narcosis (see Case Study 4) may inhibit ventilation, and divers sometimes consciously override the hypercapnic ventilatory stimulus and hypoventilate ("skip-breathe") to conserve air. Skip-breathing also can be responsible for headaches after diving.

The importance of adequate respiration is not usually stressed during diver training, and a diver who expects the same respiratory performance at depth as on land may be surprised by the breathlessness that can occur should sudden exertion be required in an emergency. As Case Study 4 indicates, dyspnea is a frightening experience, and panic is a common response. Newly trained divers are particularly susceptible to making emergency ascents when dyspnea occurs. A diver overcome by an overwhelming desire to surface and breathe free air may ascend too rapidly and risk AGE, DCS, or both.

An episode of respiratory insufficiency underwater can be a learning experience, but it is not an ideal lesson (Case Study 5). Since the normal unconscious regulation of respiration at sea level may be compromised during diving, divers should beware of incipient dyspnea, ventilate adequately, and minimize exertion. Sufficient ventilatory reserve should be maintained so that sudden, unexpected activity does not cause breathlessness and panic. If breathlessness occurs, the best way to avoid becoming in extremis is to stop all activity and let the ventilation return to normal.

Case Study 5: Deep-Water Blackout. During a dive to 54 msw (180 fsw) in a water-filled pressure chamber, a

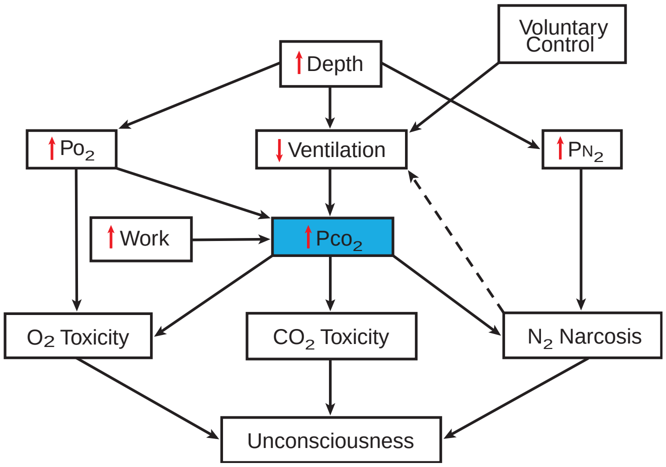


Fig. 31-17. Deep air diving is dangerous because interactions of depth, work, oxygen, nitrogen, and carbon dioxide affect respiration and consciousness. Carbon dioxide is the primary factor controlling respiration during diving when the hypoxic ventilatory drive is absent in the presence of hyperoxia (PO_2). Various factors contribute to increased PCO_2 in blood (center of diagram). Hyperoxia shifts the carbon dioxide dissociation curve to the right (the Haldane effect), which increases the PCO_2. Gas density increases as depth increases, which raises the work of breathing and decreases ventilation. Nitrogen narcosis increases with depth and may depress ventilation, as do other anesthetics. (Because this effect is hypothetical, it is shown as a dashed line.) Some divers reduce ventilation voluntarily to save gas, whereas others have poor ventilatory response to elevated carbon dioxide. Elevated carbon dioxide potentiates both nitrogen narcosis and central nervous system oxygen toxicity, and carbon dioxide itself is narcotic. In the presence of hyperoxia, dyspnea caused by carbon dioxide is less effective as a warning of altered consciousness. The risks of unconsciousness from oxygen and carbon dioxide toxicity and nitrogen narcosis (all of which increase with depth) are exacerbated by physiological interactions among oxygen, nitrogen, and carbon dioxide.

diver performed moderate exercise while swimming against a trapeze at an oxygen consumption of 2 L/min. He was using an Mk 15 UBA (similar to the Mk 16 [see Figure 31-15]) with an oxygen partial pressure of 1.4 ata in nitrogen. Despite orders from the Diving Supervisor to slow down, he increased his workload until he became unconscious. He revived immediately on removal from the water.

Interactions Between Gases and Impaired Consciousness

Carbon dioxide retention is exacerbated as depth increases by greater work of breathing. Inspired carbon dioxide partial pressures of 10% to 15% surface equivalent are narcotic and can affect a diver's consciousness.[1] When the oxygen partial pressure is elevated, hypercapnia loses its effectiveness as a warning signal of respiratory embarrassment or of impending unconsciousness. Excess carbon dioxide potentiates nitrogen narcosis, and narcosis can cause the hypercapnic ventilatory drive to be overlooked (see Case Study 4). Elevated carbon dioxide increases cerebral blood flow and raises oxygen delivery to the brain, increasing the risk of CNS oxygen toxicity.

Thus, diving can impair consciousness through the combined effects of nitrogen narcosis, carbon dioxide intoxication, and oxygen toxicity. These effects are exacerbated by exercise and gas density, which further increase carbon dioxide retention. Figure 31-17 illustrates the interactions among gases, exercise, and depth that increase the risk of unconsciousness.

Individual Susceptibility to Impaired Consciousness

Susceptibility to carbon dioxide retention, oxygen toxicity, and nitrogen narcosis vary widely from one individual to another. Some divers—the carbon dioxide retainers—have poor ventilatory response to inspired carbon dioxide[11]; they are believed to be at el-

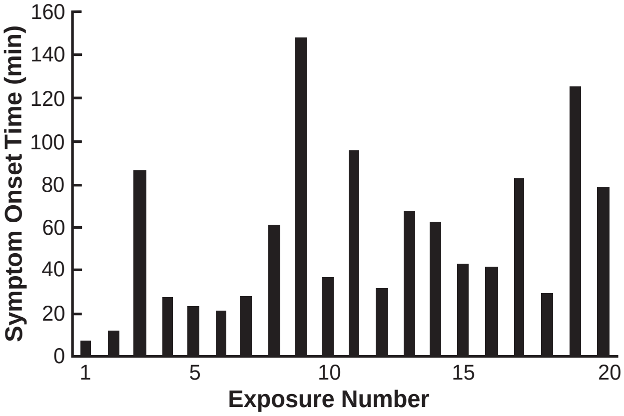


Fig. 31-18. Variation in individual susceptibility to symptoms of CNS oxygen toxicity. The time to symptom onset is illustrated for a single individual who was exposed to 100% oxygen at 21 msw (70 fsw) on 20 days over 3 months. The average onset time was 44 minutes, with a range of 7 to 148 minutes. Data source: Donald K. *Oxygen and the Diver*. Welshpool, Wales: The SPA Ltd; 1993: 45, 46.

evated risk of CNS oxygen toxicity due to increased cerebral oxygen delivery. Studies also have shown wide variability of the latent period before CNS toxicity for the same individual. Experiments[13] by the British during World War II found that the time to symptom onset varied randomly from 7 to 145 minutes for a single diver who made 20 exposures at 21 msw (70 fsw) while breathing 100% oxygen (Figure 31-18).

A few individuals have made compressed air (21% O_2) dives to depths of 90 to 150 msw (300–500 fsw) and have returned safely, despite nitrogen and oxygen stresses that would incapacitate most people. Other divers have developed severe DCS or have not returned, probably owing to loss of consciousness. There are individual differences in susceptibility among divers but no way to predict who is susceptible or resistant or how individual susceptibility varies from day to day.

DECOMPRESSION PROCEDURES

From the point of view of DCS, diving today is a relatively safe activity, especially when compared with the situation at the turn of the 20th century, when permanent paralysis and death were common (Case Study 6):

Case Study 6. Fatal Decompression Sickness in 1900. A Royal Navy diver descended to 45 msw (150 fsw) in 40 minutes, spent 40 minutes at depth searching for a torpedo, and ascended to the surface in 20 minutes with no apparent difficulty. Ten minutes later, he complained of abdominal pain and fainted. His breathing was labored, he was cyanotic, and he died after 7 minutes. An autopsy the next day revealed healthy organs but gas in the liver, spleen, heart, cardiac veins, venous system, subcutaneous fat, and cerebral veins and ventricles.[15]

By the present US Navy Standard Air Decompression Tables, this diver should have had 174 minutes of decompression time.[1] Decompression risk is relatively low for divers who follow standard decompression tables, such as those published in the *US Navy Diving Manual*,[1] but even divers who adhere to accepted tables may develop the less-serious forms of the disease and, occasionally, severe problems. Decompression tables specify rules for the time at maximum depth, decompression stops, and surface intervals between dives. More recently, diver-worn digital computers have automated the process of decompression calculations, making them simpler and less prone to the kinds of errors that divers make when working with tables. Neither dive tables nor dive computers guarantee freedom from risk of DCS, however, and both are the subjects of controversy, particularly as their specified safety limits disagree widely.

In 1993, the rate of ascent to the first decompres-

TABLE 31-1

NO-STOP DIVE TIMES AT FIVE DEPTHS

	Longest (No-Stop) Bottom Time Allowed at the Indicated Depth Without Required Decompression Stops				
	15 (msw)	**18 (msw)**	**24 (msw)**	**30 (msw)**	**39 (msw)**
Dive Table or Computer	**Time (min)**				
56 USN Tables	100	60	40	25	10
RN Tables	85	60	30	20	11
DSAT Tables	80	55	30	20	10
DCIEM Tables	75	50	20	10	5
BSAC Tables	74	51	30	20	13
EDGE	73	52	31	20	10
DataMaster	70	51	29	19	10
Datascan2	65	49	30	19	5
Aladin	62	45	23	15	7
Monitor	61	45	23	15	9

BSAC: British Sub Aqua Club; DCIEM: Defense Civil Institute of Environmental Medicine [United States]; DSAT: Diving Science and Technology [Canada]; EDGE: Electronic Dive Guide Experience; RN: Royal Navy; USN: US Navy

Data source: Lewis JE, Shreeves KW. *The Recreational Diver's Guide to Decompression Theory, Dive Tables, and Dive Computers*. Santa Ana, Calif: Professional Association of Diving Instructors; 1990.

sion stop was changed in the *US Navy Diving Manual* from 18 msw / min (60 fsw / min) to 9 msw / min (30 fsw / min).[1] The safety stop, a development in recreational diving during the 1990s, interrupts ascents from no-stop dives with a 3- to 5-minute stage at 3 to 6 msw (10–20 fsw).[16] Slower ascent rates and a safety stop can reduce the incidence of venous gas emboli, but their effect on the risk of DCS is uncertain. Studies with animals indicate that the effects of ascent rate can be complex.

No-Stop (No-Decompression) Dives

No-stop (ie, no-decompression) dives are the simplest, safest, and most common form of exposure. No-stop dives are short enough that the diver can return directly to the surface with an acceptably low risk of DCS. Unfortunately, there is considerable variability in the no-stop exposure limits given both by tables and dive computers. Table 31-1, for example, lists the no-stop limits at five depths for five decompression tables and five dive computer models.[17] The longest no-stop limits for any given depth are 1.4 to 3.6 times greater than the shortest limits.

In-Water Decompression Stops

If the bottom time at a given depth exceeds the stated no-stop limit for a decompression table or dive computer, the diver must remain at a shallower depth (a decompression stage or stop) long enough to allow inert gas to be eliminated harmlessly through the lungs. If the decompression stops are too short, excessive formation and growth of bubbles in the blood and tissues may result in DCS. In-water decompression stops are traditionally at 3 msw (10 fsw) intervals, with the most shallow stop being at 3 or 6 msw (10 or 20 fsw). With dive computers, however, decompression may be conducted during continuous ascent.

Experiments and observations (see a review by Vann and Thalmann[18]) have found that decompression time can be reduced by 30% to 50% with about the same or a lower risk of DCS if oxygen is used instead of air during in-water decompression stops, but this benefit comes with the risk of CNS oxygen toxicity. In-water oxygen decompression is not recommended deeper than 6 msw (20 fsw) and then only with careful attention to depth control. A back-up decompression plan should be available for times when in-water oxygen cannot be used. Air should be available as a back-up breathing gas in the event of oxygen-toxicity symptoms, and an emergency plan should be available to manage convulsions. British[19] and Canadian[20] tables have in-water oxygen decompression options but those developed and used by the US Navy do not.

Surface Decompression

During salvage of silver and gold in World War I, the weather or the military situation sometimes forced British divers to surface before completing their required in-water decompression stops (reviewed by Vann and Thalman[18]). Experience showed that this was possible if the divers were rapidly recompressed in a shipboard pressure chamber within 5 to 10 minutes of reaching the surface. Surface decompression was initially conducted with air, but subsequent studies by the US Navy (reviewed by Vann and Thalman[18]) found that decompression with oxygen was more effective. Surface decompression with oxygen (Sur-D O_2) is typically conducted with recompression on 100% oxygen to a depth of 12 msw (40 fsw). Oxygen breathing has been found acceptable at 12 msw (40 fsw) in a dry chamber, rather than 6 msw (20 fsw) for divers in the water, because dry divers are at a lower risk of oxygen toxicity (reviewed by Vann and Thalman[18]). The US Navy has surface decompression tables for both air and oxygen, but the air table is used only in emergencies.[1]

Repetitive and Multilevel Diving

If two dives are made in close succession, inert gas remaining in the body from the first dive requires that the second dive have a reduced bottom time or longer decompression time to avoid increased DCS risk. The second exposure is known as a *repetitive dive*, and the time between the two dives is called a *surface interval*. Complete elimination of inert gas from the body can take as long as 12 to 24 hours, depending on the diver's recent dives. The US Navy Air Decompression Tables treat all dives with surface intervals of less than 12 hours as repetitive dives, but this time is as short as 6 hours for some recreational dive tables, and as long as 18 hours for the Canadian tables.[20] Repetitive diving is common among recreational divers, and four or more no-stop dives per day are not unusual over several days. Repetitive diving is thought to increase the risk of DCS, but this appears to depend on the dive table or computer that is used. Definitive data to resolve the issue are unavailable.

Multilevel diving is a variant of repetitive diving in which the diver does not return directly to the surface but ascends in stages that take advantage of the longer no-decompression times at shallow depths (see Table 31-1) while avoiding mandatory decompression stops. Commercial, recreational, and military diving (with submersibles, in Special Operations) are frequently multilevel. There are a number of multilevel dive tables, but multilevel dives are most efficiently conducted when dive computers are part of divers' equipment.

Dive Computers

The common term for a digital computer that a diver carries underwater for decompression guidance is a *dive computer*. The computer is worn on the wrist where it can be easily viewed. (An alternative term sometimes used by the US Navy is Underwater Decompression Monitor [UDM]). The first commercially successful, mass-produced digital dive computer appeared in 1983, and many others are now available. All dive computers are programmed with models, or algorithms, that are derived from the same or similar mathematical calculations as decompression tables.

A decompression model is a mathematical representation of the kinetics of inert gas exchange in body tissues with rules to preclude ascents that might result in unsafe bubble formation or growth. Because the physiology of decompression is not completely understood and because of differences between individuals, decompression models are not totally effective in avoiding DCS, although the incidence appears to be less than 1%.[21]

Figure 31-19 shows a dive computer developed for use with SEAL Delivery Vehicles (SDVs) and a schematic diagram of a typical dive computer. Its principal components include a pressure transducer that reads the diver's depth; a temperature transducer for compensating the pressure transducer for temperature change; a timer for measuring dive time; memory to store a mathematical model (ie, an algorithm) of the decompression process; a microprocessor to sample depth and temperature and to compute the diver's decompression status; and a display for presenting the current depth and decompression status to the diver. Optional components measure the diver's gas-supply pressures and allow the dive's depth–time profile to be recorded for later recall on a personal computer.

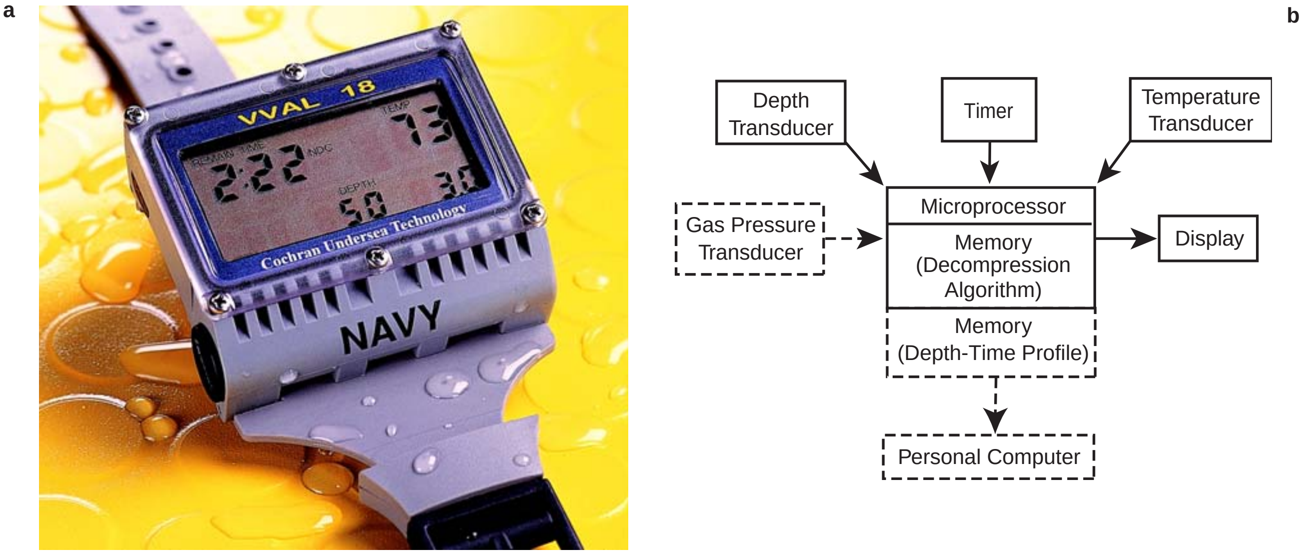


Fig. 31-19. (**a**) A dive computer for use with SEAL Delivery Vehicles (SDVs). Dive computers help a diver track his decompression status and guide his ascent to the surface. This is particularly useful for multilevel diving, wherein the diver does not return directly to the surface but varies his depth during the dive. Dive computers are easier to use than dive tables, but neither totally eliminates the risk of decompression illness. (**b**) Schematic diagram of a dive computer. Solid lines indicate mandatory components, which include depth and temperature transducers to measure and temperature-compensate the diver's depth; a timer, microprocessor, and decompression algorithm to compute decompression status; and a display to report the diver's decompression requirements. Dashed lines indicate optional components, which include a transducer to measure gas supply pressure and memory to store the depth–time profile for later recall by a personal computer. Photograph: Reprinted with permission from Cochran UnderSea Technology, Richardson, Tex.

Dive computers accurately track depth–time profiles and minimize the human errors that can occur in table selection. Dive computers are reasonably reliable, but hardware failures occasionally occur and backup computers or tables are recommended. The decompression "safety" of dive computers and tables is vigorously debated, but there are presently no data that offer compelling evidence of a higher incidence of DCS for either. Reports to the Divers Alert Network (DAN) suggest that among recreational divers with decompression injuries, AGE is less common among those who rely on their dive computers rather than conventional tables,[22] but dive computer safety has been questioned for multiple repetitive dives with short surface intervals.[23] To resolve the issue of safety will require knowledge of how the risk of decompression injury is affected by depth–time exposure. Dive computers with the capability for recording depth and time may ultimately provide data on which this knowledge can be based.

Nitrogen–Oxygen Diving

Many breathing devices can be used with gases other than air. The most common mixtures are of nitrogen and oxygen (often called *nitrox*) in which the oxygen percentage is greater than the 21% in air. Such mixes are also known as enriched air nitrox (EAN). Semiclosed- or closed-circuit rebreathers (see Figures 31-14 and 31-15) use nitrox, but the oxygen fraction varies with the depth. Nitrox mixes that contain less nitrogen than air reduce the risk of DCS if used with standard decompression procedures; however, the bottom time is often extended instead, which negates the reduction in decompression risk.

The advantages of fixed-percentage nitrox are as follows:

- reduced nitrogen absorption at depth, and
- accelerated nitrogen elimination during decompression.

And the disadvantages are

- the need for oxygen-clean equipment to limit fire hazard,
- the complexity of gas mixing,
- the requirement for accurate gas analysis, and
- the importance of depth control to stay within the mixed-gas oxygen-exposure limits for CNS oxygen toxicity (see Figure 31-5).

Figure 31-20 illustrates the oxygen and nitrogen partial pressures at various depths with air and with 36% nitrox. The 36% nitrox has a clear decompression advantage over air, owing to its lower nitrogen partial pressure, but the depth limitation due to increased risk of oxygen toxicity is also obvious. Ignoring depth limits in nitrox diving has led to fatal convulsions. Nitrox diving can be conducted with reasonable safety, but additional training in physics, physiology, and gas mixing and analysis is advisable.

The most widespread application of fixed-percentage nitrox diving is the National Oceanic and Atmospheric Administration's (NOAA's) adaptation of the US Navy Standard Air Tables[1] for 32% oxygen.[7] The no-stop exposure limit at 15 msw (50 fsw) is 200 minutes with 32% nitrox, for example, while the limit is only 100 minutes with air. Nitrox is not suitable for deep diving because of the increased risk of CNS oxygen toxicity. NOAA's upper limit for oxygen partial pressure exposure is 1.6 atm, which makes 39 msw (130 fsw) the greatest allowable depth with 32% oxygen. If the oxygen partial pressure is limited to 1.4 atm, the maximum depth is reduced to 33.8 msw (111 fsw).

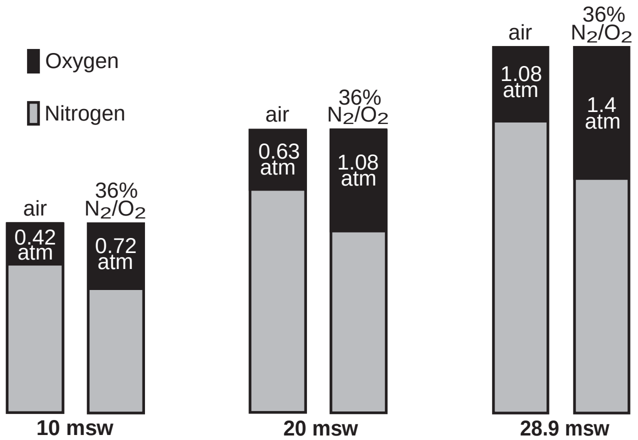


Fig. 31-20. Oxygen partial pressures in air and 36% nitrogen–oxygen mixture (nitrox). The 36% nitrox provides a decompression advantage over air by reducing the nitrogen partial pressure to which a diver is exposed. Thus, 36% nitrox at 10 msw (33 fsw) has the same nitrogen partial pressure and is the decompression equivalent of breathing air at 6.2 msw (20.5 fsw). This is known as the equivalent air depth (EAD). The EAD at 20 msw (66 fsw) is 14.3 msw (47 fsw), and the EAD at 28.9 msw (95 fsw) is 21.5 msw (71 fsw). **Note:** the deepest recommended depth for 36% nitrox is 28.9 msw (95 fsw), because at this depth the oxygen partial pressure rises to 1.4 atm; the risk of central nervous system oxygen toxicity may be excessive above this pressure.

Helium–Oxygen and Trimix Diving

For diving deeper than about 45 msw (150 fsw), *heliox* (a mixture of helium and oxygen) or *trimix* (a mixture of helium, nitrogen, and oxygen) are used to eliminate or reduce nitrogen narcosis. The oxygen fraction in these mixtures is often less than 21% and is chosen to avoid oxygen partial pressures above 1.3 to 1.4 atm, which are potentially toxic to the CNS at the planned maximum depth. The US Navy developed helium–oxygen tables for bottom times of up to several hours' duration (known as *bounce* dives), but these tables are not frequently used. The commercial diving industry developed its own helium tables in the 1960s and 1970s for off-shore oil work, but commercial tables are unpublished and not readily available. Helium–oxygen bounce diving became uncommon in commercial operations of the 1980s and 1990s and has been replaced by saturation diving (see below), which is more efficient.

Omitted Decompression

A diver who surfaces before completing a decompression stop is subject to increased risk of DCS. Decompression may be omitted as a result of hypothermia, wave action, tidal flow, equipment failure, dangerous marine life (Exhibit 31-5), or running out of air. Out-of-air situations are more common with scuba than with surface-supplied equipment. The US Navy requires an on-site recompression chamber (within a travel time of 30 min) for decompression diving because of possible omitted decompression and because the risks of decompression dives are greater than those of no-stop dives.[1] No-stop dives are safer than decompression dives because decompression *cannot* be omitted.

Flying After Diving and Diving at Altitude

DCS can occur independently of diving during altitude exposures above about 5,490 m (18,000 ft; a barometric pressure of 0.5 ata). Nitrogen dissolved in the tissues at sea level has a tension of about 0.79 atm and leaves solution to form bubbles at altitude. Flying after diving increases the risk of DCS if additional nitrogen remains in the tissues after a dive. To reduce the DCS risk from flying too soon after diving, divers are advised to wait long enough at sea level until nitrogen dissolved in their tissues is eliminated harmlessly through the lungs. The *US Navy Diving Manual*[1] provides a table of preflight surface intervals before flying is considered safe. These surface intervals range from 0 to 24 hours depending on the flight altitude and the severity of the previous diving exposures. The US Air Force re-

EXHIBIT 31-5

HAZARDOUS MARINE LIFE

Hazards to humans in the marine environment in order of frequency of injury are (*a*) dermal irritations or infections, (*b*) stings or envenomations, (*c*) poisonings from ingestion, and (*d*) trauma or attack. The first category includes coral or barnacle cuts, seaweed dermatitis, sea cucumber or sponge irritation, sea louse or annelid worm bite, bristleworm sting, bathers' itch, fish handlers' disease, marine granuloma, and parasites. Envenomations result from stingray, scorpionfish, toadfish, catfish, weeverfish, starfish, sea urchins, jellyfish, fire coral, cone shells, octopus, sea snake, crown of thorns, and various worms. Poisonings include or can occur from scromboid (pseudoallergic), tetrodotoxin/fugu (pufferfish), ciguatera, paralytic shellfish, hallucinatory fish, and fish blood, roe, or liver. Attacks or trauma can occur from sharks, barracuda, moray eels, sea lions, triggerfish, surgeonfish, crocodiles, electric eels, swordfish, and giant clams.

The sources listed below should be consulted for specific recommendations, but the usual ABCs of first aid—ensure that the casualty's *a*irway is patent, is *b*reathing without assistance, and *c*irculation is intact—are the rescuer's primary concern, followed by evacuating the casualty to a competent medical facility. Wound cleansing and antibiotics can be particularly important to remove debris and bacteria and to control infection. Allergic reactions are usually minor but can be life-threatening. The Divers Alert Network can be called at (919) 684-8111 or (800) 684-4DAN for advice or referral to local authorities.

Sources: (1) Edmonds C. *Dangerous Marine Creatures*. Flagstaff, Ariz: Best Publishing Co; 1995. (2) Auerbach PS. *Hazardous Marine Life*. Flagstaff, Ariz: Best Publishing Co; 1997.

quires a 24-hour wait after any diving before flight.[24]

Diving at altitude also increases the risk of DCS. Two factors are relevant:

1. Because the bodies of sea-level residents are in equilibrium with the 0.79 atm of nitrogen in atmospheric air, rapid ascent to an altitude of, say, 5,486 m (18,000 ft), where the atmospheric nitrogen is only 0.4 atm, would cause a supersaturation of some 0.39 atm. The supersaturated nitrogen dissipates over about 24 hours, but while it is present, no-stop dive times (see Table 31-1) must be reduced or additional decompression time must be given.
2. After equilibration is complete, the no-stop times are still shorter than those given in Table 31-1 because bubbles grow larger at reduced barometric pressure than at sea level. There are empirical modifications to existing decompression tables and dive computer models for altitude diving. The *US Navy Diving Manual*[1] contains altitude diving procedures relevant to military diving.

The decompression problem can be avoided entirely by diving with pure oxygen if the depth is limited to about 10 msw (33 fsw). As oxygen scuba weighs less than compressed-air scuba, this is an important consideration for altitude expeditions, whose members must carry their own equipment.

The Safety of Decompression Practice

Advances in our knowledge of decompression physiology since 1950 have been of little help in developing the decompression algorithms used by dive tables and dive computers. These algorithms are largely empirical, and their uncertainty is reflected by the dissimilarity of the computed dives (see Table 31-1). Dives are still classified as "safe" or "unsafe" according to whether some calculated measure of the excess inert gas supersaturation exceeds a threshold, but this approach has started to change with the introduction of probabilistic decompression modeling by the US Navy.[25] Although the absolute accuracy of probabilities estimated by these methods is uncertain, they allow dissimilar dives to be compared and are helpful for understanding the ambiguity of existing tables and dive computers.

Figure 31-21 shows a series of two no-stop dives to a depth of 16.5 msw (55 fsw), separated by a surface interval of 57 minutes.[17] The bottom times of both dives were chosen to give the longest allowed no-stop exposures. Residual nitrogen remaining from the first dive makes the second dive shorter. Table 31-2 gives the bottom times of the two dives for the 11 dive tables and dive computers listed in Table 31-1. The first dive times range from 50 to 77 minutes, while the times for the second dive range from 0 to 45 minutes. The wide range of the second dive times underscores the uncertainty of our knowledge of decompression safety and the underlying mechanisms of DCS. We know which time is safest (0 min), but we must make a subjective judgment as to which times are safe *enough* (ie, of acceptable DCS risk; see Chapter 30, Physics, Physiology, and Medicine of Diving).

The probabilities that DCS will develop are also given in Table 31-2, estimated at the end of the second dive by the US Navy decompression algorithm.[25] Second-dive times of 0 to 45 minutes correspond to DCS probabilities of 1.9% to 3.2%. Thus, DCS probability appears to vary gradually over a wide range of dives, and dissimilar dive profiles can have similar DCS probabilities. At present, insufficient experimental or observational data are available to confirm the estimated probabilities, although the estimates are suspected to exceed the true values. As such data and more powerful microprocessors become available, probabilistic algorithms are expected to replace the algorithms currently used by dive tables and dive computers.

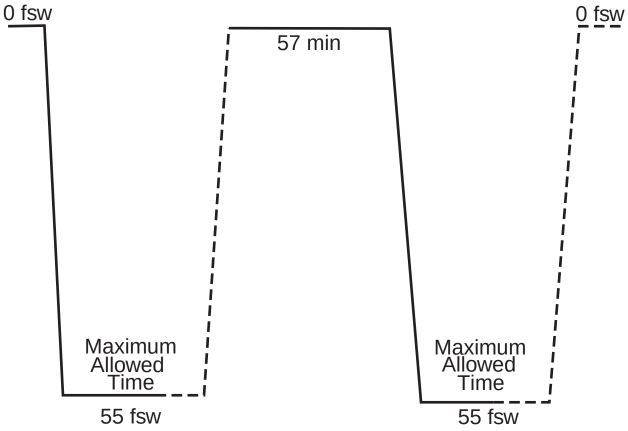


Fig. 31-21. A two-dive profile for comparing the repetitive dive exposure limits of the dive tables and dive computers listed in Table 31-1. Two no-stop exposures to 16.5 msw (55 fsw) are separated by a surface interval of 57 minutes. The maximum allowable exposure times (indicated by dashed lines) are chosen for each dive according to the requirements of the particular dive table or dive computer.

TABLE 31-2

NO-STOP BOTTOM TIMES FOR TWO DIVE PROFILES IN FIGURE 31-21*

Dive Table or Computer Model	1st Dive† (min)	2nd Dive‡ (min)	*P*DCS§ (%)
EDGE	62	45	3.2
Aladin	51	41	3.2
DataMaster	61	39	3.2
Monitor	51	35	2.9
DSAT Tables	54	26	2.7
DCIEM Tables	50	23	2.5
93 USN Tables	75	17	3.2
56 USN Tables	60	8	2.3
Datascan II	57	5	2.1
RN Tables	60	0	1.9
BSAC Tables	51	0	1.9

*times as specified by 6 dive tables and 5 dive computers (see Table 31-1).

†depth: 16.5 msw (55 fsw); surface interval between dives: 57 min

‡depth: 16.5 msw (55 fsw)

§Probabilities of decompression sickness (*P*DCS) estimated from USN 93 algorithm in Weathersby PK, Survanshi SS, Homer LD, Parker E, Thalmann ED. Predicting the time of occurrence of decompression sickness. *J Appl Physiol*. 1992;72(4):1541–1548

BSAC: British Sub Aqua Club; DCIEM: Defense Civil Institute of Environmental Medicine [Canada]; DSAT: Diving Science and Technology (United States); EDGE: Electronic Dive Guide Experience; RN: Royal Navy; USN: US Navy

Data source: Lewis JE, Shreeves KW. *The Recreational Diver's Guide to Decompression Theory, Dive Tables, and Dive Computers*. Santa Ana, Calif: Professional Association of Diving Instructors; 1990.

SATURATION DIVING

After about 24 hours at any depth, the inert gas tension in the body reaches equilibrium with the inert gas partial pressure in the ambient atmosphere, and the decompression time achieves its maximum length, independent of dive duration. While saturation dives are logistically complex, they avoid the stresses of multiple bounce dive decompressions in circumstances where long working times are desirable. Saturation diving is used most often in commercial diving, occasionally in scientific diving, and by the Navy for submarine rescue.

Saturation divers usually live in a surface chamber known as a Deck Decompression Chamber (DDC) at a "storage" pressure that is shallower than the dive site at which they work. If the chamber is on a surface ship, the divers transfer to a Personnel Transfer Capsule (PTC) through a mating hatch in the DDC, and the PTC is lowered to above or below the dive site. Figure 31-22 shows a saturation diving system with one PTC and two DDCs. Saturation diving may also be conducted from sea-floor habitats, but few of these are now in use as they are expensive and difficult to maintain.

If a diver is stored at a given depth and the worksite is deeper than the storage depth, he makes a descending, or downward, excursion to the worksite. Ascending, or upward, excursions from storage depth are usually made from underwater habitats. The *US Navy Diving Manual*[1] provides tables for excursions from various saturation depths, following which divers may return to the storage pressure without decompression stops. Downward excursions are most common, with the DDC storage depth chosen as shallow as the operation of the excursion tables allow. This minimizes the pressure at which the divers must live and the length of the final decompression to the surface. Oxygen partial pressures during excursions range from 0.4 to 1.2 atm, depending on the breathing apparatus used and the type of operation. Because helium is expensive and not readily available and because open-circuit equipment (scuba or surface-supplied) uses large gas volumes as the depth increases, exhaled gases are sometimes returned to the PTC or the surface, where they are reconditioned for reuse by removing carbon dioxide and adding oxygen.

Atmospheric Control

A saturation chamber is a closed environment whose atmosphere must be carefully controlled to maintain diver health. As the saturation depth increases, the oxygen percentage decreases so that the oxygen partial pressure does not exceed 0.5 atm, which is the approximate threshold for pulmonary oxygen toxicity (see Chapter 30, Physics, Physiology, and Medicine of Diving, for a discussion of breathing gas toxicities). At a depth of 300 msw (1,000 fsw), for example, the oxygen percentage must not exceed 1.6%.

The balance of the pressure is made up by inert gas, which is nitrogen for dives to depths of about 36 msw (120 fsw) and helium at greater depths. Most saturation diving occurs deeper than 61 msw (200 fsw), with helium as the inert gas. At depths of 300 msw (1,000 fsw) and deeper, a trimix of helium–nitrogen–oxygen or a mix of hydrogen–helium–oxygen is sometimes used to reduce the high-pressure nervous syndrome (HPNS), but it is uncertain if the nitrogen ameliorates the HPNS or only relieves some of its symptoms. The Navy currently has procedures only for helium–oxygen.[1]

Accurate gas analysis is essential to maintain safe levels of oxygen and carbon dioxide. The carbon dioxide level is typically controlled to less than 0.5% surface equivalent (SEV) by absorbent material in a closed-loop life-support system. Chamber ventilation must be adequate to keep the atmosphere mixed. In a helium atmosphere, heavier gases such as carbon dioxide and oxygen tend to pool in low or poorly ventilated areas, and toxic levels of carbon dioxide have occurred in bunks that are isolated by curtains.

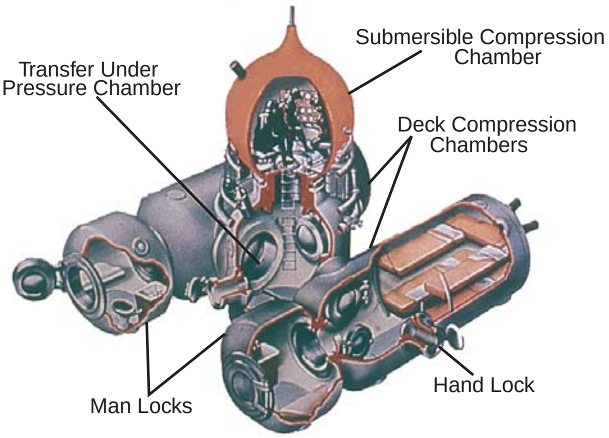


Fig. 31-22. A saturation diving system (diameter ~ 2 m) with two deck compression chambers (DDCs), two man locks, two medical (hand) locks, and a personnel transfer capsule (PTC, called a submersible compression chamber in this figure). The transfer under pressure chamber allows working divers to be moved between the worksite and living chambers (called deck compression chambers in this figure) without disturbing the off-duty divers. The man and medical locks are for transferring personnel, food, medicine, reading material, laundry, and the like to and from sea level.

Toxic atmospheric contaminants such as carbon monoxide or hydrocarbons can be eliminated only by flushing the chamber and piping systems with fresh gas, but this is costly and may be impossible on a ship with limited gas supplies. Contaminants must be prevented from entering the chamber. Carbon monoxide is produced at a rate of 8 to 10 mL per person per day by the metabolism of hemoglobin and must be monitored, but unsafe levels from this source are unusual.

Hydrocarbons can be introduced from petroleum lubricants, leaks in life-support refrigeration units, or improperly cleaned piping. Petroleum lubricants are a fire hazard as well as source of pollution. Divers produce methane at a rate of 300 to 500 mL per person per day. Some chamber systems lock out human waste immediately; others hold it in a sanitary tank. Human waste allowed to sit in a sanitary tank produces ammonia, indole, skatole, sulfur dioxide, hydrogen sulfide, chlorine, and carbon dioxide; therefore, sanitary tanks should be vented externally to prevent contaminating the chamber atmosphere. Many naturally occurring contaminants can be removed by filters in the life-support loop. Mercury is prohibited, and instruments or components such as mercury thermometers and electrical mercury switches must be avoided. The basic rule for preventing contamination is "if in doubt, keep it out."

Infection

Hygiene is important in a closed environment. High humidity leads to external otitis and skin infection. The current recommendation for preventing external otitis is to instill a solution of aluminum acetate with 2% acetic acid into each ear every morning and evening, and before and after diving. The external ear canals are filled with this solution for 5 minutes while the diver lies on one side and then the other. Bed linen should be changed every 48 hours and daily showers taken whether diving or not. Chamber surfaces require daily cleaning with a nonionic detergent solution, and the bilges should be rinsed and drained at the same time. Food spills and the like should be cleaned immediately. Divers should wear their own thermal protection suits to prevent the spread of skin infections. Suits should be rinsed with nonionic detergent and water and be hung to dry.

Hyperbaric Arthralgia

Hyperbaric arthralgia is joint discomfort or pain that occurs during compression and decreases in intensity during 24 hours or more at constant depth. Symptoms include joint cracking, a sensation of "dry and grainy" joints (sometimes described as "no joint juice"), and a feeling similar to sprain. In order of severity, affected joints are the shoulders, knees, wrists, hips, and back. Least affected joints are the ankles, fingers, and elbows. Hyperbaric arthralgia can occur during short bounce dives but is more common during deep saturation dives. Its frequency, severity, and duration increase with depth and compression rate, but task performance is usually not affected. The condition is less common with the slow compression rates used to alleviate HPNS. The origin of hyperbaric arthralgia is unknown but suggested causes are (1) changes in the nature of bubble formation with increasing pressure and (2) changes in joint fluid osmolarity leading to dehydrated articular cartilage. There is no evidence that hyperbaric arthralgia leads to joint degeneration or aseptic bone necrosis.

Depth Limits

HPNS begins at about 180 msw (600 fsw) and is manifested by tremor, decreased motor and intellectual performance, dizziness, nausea, vomiting, and, occasionally, psychosis. Focal reflex changes sometimes occur and balance may be affected. Deeper than 300 msw (1,000 fsw), electroencephalograms may have slow theta waves, and alpha activity may be depressed. Divers easily fall into microsleep if not continually aroused. HPNS symptoms can be reduced but not eliminated by slowing or interrupting compression as depth increases.

Although there is considerable individual variability, HPNS imposes a limit of not much more than 600 msw (2,000 fsw) as the maximum depth that humans can tolerate under dry conditions. The maximum depth at which practical work in the water is possible is less than 600 msw owing to excess work of breathing in the UBA, particularly during exhalation.

Decompression

Saturation decompression must occur very slowly to avoid DCS. For helium–oxygen, the US Navy uses a continuous reduction of pressure according to the following schedule:

- 1.8 msw/h from 480 to 60 msw (6 fsw/h from 1,600–200 fsw);
- 1.5 msw/h from 60 to 30 msw (5 fsw/h from 200–100 fsw);
- 1.2 msw/h from 30 to 15 msw (4 fsw/h from 100–50 fsw); and
- 0.9 msw/h from 15 to 0 msw (3 fsw/h from 50–0 fsw).[1]

To minimize decompression during sleep, the US Navy schedule stops decompression from midnight to 0600 and from 1400 to 1600. The oxygen partial pressure is maintained at 0.4 to 0.45 atm until 10 msw (33 fsw) and at 20% to 30% to the surface. The Royal Navy decreases the pressure in stages with periodic drops of 5 msw (16.5 fsw) over 5 minutes. Divers are required to remain near a recompression chamber for at least 2 hours after decompression and within 30 minutes' travel time to a chamber for 48 hours. Flying is prohibited for 72 hours.

THERMAL PROTECTION AND BUOYANCY

Most of Earth's waters are well below body temperature, and except for short exposures, the unprotected diver is at risk of hypothermia; *hyper*thermia, on the other hand, is an unusual hazard for divers (Exhibit 31-6). The *US Navy Diving Manual*[1] gives allowable exposure durations as a function of temperature and the means of thermal protection. Buoyancy control is closely linked to thermal protection, as almost all thermal protection methods use gas for insulation.

Historically, the hard-hat diver wore heavy woolen underwear beneath a canvas outer suit. The suit was supposed to remain dry but frequently leaked. Because the suit was filled with air, the diver wore heavy boots and weights to achieve the negative buoyancy needed to walk on the bottom. To maintain proper buoyancy the suit had to be inflated during descent and deflated during ascent. Hard-hat divers could suffer severe injury from suit squeeze (ie, barotrauma that occurs when a poorly

EXHIBIT 31-6

DIVING IN WARM WATER AND IN CONTAMINATED WATER

Hyperthermia is rare but can occur in special circumstances. At remote diving sites in hot climates, recompression chambers that treat diving casualties and deck decompression chambers (DDCs) that support saturation diving may be outdoors and exposed to the sun. This resulted in two fatalities during a saturation diving operation in the 1980s. Hyperthermia is less likely during open water diving, but US Navy divers in the Persian Gulf during the Persian Gulf War in 1991 were exposed to water temperatures in excess of 32°C (90°F) and wore ice vests to prevent overheating. Divers in confined waters (eg, in or around power plants) may also be exposed to temperatures that can cause hyperthermia. This is a particular problem in hazardous environments such as nuclear reactors, where special suits and breathing apparatuses are worn to prevent inward leaks of water contaminated by radioactive material. These suits may incorporate closed-circuit cooling water to prevent the divers' overheating. In addition to being leak proof, suits used in water or other liquids that are contaminated with biological agents or toxic chemicals must be made of materials that will not degrade during exposure. Before the diver undresses, the suits are thoroughly washed down to prevent harmful exposure of the diver himself or of support personnel.

fitting suit is insufficiently expanded; see Chapter 30, Physics, Physiology, and Medicine of Diving) if the air supply was inadequate to maintain suit volume during descent. During ascent, gas was vented from the suit to avoid uncontrolled positive buoyancy (ie, blow-up) in which the diver was propelled to the surface and risked air embolism, DCS, or mechanical injury from collision.

The wetsuit is the most common form of thermal protection used today. Made of air-filled, closed-cell neoprene foam, wetsuits are satisfactory for several hours at temperatures of 10°C to 15.5°C (50°F–60°F) but provide less protection with increasing depth as the air-filled cells compress. Minor suit squeeze sometimes occurs with tight-fitting wetsuits. As a diver descends, wetsuit compression reduces buoyancy by several pounds. A buoyancy compensator (BC) to which gas can be added or removed is typically used to make adjustments from slightly positive to slightly negative buoyancy, according to whether ascent or descent is desired. With open-circuit scuba, buoyancy increases by several pounds as compressed gas is consumed from the tanks. Swimming with fins is the common mode of propulsion with scuba and helps a diver remain warm for several hours.

The next level of thermal protection, the drysuit, is a waterproof outer garment over insulating underwear that is sufficiently warm for brief periods of ice diving. Drysuit diving requires training in buoyancy control to avoid suit squeeze or blow-up. For an untethered scuba diver in deep water, moreover, an uncontrolled descent in a drysuit could be fatal. For an untethered scuba diver in deep water wearing a drysuit, a suit squeeze could make the diver negatively bouyant, resulting in a fatal, uncontrolled descent. A blow-up from overinflation of the suit with uncontrolled ascent, on the other hand, could result in serious or fatal AGE or DCS. A diaper or other urine-collection device is essential if the drysuit is to remain dry during a dive of several hours' duration. Drysuits provide inadequate thermal protection during 6- to 8-hour exposures in –1.1°C to 4.4°C (30°F–40°F) water for resting divers, such as the operators or passengers of SDVs. Because of its greater bulk, a drysuit is more difficult to swim in than a wetsuit.

Drysuits can be inflated from the diver's breathing gas or from a separate gas supply. Argon is sometimes used for this purpose because of its good insulating properties. Helium, on the other hand, offers particular thermal challenges in diving. Helium is a poor insulator because its thermal conductivity is 4.8 times greater and its heat capacity 2.3 times greater than that of air. These characteristics cause heat loss to increase with depth. Most heat loss occurs by convection through the skin and lungs; as a result, for normal body temperature to be maintained, the ambient temperature in a saturation chamber must be 29.4°C to 32.2°C (85°F–90°F). The range of thermal comfort narrows with increasing depth. Water vapor diffuses slowly at high pressure, and evaporation provides little cooling, which makes the skin feel wet without evidence of sensible water. The comfort range for relative humidity is 50% to 70%.

Wetsuits and drysuits provide passive insulation, which delays but does not prevent body cooling. Active heating is the most effective thermal protection. The most common active heat source is hot water (not to exceed 43°C) supplied from the surface or PTC to a loose-fitting wetsuit through which hot water flows before it exits at the hands and feet (see Figure 31-9). An even distribution of flow and careful temperature control are critical for adequate heating without causing hot spots or thermal burns. During deep helium–oxygen diving, the breathing gas must be heated to prevent convective heat loss through the lungs, as this can cause a progressive hypothermia that may go unnoticed. Electric suits are under development as an alternative active heat source (particularly for SDVs) but are complex and expensive.

TREATMENT OF DECOMPRESSION SICKNESS AND ARTERIAL GAS EMBOLISM

AGE is the result of pulmonary barotrauma, while DCS is caused by the formation of bubbles in the blood and tissues. As such, the immediate goal of therapy is to reduce the volume of the offending bubbles. This may be possible for patients who are treated shortly after symptom onset, but long delays are common, and gas bubbles may have caused physical or biochemical damage that persists after the bubbles themselves have resolved. In such situations, therapy can still be beneficial by oxygenating poorly perfused tissue or by reducing edema.

The definitive treatment for DCS and AGE is increased atmospheric pressure and 100% oxygen. The additional pressure serves to reduce the size of the bubbles, and oxygen accelerates their resolution by causing nitrogen to diffuse from bubble to blood. On reaching the lungs, excess nitrogen from the blood is exhaled. Pressure (or recompression) was first used in 1909 to treat DCS (what was then called caisson disease), but oxygen was not routinely used during recompression until the 1960s.[26]

The best first aid for AGE or DCS is 100% oxygen delivered by mask. Inspired oxygen percentages near 100% are essential for the greatest effect. As injured divers are often dehydrated, owing either to the illness or to lack of sufficient fluids, rehydration is also important—orally if possible and intravenously if necessary. There are no proven adjuvant therapies, although aspirin is sometimes recommended to inhibit platelet aggregation, and steroids, to reduce edema. Lidocaine has been proposed for DCS with spinal cord involvement.

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

Therapy According to US Navy Treatment Tables

The standard US Navy therapy for DCS or AGE is recompression to 18 msw (60 fsw; 2.8 ata) while the patient is breathing 100% oxygen. Typically, the course of treatment is determined by the response of the signs and symptoms. US Navy Treatment Table 5 (Figure 31-23) may be used if

- the only symptoms are joint or limb pain, itching, rash, or local swelling;
- the absence of neurological findings is verified by physical examination; and
- the symptoms are completely relieved within 10 minutes of oxygen breathing at 18 msw (60 fsw).

Treatment Table 5 requires 135 minutes to administer, with two oxygen periods at 18 msw (60 fsw) and one oxygen period at 9 msw (30 fsw).[1] The oxygen periods at 18 msw are 20 minutes long followed by 5 minutes of air breathing to reduce the risk of CNS oxygen toxicity. Ascent from 18 to 9 msw (60–30 fsw) and 9 to 0 msw (30–0 fsw) occurs at 0.33 msw/min (1 fsw/min).

US Navy Treatment Table 6 (Figure 31-24) is used if

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

Treatment Table 6 requires 285 minutes to administer and has three 25-minute cycles at 18 msw (60 fsw) and two 75-minute cycles (60 min O_2, 15 min air) at 9 msw (30 fsw). Treatment Table 6 can be extended by up to two cycles at 18 msw (60 fsw) and two cycles at 9 msw (30 fsw).[1]

For patients with serious or life-threatening conditions, particularly those suggesting AGE, the option

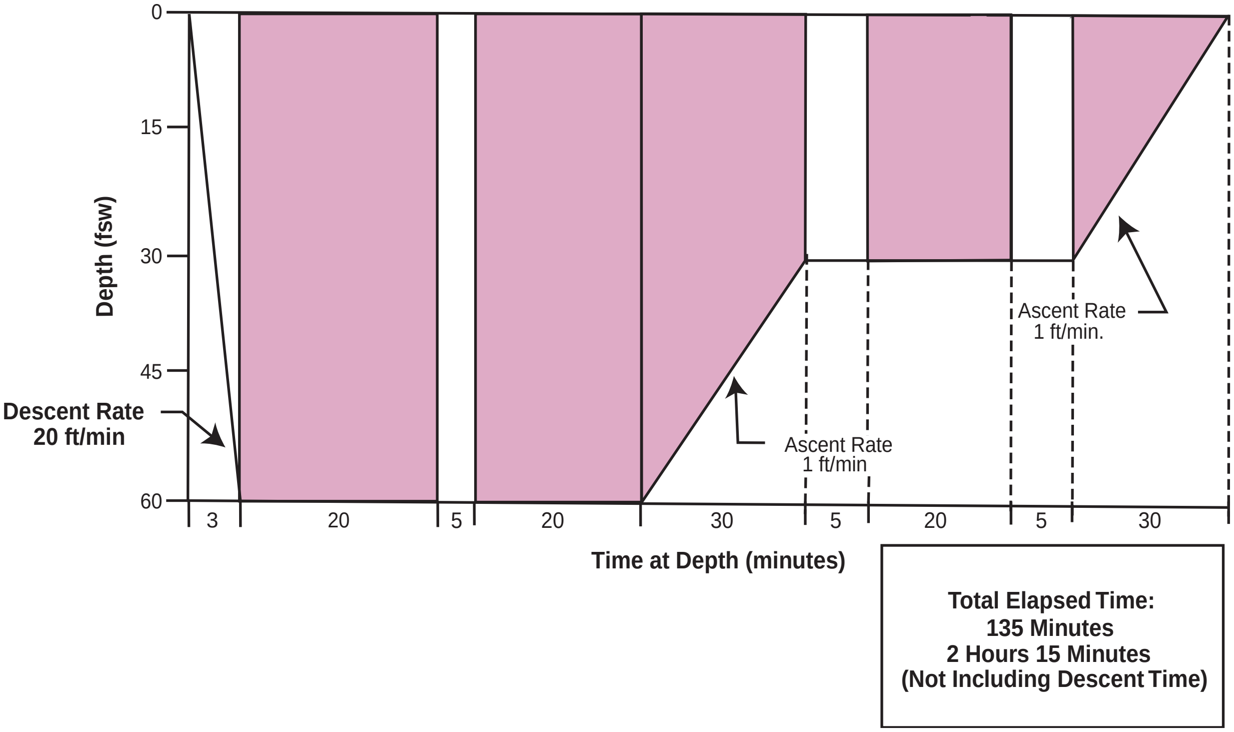


Fig. 31-23. The US Navy Treatment Table 5 Depth/Time Profile can be used only if the diving casualty has no neurological symptoms and if all symptoms are relieved within 10 minutes at 18 msw (60 fsw). Consult the *US Navy Diving Manual* before using this or other treatment tables. Reprinted from US Department of the Navy. *US Navy Diving Manual*. SS521-Ag-PRO-010. Washington, DC: Naval Sea Systems Command. Rev 4 (20 Jan 1999); Change A (1 Mar 2001): p21-40. NAVSEA 0994-LP-100-3199.

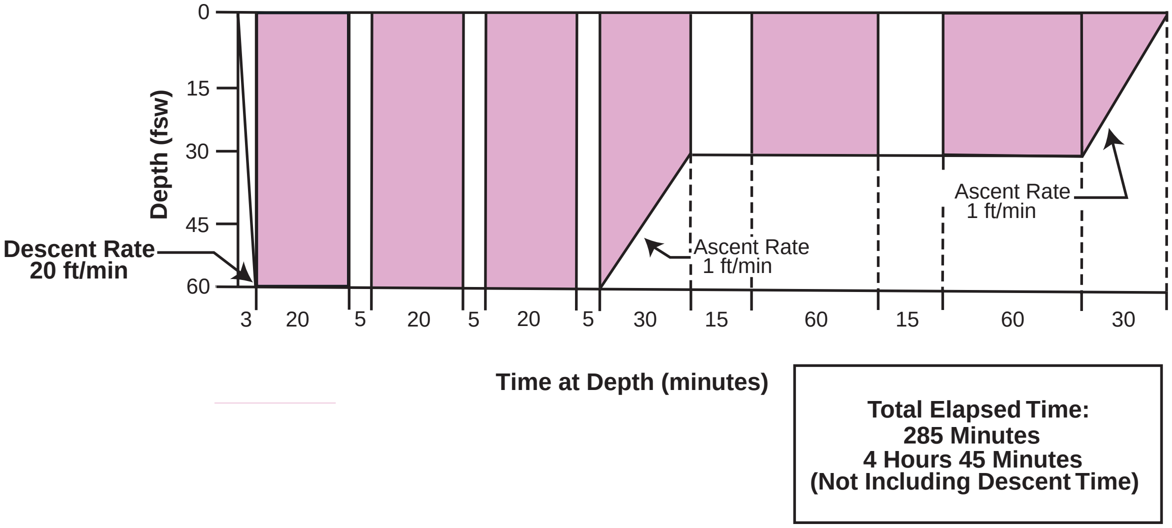


Fig. 31-24. In the US Navy Treatment Table 6 Depth/Time Profile, three 25-minute oxygen cycles at 18 msw (60 fsw) are standard, but two additional cycles may be added. Two additional 75-minute oxygen cycles may also be added at 9 msw (30 fsw). The decompression requirements of inside attendants must also be considered so that they do not themselves develop decompression sickness. Consult the *US Navy Diving Manual* before using this or other treatment tables. Reprinted from US Department of the Navy. *US Navy Diving Manual*. SS521-Ag-PRO-010. Washington, DC: Naval Sea Systems Command. Rev 4 (20 Jan 1999); Change A (1 Mar 2001): p21-41. NAVSEA 0994-LP-100-3199.

is available for further compression from 18 to 50 msw (60–165 fsw) if the patient does not stabilize during the first oxygen period at 18 msw (60 fsw). A recompression to 50 msw (165 fsw) is known as Treatment Table 6A (Figure 31-25 and Exhibit 31-7).[1] Compression to 50 msw (165 fsw) can be accomplished with air or 40% to 50% oxygen in nitrogen. The high-oxygen mix is beneficial for reducing nitrogen narcosis and additional nitrogen uptake at 50 msw (165 fsw). If relief is complete at 50 msw (165 fsw) within 30 minutes, the patient can be decompressed at 0.91 msw/min (3 fsw/min) to 18 msw (60 fsw) (NOTE: procedures for Treatment Table 6 are now followed).

Should relief at 50 msw (165 fsw) be unsatisfactory after 30 minutes, the time at depth can be extended to 120 minutes, after which decompression to 18 msw (60 fsw) is accomplished using US Navy Treatment Table 4 (Figure 31-26).[1] When the patient arrives at 18 msw (60 fsw), decompression may continue according to Treatment Table 4 if either the patient's condition is satisfactory or the recompression chamber can be held at 18 msw (60 fsw) for at least 12 hours, followed by decompression according to US Navy Treatment Table 7 (Figure 31-27). Patients with residual symptoms at the end of an extended treatment on Treatment Table 6 or Treatment Table 6A, or patients who cannot tolerate further oxygen due to pulmonary toxicity, may be given repetitive treatments on subsequent days. (Mild pulmonary toxicity is reversible within hours to a day.) Typically, one to six additional treatments are given, which may be according to Treatment Tables 5 or 6, or sometimes at 9 msw (30 fsw) for 60 to 90 minutes. The rule of thumb is that repetitive treatments end when no clinical improvement can be demonstrated during two successive recompressions.

For a seriously ill patient, saturation therapy is an alternative to multiple repetitive recompressions. During saturation treatment, the patient is maintained at 18 msw (60 fsw) until he or she stabilizes. This avoids the nontherapeutic decompression to sea level, which may cause symptoms to return. After at least 12 hours at 18 msw (60 fsw) during which the patient may breathe oxygen and air in 25-minute cycles (pulmonary toxicity allowing), decompression is conducted according to Treatment Table 7 over 56 hours.[1] Saturation therapy should not be attempted without adequate facilities and personnel. A scrubber for removing carbon dioxide is required, as is staffing to support 24-hour opera-

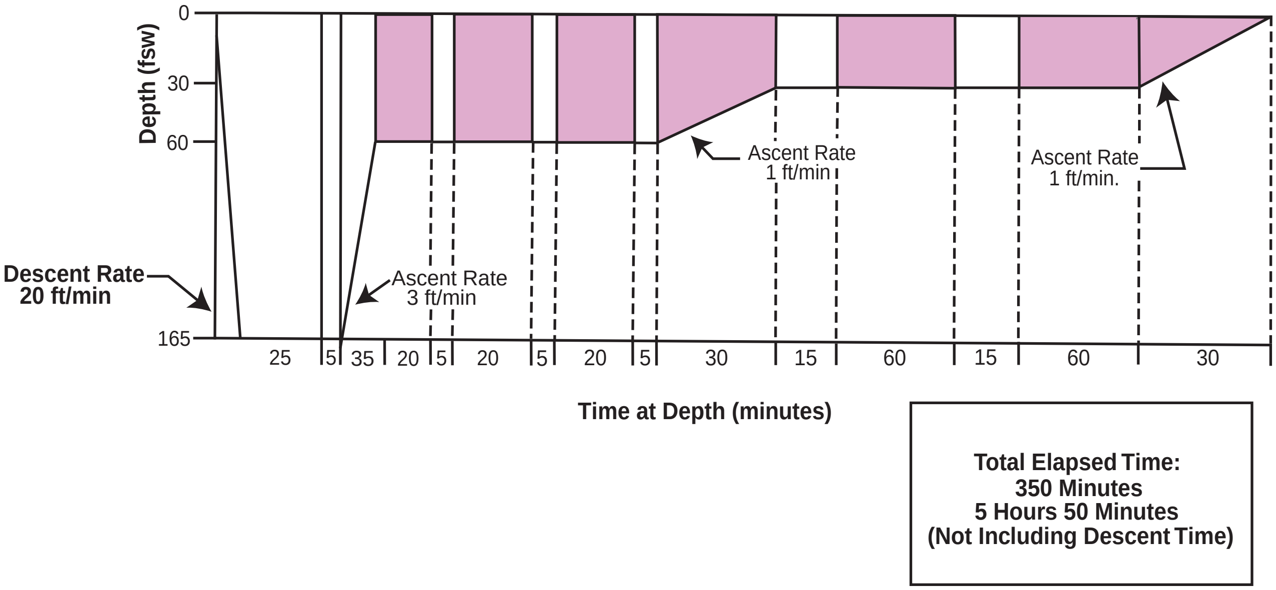


Fig. 31-25. In the US Navy Treatment Table 6A Depth/Time Profile, as long as 20 minutes may be spent at 18 msw (60 fsw) before making the decision to compress to 50 msw (165 fsw). The advantage of compression to 50 msw (165 fsw) over 18 msw (60 fsw) is greater reduction in bubble volume. A spherical bubble is compressed to 35% of its original volume at 18 msw (60 fsw) and to 17% at 50 msw (165 fsw). At 50 msw (165 fsw), high-oxygen treatment gas may be breathed to reduce nitrogen uptake. Consult the *US Navy Diving Manual* before using this or other treatment tables. Reprinted from US Department of the Navy. *US Navy Diving Manual.* SS521-Ag-PRO-010. Washington, DC: Naval Sea Systems Command. Rev 4 (20 Jan 1999); Change A (1 Mar 2001): p21-42. NAVSEA 0994-LP-100-3199.

EXHIBIT 31-7

IS RECOMPRESSION TO 50 MSW NECESSARY, OR IS RECOMPRESSION TO 18 MSW ADEQUATE?

When Treatment Tables 5, 6, and 6A were introduced in the 1960s, Table 6A was specifically prescribed for treating arterial gas embolism (AGE). Subsequently, AGE was recognized to be difficult to diagnose, and treatment at 18 msw (60 fsw) was found in experiments with animals to be as good as treatment at 50 msw (165 fsw). Some people recommended that treatment at 50 msw (165 fsw) be abandoned in favor of treatment at 18 msw (60 fsw), but others argued that success was not always achieved at 18 msw (60 fsw) and further recompression to 50 msw (165 fsw) was sometimes required. This unresolved controversy was reviewed by Thalman in 1996.[1] The *US Navy Diving Manual*[2] adopted a compromise in which treatment for all forms of decompression illness could begin at 18 msw (60 fsw) with the option for further descent to 50 msw (165 fsw) if indicated by inadequate resolution at 18 msw (60 fsw).

The drawbacks of recompression to 50 msw (165 fsw) must be recognized. The patient and attendants are subject to nitrogen narcosis, fatigue can be significant for those inside and outside the chamber, and there is additional risk of decompression sickness. The use of helium–oxygen has been proposed as an alternative gas to air for recompression deeper than 18 msw (60 fsw) and is undergoing study.

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[1] Thalmann ED. Principles of US Navy recompression treatments for decompression sickness. In: Moon RE, Sheffield PJ, eds. *Treatment of Decompression Illness.* Kensington, Md: 45th Undersea and Hyperbaric Medical Society Workshop, June 1996: 75–95.

[2] US Department of the Navy. *US Navy Diving Manual*. SS521-Ag-PRO-010. Washington, DC: Naval Sea Systems Command. Rev 4 (20 Jan 1999); Change A (1 Mar 2001). NAVSEA 0994-LP-100-3199.

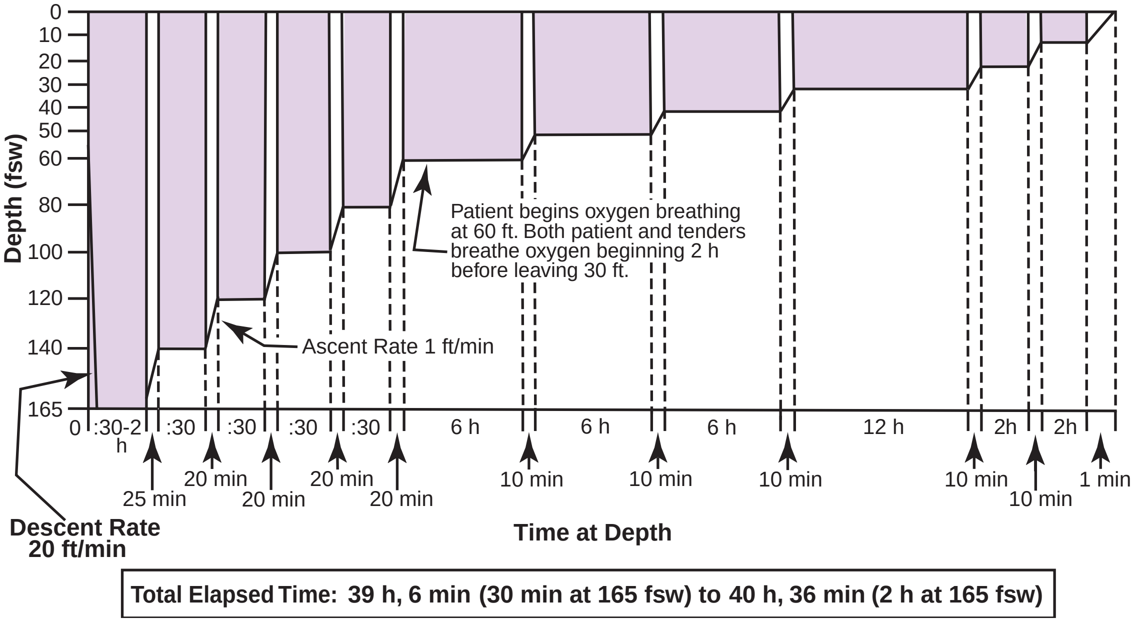

Fig. 31-26. In the US Navy Treatment Table 4 Depth/Time Profile, time at 50 msw (165 fsw) is from 30 minutes to 2 hours. At 50 msw (165 fsw), high-oxygen treatment gas may be breathed to reduce oxygen uptake. Oxygen breathing at 18 msw (60 fsw) is in cycles of 25 minutes interrupted by 5 minutes of air breathing. Consult the *US Navy Diving Manual* before using this or other treatment tables. Reprinted from US Department of the Navy. *US Navy Diving Manual.* SS521-Ag-PRO-010. Washington, DC: Naval Sea Systems Command. Rev 4 (20 Jan 1999); Change A (1 Mar 2001): p21-43. NAVSEA 0994-LP-100-3199.

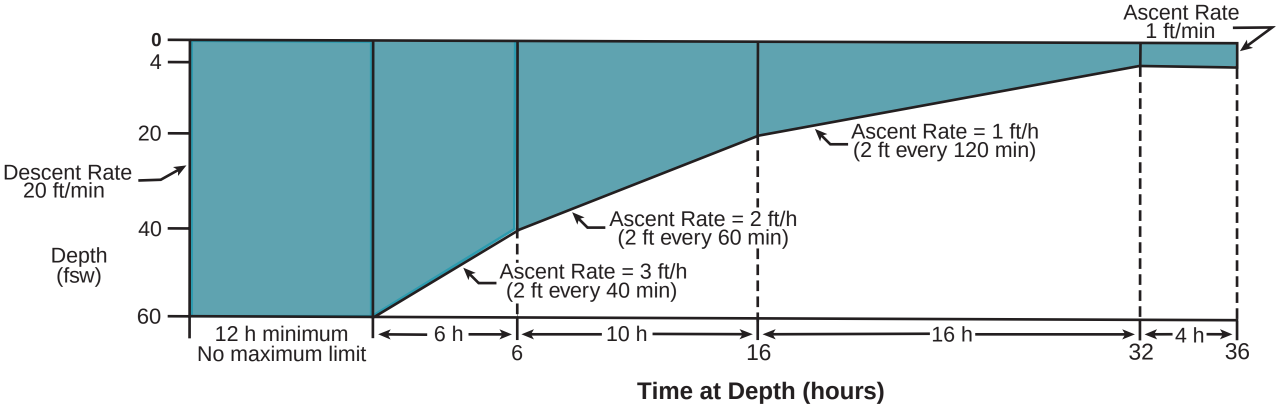

Fig. 31-27. The US Navy Treatment Table 7 Depth/Time Profile is an extension at a depth of 60 fsw of Treatment Tables 6, 6A, and 4 (Figures 31-24 through 31-26). At 50 msw (165 fsw), high-oxygen treatment gas may be breathed to reduce nitrogen uptake. Oxygen breathing at 18 msw (60 fsw) is in cycles of 25 minutes interrupted by 5 minutes of air breathing. Consult the *US Navy Diving Manual* before using this or other treatment tables. Reprinted from US Department of the Navy. *US Navy Diving Manual.* SS521-Ag-PRO-010. Washington, DC: Naval Sea Systems Command. Rev 4 (20 Jan 1999); Change A (1 Mar 2001): p21-43. NAVSEA 0994-LP-100-3199.

tions. During complex or extended treatments, careful planning is required to ensure that the decompression status of the inside attendants remains satisfactory, as they generally do not breathe as much oxygen as the patient.

The best type of chamber for treating a patient with DCS is large enough to accommodate a medical attendant as well as the patient. The closest available chamber, however, may be used for hyperbaric oxygen therapy and can accommodate only a single person. Such “monoplace” chambers and hyperbaric oxygen therapy are described in Exhibit 31-8.

Decompression Sickness in Saturation Diving

DCS that occurs during or after the slow decompression from saturation dives usually manifests as mild knee or leg pain. Neurological symptoms are very rare. The onset of DCS is subtle and may begin with aching or a feeling in the anterior thighs similar to that after hard exercise. This is treated by recompression and administration of high oxygen partial pressures. For occurrences under pressure, oxygen partial pressures of 1.5 to 2.5 atm in helium–oxygen are administered as in Treatment Table 6, with a cycle of 20 minutes of high–partial pressure oxygen (1.5–2.5 atm and 5 min of low–partial pressure oxygen [0.3–0.5 atm]).

Recompression can occur in 3-msw (10-fsw) stages until attaining the depth of relief at which the diver is held for 2 to 6 hours, depending on response. The standard saturation decompression schedule is resumed on relief of symptoms. Relief usually occurs within 10 msw (33 fsw) of the onset depth, but further recompression is not recommended if symptoms are not relieved within 20 msw (66 fsw), as the pain may not be caused by DCS, and other causes should be investigated. For DCS that occurs after reaching the surface and breathing air, a diver may be treated according to Treatment Table 6.

DCS that occurs 60 minutes or more after an ascending excursion can be treated as described above, but DCS that occurs within 60 minutes of ascent from an excursion dive deeper than the saturation depth should be considered serious, even if pain is the only symptom. Neurological symptoms, particularly those related to the inner ear, are not uncommon after rapid ascent from excursions. Recompression to at least the excursion depth should be immediate, and further recompression is warranted if symptoms do not improve significantly within 10 minutes. High oxygen partial pressures should be administered for at least 2 hours as described above, after which the patient should be held at depth for 12 hours before resuming standard saturation decompression.

MEDICAL STANDARDS FOR DIVING

Diving medical officers (DMOs) examine candidates for initial diving training, provide advice to diving officers concerning the medical aspects of diving operations, examine divers before or after dives, offer routine medical care, and treat divers for diving injuries. Some conditions (eg, epilepsy)

EXHIBIT 31-8

RECOMPRESSION CHAMBERS AND HYPERBARIC OXYGEN THERAPY

There are two principal types of hyperbaric chambers: multiplace and monoplace. Multiplace chambers are steel or aluminum, can accommodate two or more people, and are usually compressed with air. Their depth capability is often greater than 18 msw (60 fsw). Multiplace chambers generally have two or more compartments that allow personnel to be transferred in or out while at pressure. Depending on size, a compartment may accommodate as many as 12 patients who receive 100% oxygen by mask or “head-tent.” The compartment is periodically ventilated with air to keep the carbon dioxide level below 1.5% surface equivalent and to maintain the oxygen level below 23% to limit the fire hazard. Critical care nursing can be provided should mechanical ventilation or intravenous drug infusion be required.

A monoplace chamber is generally an acrylic cylinder that is compressed with 100% oxygen and accommodates a single patient in the supine position. Its depth capability is often 13.5 to 18 msw (45–60 fsw). Monoplace chambers do not allow for direct patient access although air breaks during oxygen breathing often can be given by mask for conscious patients. Monoplace chambers have been used effectively for decompression illness therapy, but depending on depth capability, they may not be able to provide therapy in accordance with the standard treatment tables. Because of their lower cost, there are many more monoplace chambers than multiplace chambers. Lightweight, inflatable monoplace chambers that can be compressed with air are under development. These will provide the capability for on-site treatment in remote locations or for transport under pressure to larger recompression facilities.

Decompression illness is a relatively rare phenomenon, and most recompression facilities are used more frequently to provide hyperbaric oxygen therapy for other indications. Hyperbaric oxygen therapy is recommended for conditions caused by gas bubbles, inadequate perfusion, and metabolic poisons, including

- decompression sickness, arterial gas embolism, or iatrogenic gas embolism;
- crush injury, compartment syndrome, or acute traumatic ischemia;
- blood-loss anemia;
- necrotizing soft-tissue infections such as gas gangrene or nonclostridial fasciitis;
- refractory osteomyelitis;
- osteoradionecrosis;
- compromised skin grafts or flaps;
- thermal burns;
- intracranial abscess;
- selected problem wounds; and
- carbon monoxide, cyanide, or smoke inhalation.

Recompression facilities can be located by calling The Undersea and Hyperbaric Medical Society, Bethesda, Md, at (301) 942-2980, or the Divers Alert Network, Durham, NC, at (919) 684-2948 or (919) 684-4DAN.

The rationale and methods for using hyperbaric oxygen in these conditions are described in Hampson NB. *Hyperbaric Oxygen Therapy: 1999 Committee Report*. Bethesda, Md: The Undersea and Hyperbaric Medical Society; 1999.

are absolute contraindications for diving, while others (eg, upper respiratory infection) are temporarily disqualifying or disqualifying until corrected. Table 31-3 summarizes the recommended relative and absolute contraindications. Conditions that might be allowed for recreational divers may be disqualifying for military divers; the US Navy regulations guide the practice of military diving for all services.

The *US Navy Diving Manual*[1] provides general guidelines for return to diving after DCS or AGE, but each case requires review by a DMO. For example, a diver who had complete relief of Type I DCS during Treatment Table 5 may return to diving 48 hours after therapy is complete, and a diver who required therapy with Treatment Table 6 for complete relief of Type I (minor) symptoms may resume diving after 7 days. If there is any question about the presence of Type II (serious) symptoms,

TABLE 31-3
RECOMMENDED* ABSOLUTE AND RELATIVE CONDRAINDICATIONS TO DIVING

System	Absolute Contraindications	Relative Contraindications
Ears and Upper Respiratory	Open tympanic perforation Inability to equalize the middle ear Tube myringotomy Inner ear surgery (eg, stapedectomy, ossicular chain) Permanent obstruction of the external canal Meniere's disease or other inner ear disease Chronic mastoiditis or mastoid fistula History of vestibular barotrauma Inability to retain mouthpiece Deafness in one ear	Middle ear barotrauma Recurrent or chronic sinus, external canal or middle ear infections Allergies of the nose and upper respiratory tract
Pulmonary	History of spontaneous pneumothorax Reactive airways disease (asthma) of any origin Chronic obstructive pulmonary disease Restrictive lung disease History or radiographic evidence of pulmonary blebs, bullae, or cysts	Childhood asthma without residual hyperactivity or air trapping Pneumothorax due to barotrauma, penetrating injury, or surgery without air trapping
Cardiovascular	Aortic or mitral stenosis History of myocardial infarction Angina or coronary artery disease Cardiac septal defects Complete or fixed second-degree heart block Wolf-Parkinson-White syndrome with paroxysmal atrial tachycardia or syncope Exercise induced tachyarrhythmias Fixed-rate pacemaker Hypertension with evidence of end-organ damage Drugs that inhibit normal exercise Peripheral vascular disease that limits exercise	———
Neurological	Seizure disorder Brain or spinal cord tumor Cerebrovascular accident or transient ischemic attack Demyelinating disease Spinal cord trauma with neurological deficit Head injury with sequelae Intracranial surgery Central nervous system aneurysm or vascular malformation Migraine headaches Episodic loss of consciousness	History of head trauma with loss of consciousness but no sequelae Chronic headaches History of neurological decompression sickness with residual
Hematological	Unexplained anemia Polycythemia or leukemia Sickle cell disease	Acute anemia
Endocrine	Insulin-dependent diabetes mellitus Diabetes mellitus treated by diet or oral agents with history of hypoglycemia	Non–life-threatening hormonal excess or deficiency Renal insufficiency Obesity
Reproductive	Pregnancy in any stage	———

(*Table 31-3* **Continues**)

Table 31-3 **Continued**

Psychiatric	Inappropriate motivation Claustrophobia or agoraphobia Active psychosis or psychosis while receiving psychotropic drugs Panic disorder Alcohol or drug abuse Suicidal ideation with or without severe depression Significant anxiety state Manic state	———
Ophthalmological	Radial keratotomy Uncorrected visual acuity inadequate to find diving buddy or boat if corrective lenses Corrected visual acuity inadequate to read instruments	———
Gastrointestinal	Uncorrected abdominal wall hernia Paraesophageal or hiatal hernia Chronic or recurrent obstruction Severe gastroesophageal reflux	Peptic ulcer disease Malabsorption Functional bowel disorders Inflammatory bowel disease
Musculoskeletal	Low back pain with neurological symptoms Disability that would hamper work in the water or with diving equipment Juxtaarticular aseptic bone necrosis	Acute sprain or strain Acute trauma

[*] Sources for the rationales for these recommendations: (1) Bove AA, Davis JC. *Diving Medicine.* 2nd ed. Philadelphia, Pa: WB Saunders; 1990. (2) Davis JC, Bove AA. *Medical Examination of Sports SCUBA Divers.* Flagstaff, Ariz: Best; 1986. (3) Linaweaver PG, Vorosmarti J. *Fitness to Dive: 34th UHMS Workshop Report*. Kensington, Md: The Undersea and Hyperbaric Medical Society; 1987.

the diver should be examined by a DMO.

For Type II symptoms that were completely relieved after the second oxygen-breathing period at 18 msw (60 fsw) during Treatment Table 6, the diver may resume diving after 14 days with a DMO's concurrence, if the symptoms involved only patchy peripheral paresthesias (numbness, tingling, or decreased sensation). However, if there were symptoms suggesting AGE, cardiorespiratory involvement, or neurological involvement other than patchy peripheral paresthesias, diving should not be allowed for 4 weeks—and then only after further examination. If the symptoms required treatment according to Treatment Tables 4 or 7, diving should not be allowed for 3 months. Up-to-date regulations should be consulted, as these proscriptions can change and may differ among the services.

SUBMARINE RESCUE AND ESCAPE

If a submarine is disabled and cannot surface, the crew may be rescued by a submersible chamber or small submarine, or may be required to leave the submarine through an "escape trunk" that allows passage from low submarine pressure to higher water pressure, from which they ascend through the water to the surface. While not specifically a diving activity, submarine rescue and escape expose sailors to pressure changes that cause illnesses associated with diving. Submarine rescue has not been needed by the US Navy since 1939 (Exhibits 31-9 and 31-10).

The US Navy has two methods for rescuing the crew of a disabled submarine. One method uses the Submarine Rescue Chamber (SRC), which is lowered from a surface vessel and mated to a hatch on the downed submarine. The SRC, which was developed in 1931, saved 33 men from the *USS Squalus* in 1939.

The second method, which is the primary rescue method, uses the Deep Submergence Rescue Vehicle (DSRV), a submersible that mates with the disabled submarine and transfers surviving crewmembers to the surface or another submarine (Figure 31-28). The DSRV can be pressurized internally if the submarine itself is under pressure, but as there are currently no surface chambers that allow transfer under pressure, rescued personnel must be decompressed to sea level and recompressed in a surface-based chamber for subsequent saturation decompression. The initial decom-

EXHIBIT 31-9

THE RATIONALE FOR SUBMARINE ESCAPE AND RESCUE

Given the paucity of recent US Navy submarine disasters ... one might conclude it is not prudent to devote too much of the Navy's resources ... to submarine rescue or escape. Submarine sailors, as well as airline passengers, wisely do not spend much time pondering the possibility that they may be embarking on a one way trip. Statistically speaking, submarine and commercial airline travel is very safe.

. . . .

It is frequently argued that since US submarines spend 95 percent of their time operating in waters [in which the ocean bottom is] deeper than the hull crush depth, discussions of escape or rescue are for the comfort of wives and mothers back home. Nevertheless, the chance of a mishap is much greater while the submarine is over the continental shelf because of the increased risks associated with sea trials, diving and surfacing, and transiting with open hatches and other hull penetrations in areas with greater sea traffic density, such as near major seaports.

Reproduced from: Molé DM. *Submarine Escape and Rescue: An Overview* [thesis]. Washington, DC: Department of the Navy, Submarine Development Group One; 1990: 1.

EXHIBIT 31-10

SUBMARINE ESCAPE AND LOCK-OUT TRAINING IN THE US NAVY

There has been no need for submarine rescue in the US Navy since the sinking of the USS *Squalus* in 1939. The USS *Thresher*, which sank in 2,520 msw (8,400 fsw) in 1963, and the USS *Scorpion*, which sank in 3,000 msw (10,000 fsw) in 1968, were too deep for rescue. The Navy conducted submarine escape training until the 1980s in two 100-ft-tall fresh-water towers in Groton, Connecticut, and Pearl Harbor, Hawaii, but these have been decommissioned due to concern over the incidence of arterial gas embolism (about 1 in every 22,000 exposures). Other navies continue submarine escape training in land-based towers. The US Navy presently has three 50-ft towers that are used primarily for submarine lock-out (leaving the submarine through an escape hatch) training for Special Operations personnel.

Source: Molé DM. *Submarine Escape and Rescue: An Overview* [thesis]. Washington, DC: Department of the Navy, Submarine Development Group One; 1990: 1.

pression to sea level may result in a high incidence of DCS. The DSRV is expensive to maintain, requires dedicated aircraft for transportation, takes at least 24 hours to arrive at a nearby port, and must be carried by ship or submarine to the disabled submarine.

To escape from a disabled submarine, several sailors enter an escape trunk, which is then isolated from the submarine by closing an inner hatch. The escapees don an appliance known as the Steinke hood, which is a life jacket with an attached hood that allows continuous breathing. The hood is plugged into an air-charging system, and the escape trunk is flooded with sea water and compressed to ambient sea pressure. The outer hatch of the trunk is opened, and the escapee leaves the trunk and is carried to the surface by the positive buoyancy of the hood. Air expanding in the hood vents to the sea during ascent. The risk of air embolism is small if the escapee breathes normally. This system has several drawbacks, however:

- if compression of the escape trunk is slow and the depth is great, the risk of DCS is significant;
- the escapees operate the trunk themselves, which may be difficult if they are frightened or compromised by contaminated air, carbon dioxide, hypoxia, pulmonary oxygen toxicity, or nitrogen narcosis;
- if an escapee is caught in the trunk, it becomes unusable by others; and
- unless rescue is immediate when the escapee reaches the surface, the chance of survival is small because no thermal protection is provided.

Medical officers who supervise training exercises or actual escapes must be prepared to manage casualties from cerebral air embolism, DCS, trauma, burns, and exposure.

The Royal Navy has a different escape system: a one-man lock that can be flooded and pressurized very quickly. This system offers some advantages. The escapee is completely enclosed by a hood that is attached to a survival suit. On entering the lock, the escapee plugs into an air supply, and the lock is automatically flooded and pressurized to eliminate the possibility of operator error. During compression, the pressure doubles every 4 seconds, which

Fig. 31-28. One of the US Navy's Deep Submergence Rescue Vehicles (DSRVs; center, in 1985) is locked-on to (ie, mated with) the after escape trunk of a surfaced submarine. Photograph: US Navy.

Fig. 31-29. An escapee, enclosed in a survival suit and hood, ascends from a submerged submarine using the Royal Navy method. The US Navy is in the process of adopting this escape method. Photograph: Royal Navy, London, England.

reduces nitrogen uptake and the risk of DCS. (The middle ear space seems to equilibrate passively during this rapid compression, or rupture of the tympanic membrane is not noticeable.) The outer hatch opens when the lock reaches ambient pressure, and the escapee is buoyed rapidly to the surface without risk of entanglement in the trunk (Figure 31-29). The survival suit is designed for at least 24 hours in cold seas and bad weather. This system has been tested at sea from a depth of 180 msw (600 fsw). The US Navy plans to retrofit its submarines with the British escape system.

SUMMARY

Military diving operations have special medical requirements because of the physiological stresses imposed by barometric pressure, breathing gas composition, and immersion. Personnel at risk include some 4,000 to 5,000 divers in the US military (Navy, Marine Corps, Army, Air Force, Coast Guard) and 5,000 to 10,000 military divers from other nations. For US forces, the *US Navy Diving Manual*[1] provides guidance on the conduct of diving operations and associated medical assistance. In addition to diving, UMOs in the US Navy support the escape and rescue of crewmembers who may be trapped in disabled submarines.

Diving history provides a useful context in which the interactions of mission and environment can be appreciated from a medical perspective, and examples drawn from combat swimming and diving operations during World War II serve to illustrate the problems of breath-hold and oxygen diving. UBAs, which provide a reliable source of breathing gas and extends the dive time, introduce additional problems that can occur with increasing depth, including the need for thermal protection and alterations of consciousness from interactions of oxygen, carbon dioxide, and nitrogen.

Ascent from depth must become progressively slower as the surface is approached to avoid DCS from bubbles that form in the diver's body. Decompression schedules and diver-worn computers provide guidance concerning how to ascend with acceptable DCS risk. After 12 to 24 hours, divers become saturated with inert gas and may remain at depth indefinitely without accruing additional decompression time. Saturation dives require special pressure chambers in which the divers live while not actively working underwater.

Failure to follow the decompression prescriptions of dive schedules or computers (and sometimes even if these prescriptions are followed) can lead to DCS or AGE. These conditions usually can be successfully treated by recompression while the patient breathes 100% oxygen, which causes bubbles to resolve. The pulmonary, cardiovascular, and neurological systems require particularly close evaluation during medical examinations to determine fitness to dive for initial training or for the return to diving after DCS or AGE.

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