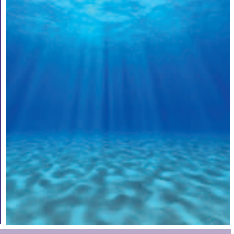
PART 10

Marine Medicine



CHAPTER 69

Drowning and Submersion Injuries

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To have faith is to trust yourself to the water. When you swim you don't grab hold of the water, because if you do you will sink and drown. Instead you relax, and float. A. Watts[247]

A lack of oxygen does not simply involve stoppage of the engine, but total ruin of what we took to be the machinery. J.S. Haldane[91]

Humankind is surrounded by water; it covers 75% of Earth's surface, and is integral to our survival and development. Water can be dangerous, responsible for hundreds of thousands of drowning deaths per year. The earliest recorded drowning resuscitation is from Syria in 1237 BC, when two soldiers rescued the king of Aleppo from the Orontes River. The king is shown being held upside down as part of an inversion technique used for thousands of years in attempts to revive drowning patients. During the late 16th century, several societies were founded in Europe in response to increasing numbers of deaths on commercial waterways, where boating and shipping were the primary mechanisms of transportation. Drowning became a substantial public health issue, and in response, several national societies were formed. With proliferation of swimming pools and rapid expansion of recreational water activities, drowning became not only an occupational hazard but also a significant recreational hazard. This chapter reviews classification, pathophysiology, clinical presentation, treatment, and prevention of drowning, and emphasizes the importance of safety and injury prevention.

CLASSIFICATION AND TYPES OF SUBMERSION INJURIES AND DROWNING SCENARIOS

Drowning is an international public health problem that has been complicated by lack of a uniform definition, proliferation of confusing subdefinitions, inadequate epidemiologic studies, and conflicting clinical management paradigms. In response to dialogue at the 2002 World Conference on Drowning, a consensus panel and the World Health Organization adopted the following definition: "Drowning is the process of experiencing respiratory impairment from submersion/immersion in liquid."[238] According to this new classification system, drowning outcomes should be classified as drowning death, drowning with morbidity, and drowning without morbidity. Drowning is now considered a process and not an outcome. Other water-related conditions that do not primarily involve the airway and respiratory system are submersion injuries rather than drowning. For example, this definition of drowning specifically excludes water rescues during which submersion does not involve the respiratory system; if the rescued person maintains his or her airway above water throughout the event, it would be considered a submersion injury rather than a drowning.[237]

The consensus panel of 2002 also concluded that the terms *wet drowning, dry drowning, active drowning, passive drowning, silent drowning, near-drowning,* and *secondary drowning* should no longer be used in drowning terminology.[238] However, because these terms are still widely in use by medical professionals and laypersons, it is important to be familiar with them.

One of the most common previous classification systems divided drowning into two possible outcomes: drowning and near-drowning. This system was problematic because the term *near-drowning* had 20 different published and often conflicting definitions. The preferred nomenclature is *nonfatal* drowning. Data on nonfatal incidents were often excluded from epidemiologic and research studies of drowning because the victim had survived. This was a detriment because information about survival of drowning can have a major impact on public health initiatives and optimization of treatment protocols.[163,238] Excluding nonfatal drowning incidents from statistics on drowning meant that such incidents were often underreported; excluding information about survivors of a life-threatening event from epidemiologic data is rare in medicine. The term near-drowning should no longer be used in drowning terminology; it should be replaced by the terms preferred since 2002 for the subsets of drowning: *drowning death, drowning with morbidity,* and *drowning without morbidity.*[237,253]

Another common previous classification system differentiated drowning incidents by presumed physiologic mechanism: wet or dry. Wet drowning, a term that described aspiration of water during the drowning process, was thought to occur in 80% to 90% of drowning deaths, with the remaining individuals experiencing dry drowning, in which no water is found in the lungs, presumably due to laryngospasm. Reanalysis of the original autopsy studies leading to these distinctions has brought this classification into question. The actual existence of dry drowning has not been proven; additionally, if laryngospasm does occur, it likely relaxes with progressing hypoxemia. Existence of such a reflex holds no prognostic or treatment significance. The terms wet drowning and dry drowning should not be used.

Other classifications that describe the mechanism of drowning include *active drowning, passive drowning,* and *shallow water syncope* or *shallow water blackout. Active drowning* and *passive drowning* are historical terms that most likely represented witnessed and unwitnessed drowning incidents, respectively.[238] The terms have no usefulness for epidemiologic or clinical understanding of drowning. *Shallow water syncope* is a syndrome that occurs primarily among competitive swimmers, free-divers, and spearfishers who attempt to stay underwater for long periods without the need to breathe. Individuals hyperventilate before submersion, resulting in hypocapnic respiratory alkalosis. By artificially reducing the proportion of carbon dioxide in arterial blood, the time until a person needs to breathe is prolonged; at the same time, the body's consumption of oxygen may lead to such a low level of blood oxygen that the person becomes unconscious. Because the compulsion to breathe from hypoxemia is less potent than the drive from acidosis or hypercapnia, a person may become unconscious from hypoxemia before the carbon dioxide level rises adequately to stimulate breathing, which leads to drowning.

INCIDENCE AND EPIDEMIOLOGY

The World Health Organization Global Burden of Disease Update estimated that 372,000 people drowned worldwide in 2012; this represented 7% of all injury-related deaths.[253] Statistical data likely underestimate the true incidence of drowning. This is largely due to underreporting, particularly in middle- and low-income countries, where many drowning victims never make it to a hospital. It is estimated that for every reported drowning death, another four go unreported.[101] Codes from the *International Classification of Diseases 10th edition* (ICD-10) have recently been altered to improve drowning and subtype categorizations, but many

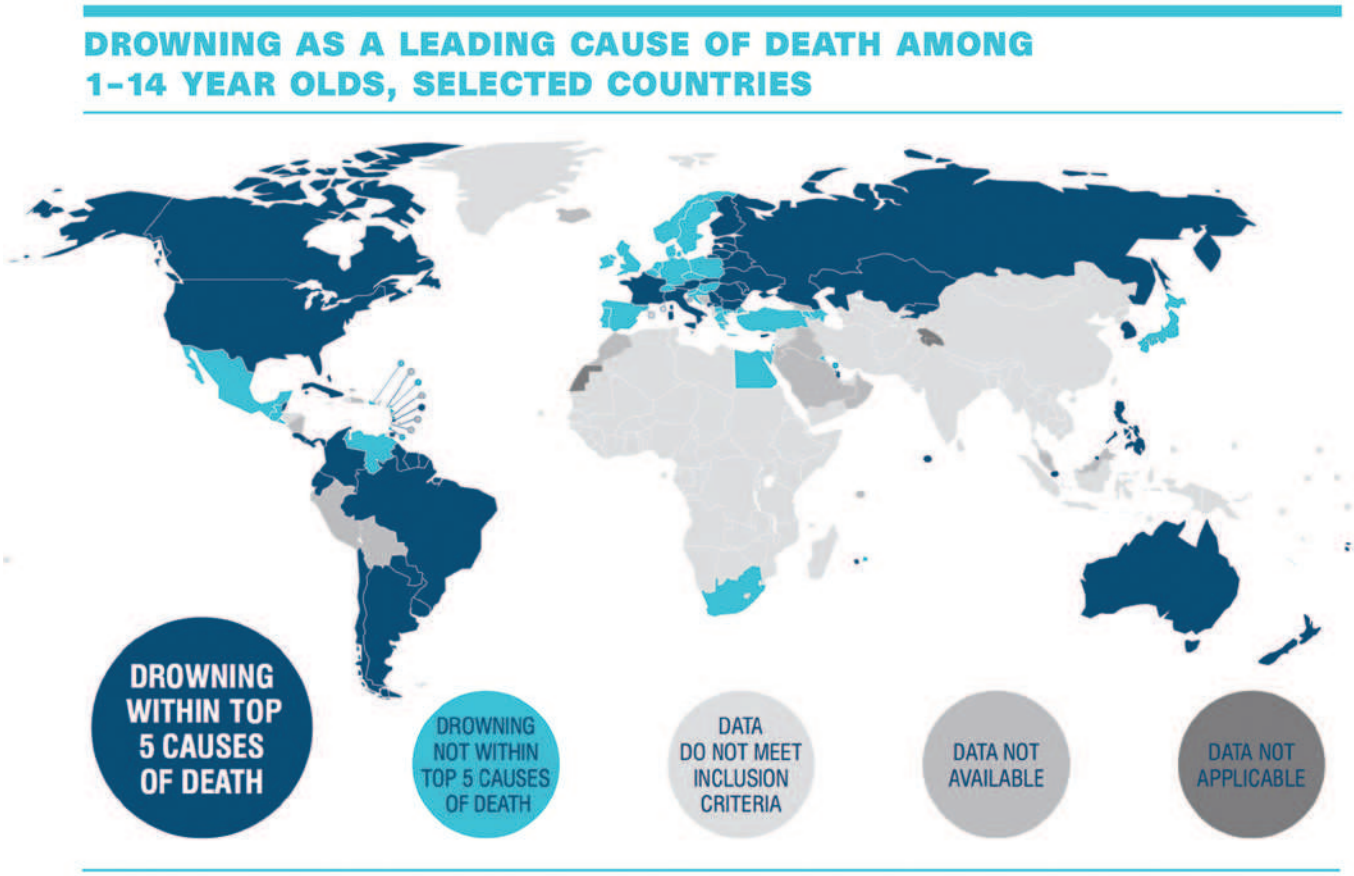

FIGURE 69-1 According to the World Health Organization, drowning is a leading cause of death among those 1 to 14 years of age worldwide. *(From WHO. Global report on drowning: preventing a leading killer. 2014.* http://www.who.int/violence_injury_prevention/global_report_drowning/en/*.)*

countries still fail to report sufficiently specific codes to the World Health Organization with regard to data on drowning deaths.[136] A further complication is that drowning deaths caused by floods and natural disasters are not reflected in these numbers; such events include the great tsunami of December 2004, which resulted in more than 100,000 deaths by drowning, and Hurricane Katrina in New Orleans in 2005. Drowning deaths resulting from assaults, suicides, and boating accidents are not typically classified as drowning, because they are usually classified based on the primary cause of death, and secondary diagnoses (e.g., drowning) are not captured.

Worldwide, drowning occurs overwhelmingly (>91%) in low- and middle-income countries. Drowning is the third leading cause of accidental injury death wordwide. India and China both have particularly high drowning mortality rates,[139] together contributing 43% of all drowning deaths worldwide and 41% of the total disability-adjusted life years attributed to drowning worldwide. In China in 2001, drowning was the leading cause of injury death among children between 1 and 14 years of age.[252] The global burden of drowning in 2002 is shown in Figure 69-1; territories are sized in proportion to the absolute number of people who died from drowning that year.

In the United States, the National Center for Injury and Prevention Control reported 4308 fatal unintentional drowning deaths in 2012, or 1.37 deaths per 100,000 people.[41] Boating-related incidents accounted for an additional 651 deaths, with 71% of those attributed to drowning.[41] Drowning ranks as the 10th leading cause of injury death overall in the United States, but it overwhelmingly affects younger age groups: Drowning is the leading cause of death by unintentional injury among children between the ages of 1 and 4 years, and ranks second among children between the ages of 5 and 9 years; it is the sixth leading cause of injury death for those between the ages of 15 and 24 years.[40] California, Florida, and Texas reported the highest numbers of drowning deaths in 2006.[38] It is estimated that for every pediatric drowning death, an additional four children are hospitalized for nonfatal drowning; many of these children require prolonged intensive care and may suffer permanent neurologic disability.[40,136]

The economic costs of drowning are among the highest of any injury group, largely because the most severe complications and most deaths occur in individuals between the ages of 0 and 15 years. At this young age, there is a large impact on future economic productivity. An estimated 1 million disability-adjusted life years were lost as a result of premature death or disability from drowning worldwide in 2004.[196,252] A single nonfatal drowning survivor with severe neurologic impairment can accrue more than $4.5 million in medical treatment costs over a lifetime.[40]

RISK FACTORS

Drowning tends to involve specific populations, age groups, and locations (Box 69-1). Understanding risk factors is important to implementing prevention and public health programs to decrease the incidence of drowning.

AGE

Young people have the highest likelihood of drowning. A bimodal age distribution characterizes childhood drowning, with

BOX 69-1 Factors Common to Drowning Incidents

Age: toddlers, teenage boys
Location: home swimming pools, bathtubs, buckets
Gender: males more often than females
Race: black children are at higher risk than other groups
Drugs: particularly alcohol
Trauma: secondary to diving, falls

children less than 4 years old and adolescent males accounting for the peaks. There is an additional smaller peak later in life among persons over 65 years of age.[192] In 2005, of all children between the ages of 1 and 4 years who died in the United States, 31% died from drowning.[40] Children are at high risk of drowning because many cannot swim or swim well, they have a large head-to-body ratio (increasing the risk of head submersion), there is a lack of barriers around pools and bathtubs, and there are lapses in supervision. Toddlers are especially at risk because they are able to move about independently but are unable to recognize water hazards or to effect self-rescue. Despite the American Academy of Pediatrics (AAP) recommendation never to leave a child unsupervised in the bathtub, one study reported that nearly 33% of parents leave children unattended in this setting.[214] Children bathing with a sibling who was less than 2 years old who were unsupervised for a brief time accounted for 22% to 58% of bathtub drowning deaths.[34] Drowning among adolescents and teenagers is often seen with risk-taking behaviors, lack of supervision, and drug and alcohol intake. The scene of drowning in this age group is most commonly a natural body of water, such as a lake, pond, or river. Incidents often occur far from medical assistance and may take place where rescue is challenging; however, these events are often witnessed.[7,32,45,192,193] Among people who are more than 65 years old, deaths are evenly divided between open-water drowning and bathtub-related drowning, often from falls or exacerbations of concomitant illnesses (e.g., cardiac arrhythmias), which may not be recognized at autopsy.[192]

GENDER

In 2012, males were three times more likely than females in all age groups to die from unintentional drowning in the United States.[38] The incidence of drowning in females peaks at 1 year of age and declines throughout the rest of life. This trend is thought to be due to increased risk-taking behavior and greater alcohol consumption among males as compared with females. Worldwide, males have a higher mortality rate from drowning as compared with females in all age groups and regions.[184]

RACE

In the United States between 1999 and 2010, the rate of fatal drowning for blacks was 1.4 times higher than that of whites.[41] The rate was twice as high for Alaska natives and Native Americans as for whites.[40] In Alaska, drowning rates are twice as high among Native American children as compared with white and black children.[6] In the 10- to 14-year-old age group, the fatal drowning rate for blacks is 3.7 times that of white children of the same age, and is 2.6 times higher for Alaska native and Native American children.[38,79] This pattern is reflected in the military, where black soldiers drown 62% more often than white soldiers.[15] The reasons behind racial differences in drowning rates are unclear, but suspected factors are decreased routine access to swimming pools, less emphasis on swimming lessons, and less participation in recreational water-related activities.[161] If, overall, minorities participate in fewer water-related activities, their drowning rates per exposure may be even higher than reported.[27]

LOCATION

Familiar or Unfamiliar Places

Any body of water, no matter how shallow or small, can be the site of drowning. Oceans, seas, and rivers account for fewer drownings than do backyard pools, recreational lakes, bathtubs, and buckets of water. In the United States, drownings involving young children occur primarily in fresh water; children less than 1 year of age most often drown in bathtubs, buckets, or toilets, whereas those between 1 and 4 years of age most often drown in residential pools.[5,14,23,32,45,161,192] Despite these statistics, only a small proportion of home pools are properly protected, and most are easily accessible by ambulatory toddlers and curious children.[204,218] One study found that most young children who drowned in residential pools had been out of sight of adults for less than 5 minutes, and were in the care of one or both parents at the time.[188] Among individuals between the age of 5 and 64 years, drowning typically occurs in open-water recreational settings such as lakes, rivers, and oceans.[32,185] Persons over age 65 have the highest rates of bathtub drowning per age group.[192]

Visitors to domestic and international locations are at a higher risk for drowning than natives in the same region. After motor vehicle accidents and homicide, drowning is the leading cause of injury death among U.S. citizens traveling abroad.[231] In island locations, it is the leading cause of injury death; it accounts for 63% of traveler deaths by injury as compared with 3.5% for native citizens of the respective countries.[88,231] Lack of familiarity with the environment,[133] lack of understanding of local hydrology, overestimation of abilities, and use of alcohol while vacationing are likely contributors to drowning rates among travelers.

Submerged Vehicle Incidents

Some studies suggest that approximately 10% of all drownings occur via a submerged vehicle. During natural disasters, drowning in a submerged vehicle occurs in as many as 10% of all motor vehicle accident deaths.[216,256] A more recent study[219] demonstrated that, of 83 drowning deaths in vehicles, more than 92% of victims had insignificant traumatic injuries, suggesting that the primary source of death in submerged vehicles is drowning rather than trauma.

Many suggestions in the popular media about how to manage entrapment in a submerged or submerging vehicle are incorrect and can paradoxically lead to increased risk of death. These poor suggestions include allowing the passenger compartment to fill with water so it will be easier to open doors, waiting until the vehicle hits the bottom to maintain orientation, relying on kicking out the windshield or opening the door to exit, and relying on breathing trapped air in the passenger compartment.

One series of Canadian studies looked at ways to deal with a submerging vehicle situation and possible ways to reduce fatality rates. These data demonstrated that a vehicle floats for 30 seconds to 2 minutes prior to sinking. In this phase, windows can be easily opened and used for exit, assuming that the risk outside the vehicle (e.g., swift water or a nonswimmer exiting into deep water) is not greater than the risk inside the vehicle. In one trial, three adults were able to release a child mannequin from a rear child seat and exit the vehicle within 51 seconds. During the sinking phase (after 2 minutes), occupants can breathe as water rises inside the vehicle but chances for escape and survival decrease considerably because rising water pressure makes it more difficult to exit through doors and windows. In the third, submerged phase, the vehicle is full of water and no air pockets exist, so the chance of survival is negligible. The following escape procedure is recommended: Immediately unfasten all seat belts and then open all the windows. If children are present, they should be released from restraints and held by an adult. Once all passengers are free of restraints, children should be assisted out of the open windows first, followed by adults (Box 69-2).[97]

Scuba Diving Accidents

The Divers Alert Network reported 138 deaths internationally in 2006 from scuba diving accidents:[58] Fifty-one of these were in the United States, with Florida having the highest incidence, followed by California. The cause of death was reported in 58 cases (77% of the total); of those, 86% were attributed to

BOX 69-2 Submerged Vehicle Escape Procedure

Seat belts: unfastened
Windows: open
Children: if present, released from restraints and brought close to an adult who can assist in their escape
Out: children should be pushed out of the window first, and followed immediately

From McDonald GK, Giesbrecht GG: Vehicle submersion: a review of the problem, associated risks, and survival information. *Aviat Space Environ Med* 84:498-510, 2013.

drowning, by far the most common cause of death in this series. Several patients had known preexisting medical conditions, including cardiovascular disease, which may have contributed to drowning.

Water Birthing Incidents

Water birth (i.e., labor and delivery while immersed in water) may be a risk factor for drowning. Documented adverse neonatal outcomes from underwater birth include unexplained death, drowning, asphyxiation, water intoxication, hyponatremia with seizures, water aspiration leading to respiratory distress and failure, pulmonary edema, hypoxic-ischemic encephalopathy, and pneumonia and other infections, including *Pseudomonas* and *Legionella* infection.[70,76,169,180] Other observational series have shown similar Apgar scores, rates of neonatal resuscitation, and complications for both regular births and water births.[78,169] There has been a vigorous debate in the pediatrics and obstetrics community regarding the safety of this birth modality, but what is undisputed is that there are few reliable data demonstrating the safety of water birthing.[207] In 2014, the AAP and American College of Obstetricians and Gynecologists (ACOG) issued a joint position paper stating that "the practice of immersion in the second stage of labor (underwater delivery) should be considered an experimental procedure that only should be performed within the context of an appropriately designed clinical trial with informed consent." However, the authors[102] of this position paper have been criticized for nearly exclusive reliance on low-level evidence in case studies, while failing to cite or review higher-level evidence in prospective studies; using an outdated literature review; and misrepresentation of studies that were reviewed (evidencebasedbirth.com/waterbirth/). It should be noted that, in contrast to the AAP-ACOG position paper, the American College of Nurse Midwives,[6b] the American Association of Birth Centers,[6a] and the Royal College of Midwives[203a] all released counterstatements declaring evidence-based support for the safety of water births, and specifically the low drowning risk for neonates during these deliveries.

ABILITY TO SWIM

Although popular, swimming programs for young children do not fully protect against drowning; children should always be supervised while swimming. There is some evidence that children ages 1 to 4 years with some formal swim instruction were less likely than matched controls (3% versus 26%, respectively) to die by drowning.[31] The latest recommendation from the AAP is that children as young as 1 year old can be enrolled in formal swimming courses. This does not replace the need for direct supervision and use of pool barriers and alarms. Swimming lessons are recommended for nearly all children after the age of 4 years. Prevention focuses on supervision, restricting access to home pools, and reducing use of drugs and alcohol among teenagers around water.[4-6]

Highly experienced swimmers are not immune from drowning; competitive swimmers and breath-hold divers sometimes engage in intentional hyperventilation and die as a result of shallow water syncope. Breath-hold diving is defined as in-water activity without self-contained or surface-supplied breathing gas. Breath-hold activities include snorkeling, spearfishing, and free-diving, from which there were 34 fatal drowning cases reported worldwide by the Divers Alert Network in 2006.[38] More than half occurred in the United States, primarily in Florida, Hawaii, California, and Texas; this may be the result of higher rates of reporting in the United States than other countries and suggests that safety and prevention strategies should be a focus in specific geographic regions. Twelve cases were associated with blackout due to hypoxemic loss of consciousness, likely as a result of intentional hyperventilation to prolong breath-holding time.

According to the International Swimming Federation, open-water swimming is defined as any competition that takes place in a lake, river, or ocean. Several studies have demonstrated a risk for hypothermia during open-water swimming competitions, but the amount of risk varied depending on the modality of temperature measurement used and remains unclear.[28,36]

ALCOHOL AND DRUGS CONTRIBUTING TO DROWNING INCIDENTS

Alcohol has been implicated as a contributing factor in 25% to 50% of recreational water-related deaths and in 20% of boating-related fatalities.[33,60,131,133,159] Studies have demonstrated blood alcohol levels of 100 mg/dL or more as a factor in 25% of boating-related fatalities,[33] in 25% of teen drowning deaths,[193] and in 33% of drowning deaths of young adults between the ages of 20 and 34 years. In Australia, 21% of people who drowned during a 5-year period had measurable blood alcohol levels.[69] Despite federal laws that prohibit alcohol use during recreation on open water, a survey of adults in the United States showed that 31% of 597 respondents reported operating a motorboat while under the influence of alcohol; these operators were overwhelmingly males between 25 and 34 years of age.[134] Impaired judgment caused by intoxicants can lead to accidents as well as loss of body heat, decreased laryngeal reflexes, higher risk of aspiration, decreased supervision of children, and decreased use of safety devices, such as personal flotation devices (PFDs).[192,260] Illicit recreational drug use with and without alcohol has been reported as a factor in water-related deaths.[33] All types of drugs that affect judgment can result in watercraft overcrowding, speeding, and inattentive or reckless handling, and in failure to wear PFDs.

PREEXISTING DISEASE

Risk of submersion injury or death in water increases significantly when medical conditions compromise the chance for self-rescue and survival. Situations are particularly dangerous for unpredictable medical conditions, such as cardiac and neurologic conditions. Individuals with relevant preexisting conditions should never be alone in the water and must optimize treatment for the underlying condition before engaging in water-related activities.

Seizure disorders increase the risk of drowning among adults and children in both natural bodies of water and in homes.[16,18,29] Drowning is the most common cause of death by unintentional injury among patients with seizure disorders.[190] One study found the risk of drowning among people with epilepsy to be 15 to 19 times greater than that of the general population.[16] It is when a patient is alone that the risk of death increases drastically. Other neurologic conditions (e.g., cerebrovascular accident, arteriovenous malformation) that manifest as seizure have also been associated with drowning.[81]

Cardiac disorders associated with dysrhythmias may manifest while a person is submerged in water; however, the exact mechanism for rhythm disturbance as a result of submersion remains unclear.[2,25] Prolonged QT syndrome, a conduction disorder associated with sudden death, has been linked by forensic molecular screening to some drowning deaths, indicating a theoretically possible gene-specific dysrhythmogenic presentation of prolonged QT syndrome triggered by swimming.[3,137] Adverse events, such as unstable tachycardia or loss of consciousness from myocardial infarction, may lead to drowning deaths.

CHILD ABUSE, HOMICIDE, AND SUICIDE

Abuse or neglect should be investigated in any suspicious fatal or nonfatal pediatric drowning; thorough social service and legal investigation is needed. One series found abuse or neglect to account for 19% of bathtub drowning deaths among patients less than 5 years old.[193] In the adult population, homicide by drowning is relatively uncommon. The postmortem determination of death by drowning (as opposed to body disposal in water after another mechanism of homicide) is very difficult to make. Drowning by suicide is relatively uncommon.[175,240,251] An Australian study of 123 suicides by drowning found women to more commonly choose a bathtub or the ocean for drowning, whereas men selected rivers, ditches, and lakes.[35]

BOATING-RELATED DROWNING

Recreational and commercial boating accidents are among the leading causes of unintentional drowning. Deaths result from

capsized vessels or falls overboard. The risk of drowning is determined by the water depth and temperature, distance from shore, and currents, including hydraulics at the bases of dams or spillways. Alcohol use, lack of personal protective equipment, and unsafe boating practices are involved in many boat-related drowning incidents.[53,131] In 2013, the U.S. Coast Guard reported 2620 injuries and 560 deaths as a result of recreational boating accidents;[236] Of these deaths, 431 were the result of drowning; 362 (84%) of these victims were not wearing PFDs or lifejackets. Studies suggest there is significant underuse of PFDs among recreational boaters.[115,191] In the United States in 2013, alcohol was the leading contributing factor in 16% of fatal boating accidents, followed by operator inexperience and careless or reckless operation. Most fatal accidents occurred with open motorboats, followed by canoes and kayaks. States with the highest death rates (i.e., > 10 per 100,000 registered boats) were Alaska, Hawaii, Montana, Idaho, Utah, Texas, Louisiana, and Vermont.[236]

The international commercial fishing industry also contributes to boating-related drowning deaths. The International Labor Organization estimated that 45 million people worldwide were employed in aquaculture industries in 2007[103]; most are located in Asia and Africa. Death rates among commercially employed fisherman are among the highest of any occupation.[132,199] In the United States, death rates of fishermen are 16 times higher than those of police and firefighters and 8 times higher than those of persons who drive motor vehicles for a living.[235]

Another population affected by water transport and drowning is that of refugees seeking asylum, who are often in rough weather and poorly equipped, overcrowded boats without lifejackets. A 2004 study reported that 4000 asylum seekers are estimated to drown annually at sea.[61,189] The 2014 crisis of refugees fleeing war-torn Middle Eastern countries has resulted in numerous drowning deaths. According to the United Nations High Council on Refugees (UNHCR), there were 410 documented drowning deaths in the first 6 weeks of 2015 among 80,000 people crossing the eastern Mediterranean, which is a 35-fold increase from 2015.[233a]

PATHOPHYSIOLOGY

The pathophysiology of submersion has been extensively studied in animal models, yet there remain ambiguities with regard to the exact sequence of events and mechanisms of drowning in humans. The effect of submersion on mammals was initially reported in scientific literature during the late 1800s, and most early research involved animal models. In a classic monograph from 1965, Greene noted that "inundation of the upper airway, the bronchial tree and segments of the alveolar spaces blocks gas exchange in the lung and produces asphyxia. Thus drowning involves the rapid development of hypoxemia, hypercapnia, and acidosis with the associated sequence of hypertension, bradycardia, apnea, and terminal gasping."[86] Although these observations remain largely accurate for humans, they were based on mammalian experiments designed to simulate drowning.[100,135,170] Early animal research examined the hemodynamic and electrolyte effects of drownings that occurred in water types of different osmolality (i.e., saltwater versus freshwater drowning). Although some models indicated a pathophysiologic difference between the two, this has proved to be of limited applicability in humans. Early canine experiments showing hypervolemia in freshwater aspiration and hypovolemia and hypernatremia in saltwater aspiration[157] have not been reproduced, and human research has failed to replicate the course observed in animals.[205] Hypervolemia has not been observed in human freshwater aspiration, and most humans have total fluid aspiration of less than 4 mL/kg.[141,153] More than twice that amount is required to effect the changes in blood volume seen experimentally. Similarly, an aspirated amount of 22 mL/kg is required before systemic electrolyte changes result[152]; therefore, the distinction between saltwater and freshwater drowning is of limited utility for humans. Most authorities recommend discontinuation of this distinction so the focus can be on the common pathway of hypoxemia, acidosis, pulmonary injury, and multiorgan system failure that remain the hallmarks of drowning pathophysiology.[8,81,127,149,178,181,182,238]

THE HUMAN BODY AND WATER

The degree to which a body floats (i.e., its buoyancy) depends on the amount of air in the lungs, body fat, and type and distribution of clothing. When the lungs are maximally filled with air (close to total lung capacity), the body has approximately 2.5 kg (5.5 lb) of flotation. It has greater buoyancy during inspiration as the lungs fill with air, and lesser buoyancy during expiration. Clothing can affect both heat conservation and buoyancy, and can be integral to survival in the wilderness water environment. PFDs are specifically designed to increase buoyancy (Figure 69-2).[168] The percentage of body fat is a factor in the ability to conserve body heat during prolonged immersion.

THE INITIAL EVENT

Drowning begins when the patient's airway is below the surface of a liquid medium, usually water, and the patient must breathe but cannot surface to do so. There is typically an initial period of struggle with attempted breath-holding. This struggle may not occur during trauma-associated submersion, when the patient may have been unconscious before or upon entry into water.[127,178] After gasping occurs, the initial struggle may be followed by laryngospasm to protect the lower airways from liquid in the upper airways, although the prevalence and relevance of this distinction to the ultimate pathophysiology is controversial.[127,177,205] It is now believed that all drowning patients likely aspirate at least a small amount (< 30 mL) of liquid. Particularly during cold-water submersion, the initial event is accompanied by a drive to hyperventilate caused by stimulation of thermal skin receptors, in addition to increasing hypoxemia. During this initial time, patients often swallow large amounts of water to avoid aspiration.[153] Eventually, the outcomes of breath-holding are hypoventilation, hypercapnia, respiratory acidosis, and hypoxemia. Loss of consciousness ensues and cardiopulmonary arrest follows (Figure 69-3).

The duration of tissue hypoxemia, timing of rescue, and institution of effective cardiopulmonary resuscitation (CPR) and field resuscitation are significant factors that affect whether the initial submersion injury is survivable. The duration of submersion, water temperature, and tissue susceptibility to hypoxemia determine the effects of submersion on different organ systems and likelihood of survival.

PULMONARY SYSTEM

Aspiration of water of any type and volume immediately affects alveolar ventilation, gas exchange, and mechanical characteristics of the lung.[56,74,178] Hypoxemia is the hallmark of pulmonary pathophysiology during drowning. Breath-holding and apnea lead to a rise in partial pressure of carbon dioxide and a fall in partial pressure of oxygen in both the alveolar and arterial blood. In addition, inhalation of any liquid causes surfactant disruption and alveolar collapse, resulting in areas of ventilation/perfusion ($\dot{V}/\dot{Q}$) mismatch, shunting, and further worsening of hypoxemia. Pulmonary sequelae may range from asymptomatic to acute respiratory distress syndrome (ARDS), depending on the amount of water aspirated and duration of submersion. Usually, symptoms of pulmonary involvement are present immediately; however, there are reports of delayed ARDS after an initially normal chest radiograph.[56]

Volumes of aspirated liquid of as little as 1 to 3 mL/kg are sufficient to cause disruptions in alveolar gas exchange and thus hypoxemia. Diminished pulmonary gas exchange occurs in part due to surfactant disruption as a result of aspiration. Surfactant is produced by type 2 pneumocytes, pulmonary epithelial cells responsible for maintaining alveolar surface tension, increasing pulmonary compliance, and preventing atelectasis. Disruption of surfactant leads to malfunctioning of the alveolar epithelial lining and increased $\dot{V}/\dot{Q}$ mismatch. Interstitial and microvascular damage ensues in response to alveolar-capillary basement membrane disruption, releasing an inflammatory cascade that results in pulmonary edema caused by extravasation of fluid into the alveolar space, bronchospasm, worsening $\dot{V}/\dot{Q}$ mismatch,

TYPE I PFD	TYPE II PFD
	
Off-Shore Life Jacket Best for open, rough, or remote water, where rescue may be slow coming **Minimum buoyancy** 22 lb **Advantages** • Best flotation • Turns unconscious victim face up • Highly visible **Disadvantage** • Bulky, uncomfortable	**Near-Shore Buoyant Vest** Best for calm, inland water or where there is good chance for fast rescue **Minimum buoyancy** 15.5 lb **Advantages** • Less bulky • Turns some unconscious victims face up • More comfortable than Type I **Disadvantage** • Not for long hours in rough water
TYPE III PFD	**TYPE IV PFD**
	
Flotation Aid Best for calm, inland water or where there is good chance for fast rescue **Minimum buoyancy** 15.5 lb **Advantages** • Most comfortable type • Designed for activity marked on device • Available in many styles **Disadvantages** • May have to tilt head back to avoid face-down position • Wearer's face may be covered by waves • Not for extended survival in rough water	**Throwable Device (rings, cushions)** Best for calm, inland water with heavy boat traffic, where help is always nearby **Minimum buoyancy** 16 lb (ring)–18 lb (cushion) **Advantages** • Can be thrown to victim • Good back-up to wearable PFD **Disadvantages** • Not for unconscious victims • Not for non-swimmers or children • Not for many hours in rough water

FIGURE 69-2 Current classification of the types of personal flotation devices.[104] *(Courtesy Justin Sempsrott, MD; adapted from Personal Flotation Device Manufacturers Association:* pfdma.org/choosing/types.aspx*.)*

and hypoxemia.[127] Areas of hypoxic pulmonary vasoconstriction further worsen shunting and increase pulmonary hypertension, leading to increased pressure across the alveolar-capillary basement membrane and further extravasation of fluid into the alveoli. In severe cases, patients present with ARDS, profound hypoxemia, noncardiogenic pulmonary edema, and a significant alveolar-arterial oxygen gradient. In profoundly ill patients, intubation and artificial ventilation may be required, increasing the risk of subsequent ventilator-associated lung injury and ventilator-associated pneumonia. With adequate resuscitation and treatment of patients without significant neurologic compromise, pulmonary dysfunction may initially be severe, but ultimate recovery of baseline lung function after drowning is the rule rather than the exception.[87,153]

Contamination by debris from petroleum products, sewage, sand, and organic matter is more common in saltwater and brackish water. Such debris accentuates the possibility of further inflammation and lung injury, which increases mortality rates. Inhalation of mud, sand, and other particulates may require fiberoptic bronchoalveolar lavage to cleanse the airways. Aspiration of microbes and related potential infections are discussed later. Figures 69-4 to 69-6 show examples of foreign body and contaminant aspiration after drowning.

Swimming-Induced Pulmonary Edema

Swimming-induced pulmonary edema has been described in several case reports as affecting scuba divers, Navy SEALS, and individuals (e.g., triathletes) who engage in strenuous surface swimming. The pathophysiology of this disorder is unclear, but proposed mechanisms include central blood pooling related to immersion and strenuous swimming resulting in elevated pulmonary artery pressure and diastolic dysfunction. Experimental equine data demonstrate increases in right ventricular and pulmonary arterial pressures with varying degrees of submersion in water, presumably resulting in capillary fracture. The case definition of swimming-induced pulmonary edema consists of acute hypoxemia during or immediately after a swimming event, a demonstrable chest radiograph abnormality with resolution within 48 hours in the absence of evidence of underlying pulmonary infection, and aspiration of water or attempted breathing against a closed glottis. The significance of this clinical entity is unclear, and it appears to be relatively uncommon.[147,209]

CENTRAL NERVOUS SYSTEM

The central nervous system (CNS) is highly sensitive to even brief periods of hypoxemia and is the organ system most susceptible to the negative effects of drowning. The major determinant of survival and long-term morbidity from drowning is the extent of CNS injury. Most submersion patients suffer a brief period of unconsciousness caused by cerebral hypoxemia. Full neurologic recovery rarely occurs after 10 minutes of anoxia in normothermic conditions; hypothermia under controlled circumstances can prolong this to more than 40 minutes.[254] In most cases, prolonged

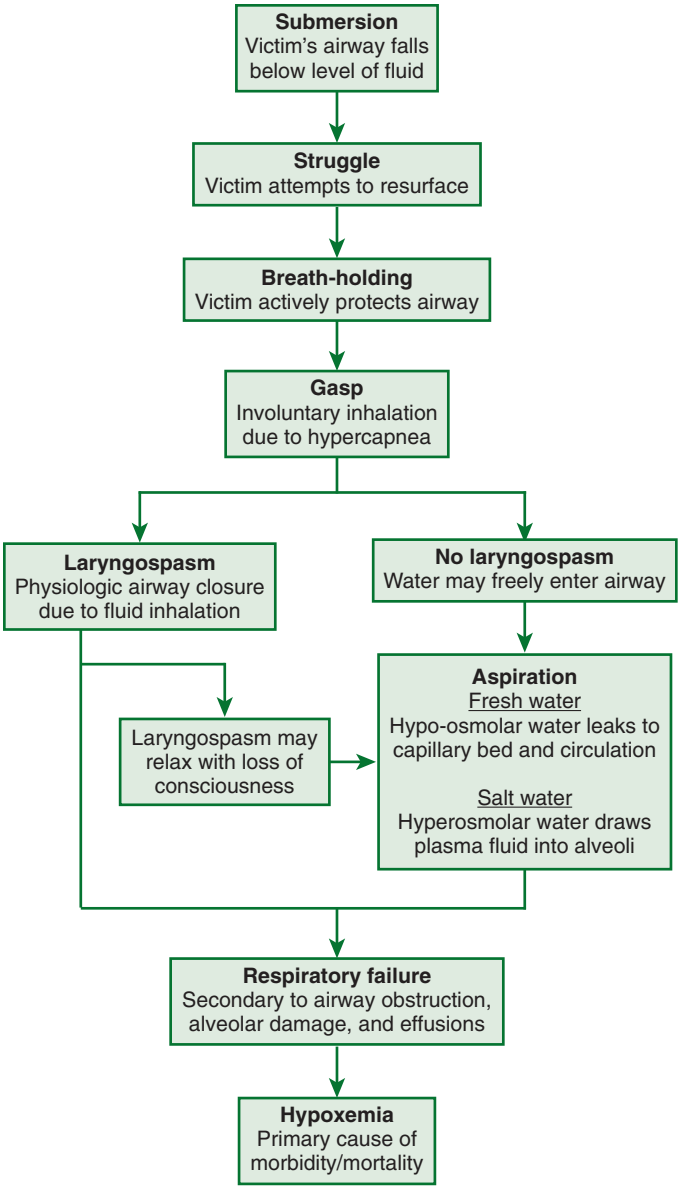

FIGURE 69-3 Pathophysiologic events of the drowning process. *(Courtesy Andrew Schmidt, DO, MPH.)*

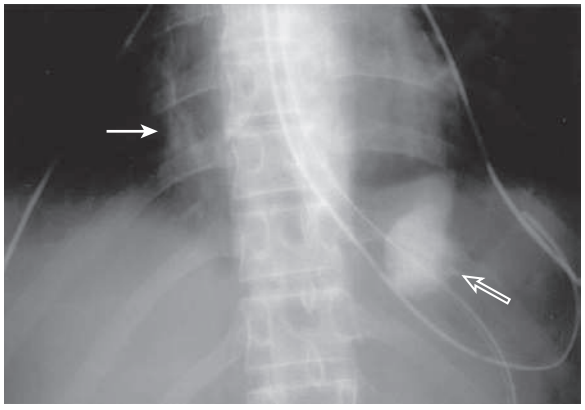

FIGURE 69-4 Chest radiograph taken in the emergency department, showing sand bronchograms in the right lower lobe *(solid arrow)*. Note sand within the gastric fundus *(open arrow)*. *(From Dunagan DP, Cox JE, Chang MC, et al: Sand aspiration with near-drowning: radiographic and bronchoscopic findings,* Am J Respir Crit Care Med *156:292, 1997.)*

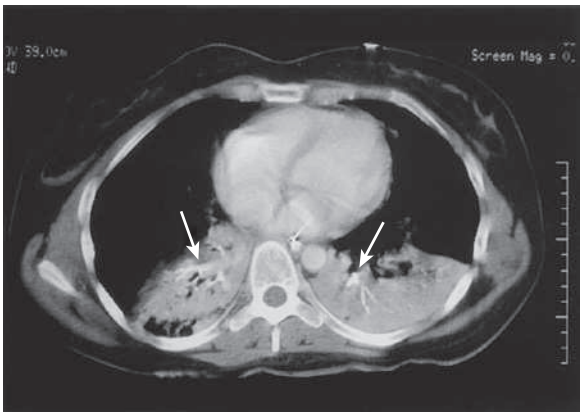

FIGURE 69-5 Chest CT scan showing bilateral sand bronchograms within the lower lobes *(arrows)* as well as significant air space opacification. *(From Dunagan DP, Cox JE, Chang MC, Haponik EF: Sand aspiration with near-drowning: radiographic and bronchoscopic findings,* Am J Respir Crit Care Med *156:292, 1997.)*

submersion leads to death from neurologic asphyxia. Even with aggressive CPR and return of spontaneous circulation, drowning is associated with significant neurologic morbidity. Patients who present either awake or with blunted mental status have a better prognosis than those who present in a coma; the mortality rate in one study was as high as 34% among patients who were comatose on arrival at the hospital.[52,151] Of surviving patients, 10% to 23% demonstrated severe and persistent neurologic sequelae.[52,151] Prolonged hypoxemia and acidosis lead to neuronal cell death and demyelination.[26,110,143] Thus, the longer the submersion time, the more CNS damage suffered. Areas of high metabolic activity in the brain that are most susceptible to damage from hypoxemia include gray matter (more than white), vascular end zones, cerebral cortex, thalamus, basal ganglia, and hippocampus. Initial computed tomography (CT) scanning of the brain after submersion that shows any abnormalities of edema, loss of differentiation between gray and white matter, or focal infarct is highly predictive of a poor outcome. Conversely, a normal initial CT scan of the brain is of little prognostic value.[195,201]

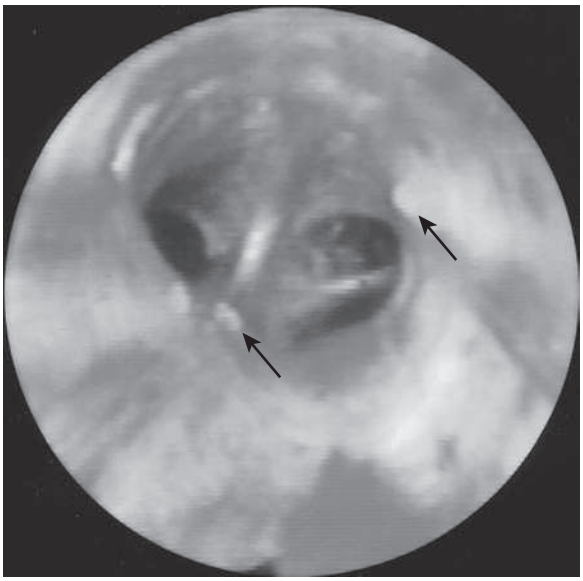

FIGURE 69-6 Fiberoptic bronchoscopy, demonstrating pieces of sand *(arrows)*, significant airway erythema, and inflammation following drowning and sand aspiration. *(From Dunagan DP, Cox JE, Chang MC, et al: Sand aspiration with near-drowning: Radiographic and bronchoscopic findings,* Am J Respir Crit Care Med *156:292, 1997.)*

After the initial hypoxic event, subsequent reperfusion of damaged neurons can result in cell lysis, as well as interstitial and cerebral edema that are further worsened by systemic acidosis, hypotension, hyperglycemia, and, if present, seizure activity.[181] Hypothermia can increase cerebral tolerance of ischemia and hypoxemia. There are multiple case reports of survival with good neurologic outcomes after prolonged submersion, particularly among children. This is thought to result from decreased cerebral oxygen demand in the setting of hypothermia.[21,51,205,206,211,225] This requires rapidly induced hypothermia before the onset of anoxic damage, which occurs with sudden submersion into very cold water. Therapeutic hypothermia (TH) in cases of cardiac arrest has resulted in an improved neurologic outcome, and there are several case reports of complete neurologic recovery after TH in drowning patients.[13,245,250] Drowning patients often arrive hypothermic at temperatures below the 32° to 34°C (90° to 93°F) goal of TH. This is an area of active research, and in the absence of randomized control trials of TH in non–ventricular fibrillation cardiac arrest, the decision to initiate TH is based on expert consensus and theoretical benefit. This is discussed further in the hypothermia section.

CARDIOVASCULAR SYSTEM

Cardiac rhythm, output, and function are affected by hypoxemia and acidosis, with more severe manifestations in prolonged submersion.[162] Dysrhythmias may occur as a result of acidosis and hypoxemia. Decreased cardiac output may result from direct effects of hypoxemia on the myocardium. Pulmonary hypertension caused by aspirated fluid can result in right ventricular overload, and further decrease cardiac output. Patients with underlying cardiac disease may be more susceptible to the effects of hypoxemia on myocardial function. A sudden cardiac dysrhythmia may precipitate submersion and drowning in patients with preexisting cardiac disease, although during postmortem analysis, this can be difficult to discern from submersion-related injury.

HEMATOLOGIC AND ELECTROLYTE DISTURBANCES

Prior data supporting the notion of systemic electrolyte disturbances from aspiration of saltwater are of little practicality for treatment of human drowning patients, because such derangements are rarely seen. Massive (i.e., > 22 mL/kg) instillation of saltwater in animal models resulted in disturbances in serum sodium, chloride, magnesium, and calcium,[46,255,260] presumably as a result of osmotic gradients across the alveolar epithelium. Humans who die by drowning aspirate less than 4 mL/kg of fluid volume.[149,152,156] Similarly, canine models have demonstrated gross hemolysis, resulting in hyperkalemia and disseminated intravascular coagulation after large-volume aspiration; however, clinically relevant changes in hematocrit and hemoglobin levels are rarely seen in human drowning patients.[153] Therefore, routine observation and laboratory analysis with treatment for measured abnormalities are all that are indicated in the hospital setting.

HYPOTHERMIA

For patients who do not drown immediately upon entering cold water, subsequent fatal or nonfatal drowning may result from hypothermia.[48,100] Water at 91.4°F (33°C) is thermally neutral, where heat loss equals heat production for a swimmer without clothes; water any colder than this leads to ongoing heat loss. The thermal conductivity of cold water is 25 to 30 times that of air.[100,130] Even in only moderately cold water, hypothermia may ensue rapidly and lead to a loss of consciousness and subsequent drowning. This is particularly true in children, who have less subcutaneous fat and relatively greater body surface area compared with adults.[21,49,84,94] Survival is unlikely after 60 minutes in water that is cooler than 32°F (0°C); however, people who have not submerged can survive for up to 6 hours at a water temperature of 59°F (15°C) (Table 69-1).[47]

TABLE 69-1 Estimated Survival Times in Cold Water

Water Temperature	Exhaustion or Unconsciousness in	Expected Survival Time
21-27°C (70-80°F)	3-12 hr	3 hr to indefinitely
16-21°C (60-70°F)	2-7 hr	2-40 hr
10-16°C (50-60°F)	1-2 hr	1-6 hr
4-10°C (40-50°F)	30-60 min	1-3 hr
0-4°C (32.5-40°F)	15-30 min	30-90 min
<0°C (<32°F)	<15 min	<15-45 min

Courtesy U.S. Search and Rescue Task Force: ussartf.org/cold_water_survival.htm.

There are many factors that affect an individual's response to cold-water immersion. Dramatic recoveries after prolonged hypothermia have been documented in children and adults.[186,211,225,259] These case reports of increased survival are associated with ice cold (< 6°C) water that rapidly induces the protective effects of the mammalian diving reflex. *Gradual* onset of hypothermia while drowning worsens outcomes. Therapeutic hypothermia (TH) is an area of active research in post cardiac arrest, where it has been shown to decrease cerebral metabolic demand and improve neurologic outcomes.[109] Protocols for TH were adopted by the American Heart Association in 2002 and by the European Resuscitation Council in 2003 for out-of-hospital cardiac arrest patients. Case reports of survival after submersion in cold water with profound hypothermia have provoked questions as to whether this would provide the same physiologic effect of neuroprotection for drowning patients who become rapidly hypothermic, and might warrant maintenance of a low core temperature in comatose drowning patients.[127,241] A panel of experts during the World Congress on Drowning in 2002 concluded that "drowning victims with restoration of adequate spontaneous circulation who remain comatose should not be actively rewarmed to temperature values above 32° to 34°C (90° to 93°F). If core temperature exceeds 34°C (93.2°F), hypothermia at 32° to 34°C (90° to 93°F) should be achieved as soon as possible and sustained for 12 to 24 hours."[245] There are several cases of full neurologic recovery after submersion with subsequent TH.[155,242] Optimal temperature management for submersion patients remains an area of active discussion.

The pathophysiologic response to hypothermia ranges from shivering that contributes to increasing the metabolic rate, to coma, massive metabolic acidosis, and spontaneous ventricular fibrillation. Patients progress from mild hypothermia, characterized by shivering, increased metabolic rate, and tachycardia, to profound hypothermia at varying rates, depending on surrounding water temperature, protection and clothing, duration of exposure, and position in the water (Figure 69-7).

Though not specifically drowning or hypothermia, in some cases, patients suffer from immersion syndrome, which involves sudden death from bradycardia, tachycardia, or ventricular fibrillation and cardiac arrest from cold-water exposure before onset of systemic hypothermia.[178] Immersion in cold water (< 5°C [< 41°F]) produces a rapid fall in core body temperature. Heat loss through convection and conduction is further compounded by increased muscle activity during swimming and struggling.

The "diving reflex" in diving mammals is a vestigial but inducible reflex in humans that may contribute to survival in cases of prolonged submersion and subsequent hypothermia.[49,84,95,141] The reflex is activated by vagal stimulation from cutaneous receptors that respond to cold by shunting blood to the brain and cardiac muscles and away from the skin, extremities, and splanchnic vascular beds, along with bradycardia and a decreased metabolic rate. Bradycardia and vasoconstriction in all vascular beds (except those serving the heart and brain) result in the preserved mean arterial pressure for those organs and overall decreased cardiac output. The reflex may be inducible in only 15% to 30% of humans,[22] but may be a contributing factor for persons who survive drowning.[64,84,121] However, some discount its role.

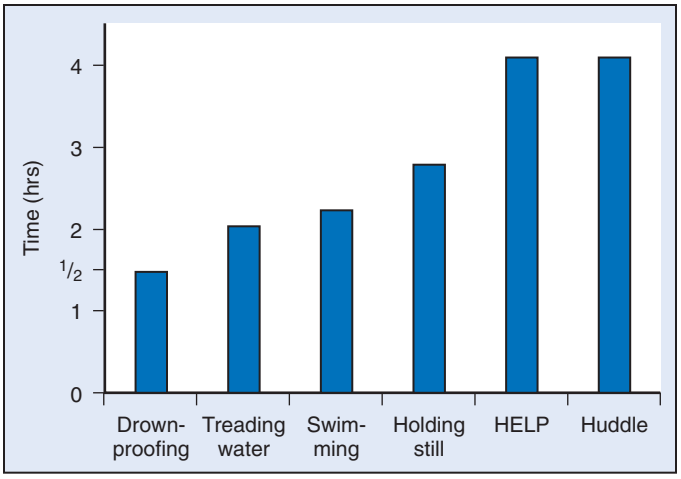

FIGURE 69-7 Survival times in cool (10° C [50° F]) water using various techniques in several situations. HELP, heat escape lessening posture. *(Data from Collis ML: Survival behaviour in cold water immersion. In Proceedings of the Cold Water Symposium, Toronto, Royal Life-Saving Society of Canada, 1976.)*

Although the mechanism is not completely understood, there are researchers that propose "autonomic conflict" as an additional mechanism of death during immersion in cold water. During submersion in cold water with breath-holding, the sympathetic nervous system is activated during the cold shock response, and the parasympathetic nervous system is activated through the diving reflex. This simultaneous activation of both limbs of the nervous system has triggered dysrhythmias in healthy human volunteers, which has been extrapolated as a mechanism of death through fatal dysrhythmias in drowning patients.[19a,53,146,229,230]

Immersion hypothermia occurs as the core temperature falls. It is discussed in detail in Chapter 8.

MANAGEMENT: THE ELEMENT OF TIME

Time plays a significant role in determining the ultimate outcome of rescue of a drowning patient. The duration of anoxic time may be unknown or unreliable for a variety of reasons, including an unwitnessed event or unrecognized call for help (e.g., the waving of hands or thrashing about may be confused with play activity). Witnesses may exhibit panic and frantic behaviors, further impairing effective rescue.

Time is of the essence. The published world record for conscious breath-holding while underwater is 17 minutes and 4 seconds,[228] and it should be noted that this feat was achieved after months of training under highly controlled circumstances, which included extensive pre-oxygenation. Most humans are unable to stay conscious underwater or avoid aspiration after a few minutes. Irreversible neurologic deficits are common after 4 to 5 minutes.[51] The longest documented period of submersion time in an unconscious patient with survival is 66 minutes[24] (a pediatric patient in cold water). Hypothermia may offer a protective benefit and prolong the time during which resuscitation can still be successful. There are case reports in which patients survived after 40 minutes of submersion in cold water with complete or nearly complete recovery.[211,225,259] The lowest recorded temperature in a human with subsequent survival and neurologic recovery was taken from a Norwegian skier trapped in a frigid river, who had a core body temperature of 13.7° C (56.6° F) when extricated.[77] Table 69-1 describes time parameters surrounding expected survival at various water temperatures.

An extremely rapid response is required in some cases, whereas no immediate response or body recovery is required in others. This decision depends on the characteristics of the patient, presumed length of submersion, possible associated traumatic injuries, and water temperature. It is unnecessary to put rescuers at risk to retrieve a drowned patient whose survival would defy physiologic reality. The initial evaluation and total time of submersion should help clinicians determine the appropriate duration of resuscitative efforts on a drowned patient with potentially significant morbidity or brain death.

CLINICAL PRESENTATION: A CASE HISTORY

This case demonstrates many of the clinical features of drowning patients. The patient was a 52-year-old previously healthy male who worked the night shift on a barge in the brackish waters of the Duwamish Slough near Puget Sound. One night, he tripped on an uncoiled rope on the deck and fell 20 feet into the water. Despite struggling to swim, he lost his orientation and drifted under the barge. Another worker saw him fall and drift under the boat, so he astutely threw his flashlight into the water away from the side of the boat. The patient saw the light, desperately swam to it, and was subsequently pulled from the water by coworkers. His total submersion time was approximately 2 minutes.

Emergency personnel transported the patient to Harborview Medical Center. In the emergency department, he was anxious but alert and oriented, and mildly short of breath with a cough. His vital signs were as follows: respiratory rate, 24 breaths/min; pulse, 115 beats/min; blood pressure, 145/100 mm Hg; and temperature, 36° C (96.5° F). The remainder of his examination was normal except for diffuse rhonchi throughout both lung fields. Arterial blood gas levels were as follows: pH, 7.48; arterial partial pressure of carbon dioxide ($PaCO_2$), 32 mm Hg; and arterial partial pressure of oxygen (PaO_2), 58 mm Hg on a face mask with high-flow oxygen. A chest radiograph showed a normal cardiac silhouette with diffuse bilateral patchy opacities. He was admitted to the intensive care unit for observation.

Within the first hour, his gas exchange worsened despite adequate alveolar ventilation. He was endotracheally intubated and placed on mechanical ventilation in the volume ventilation mode. Despite high concentrations of oxygen, his gas exchange capabilities further deteriorated, and he was given a trial of positive end-expiratory pressure (PEEP). At 15 cm H_2O pressure, the patient's blood pressure fell to 76/52 mm Hg. Volume resuscitation was increased. A pulmonary artery catheter was placed to monitor cardiac function. During the initial PEEP trial, pulmonary capillary wedge pressures were low (i.e., < 10 mm Hg). The cardiac index fell from 2.8 to 1.8 L/min/m^2 body surface area after the PEEP was increased from 10 to 15 cm H_2O. PEEP was therefore reduced to 10 cm H_2O and more vigorous volume resuscitation was instituted, after which the cardiac index and blood pressure improved so that PEEP could safely be increased, which was necessary to maintain adequate oxygenation.

Over the next 6 days, the patient's lung compliance initially decreased and then increased; his chest radiograph worsened and then slowly improved; and his gas exchange improved so that he was extubated on hospital day 10. It was another 2 months before he was able to be physically active, and he had dyspnea on exertion for approximately 6 months after his accident. At hospital discharge, pulmonary function tests showed a moderate decrease in both vital capacity and total lung capacity, mild obstruction of airflow, and severely decreased diffusion capacity for carbon monoxide. Over the next 6 months, the patient's vital capacity and total lung capacity returned to normal, but his diffusion capacity remained mildly decreased.

This case demonstrates a number of important features of drowning with aspiration and respiratory failure. First, the patient was extremely lucky that his fall was witnessed and subsequent rescue was rapid, limiting exposure and submersion times. He arrived at the hospital conscious and alert, one of the best prognostic signs, and had no comorbid conditions. His clinical course was consistent with acute lung injury from aspiration of brackish water, and the initial worsening and subsequent improvement of lung mechanics and gas exchange were typical of ARDS. Fortunately, he had no other organ failure, and his recovery was unremarkable.

There is a spectrum of types of morbidity from drowning, ranging from asymptomatic to unresponsive, apneic, and pulseless. Classification systems based on observations from first-aid providers assist with determination of which patients require hospitalization.[221]

ON-SCENE MANAGEMENT

The scene at drowning incidents is often chaotic. Patients are not usually found flailing in the water; rather, they are more commonly found floating on or motionless underneath the surface. Rescue attempts must always take into consideration the safety of rescuers to avoid creating additional victims. Safety devices should be used to tow the patient, or life preservers should be thrown to people in trouble before a human lifesaver enters the water. Persons without current training in water-rescue techniques specific to the conditions (e.g., open water, swift water, ice) should never enter the water to rescue a drowning patient. Well-intentioned but ill-advised heroic efforts can create additional victims and compound the tragedy.[68]

Upon arrival at the scene, accurate timing and documentation of the course of events, including vital signs, clinical state, and environmental conditions, should begin.[19] The scene description, estimation of time of submersion, type and temperature of the water and air, and events during transport to the hospital may be critical information. If multiple patients or rescuers are present, the accident scene may be disorganized, so it is imperative that someone assume a leadership role. After the patient is out of the water, basic life support should be instituted. Evaluation for concomitant trauma and associated injuries should be assessed once the patient is out of the water. Findings and circumstances consistent with child abuse, seizures, cardiac arrhythmias, trauma, and other medical issues should be ascertained.

Hypoxemia is the most significant consequence of submersion, so immediate attention to the airway and oxygenation is of paramount importance to the outcome. Appropriate resuscitative measures should be instituted immediately after extrication from the water and continued by rescuers at the scene. Although there are various products and training techniques designed to promote rescue breathing in the water, this is difficult in the best of circumstances, and is generally discouraged except when performed by trained rescuers (Figure 69-8). Rapid extrication from the water is the priority to ensure that CPR and rescue breathing are adequate. In-water rescue breathing should only be instituted when rapid extrication is not feasible. Transport to an emergency facility with ongoing CPR should take place unless resuscitation is determined to be futile or successful.[8] Concomitant hypothermia sometimes makes clinical determination in the field difficult at best.

THE ASYMPTOMATIC PATIENT: GRADES 0 AND 1

Patients with no other comorbid conditions who have been rescued from the water and who are alert, with a clear chest examination on auscultation, no respiratory distress, and with or without coughing, may not need further medical care but still present a dilemma. Although there are published reports of delayed complications in drowning patients who were initially minimally symptomatic, these case reports have recently come under scrutiny. More recent studies show that patients may have minimal symptoms that become progressively worse over the next 5 to 8 hours, but patients who are truly asymptomatic do not show delayed complications. Minimally symptomatic patients should be advised to seek care if they develop worsening symptoms over the ensuing 8 hours.*

Hypothermia is often difficult to ascertain at the scene, so it may be prudent to have the person evaluated, even if only briefly, at a medical facility.[8] Conscious and cooperative patients should be protected against hypothermia with passive warming techniques, protected from wind, and offered dry clothes and blankets. If the person remains asymptomatic with normal vital signs and stable arterial oxygen saturation (if testing is available) on ambient air for 10 to 15 minutes, then it is not likely that the person will require further medical care.

FIGURE 69-8 Mouth-to-mouth ventilation in the water is difficult in the best of circumstances. *(Courtesy Alan Steinman, MD.)*

THE SYMPTOMATIC PATIENT: GRADES 2, 3, AND 4

All submersion patients requiring on-scene intervention or resuscitation, or showing signs of distress (e.g., anxiety, tachypnea, dyspnea, syncope, persistent cough, presence of foam in the mouth or nose, changes in vital signs), should be evacuated or transported to a hospital for evaluation.

Protection of the airway to ensure oxygenation and ventilation is the first priority. Maintaining perfusion to reverse the metabolic consequences of acidosis comes next. The airway should be protected from aspiration by placing the patient in a lateral recumbent (i.e., recovery) position if possible. Vomiting is common with submersion, and aspiration can worsen lung injury. Measures should be taken to prevent hypothermia and shivering. Rescuers must maintain vigilance and treat cardiac dysrhythmias that may arise as a result of hypoxemia. The management actions listed in Table 69-2 can then be considered. Routine cervical spine immobilization is unnecessary and should be reserved for patients with a known or suspected significant mechanism of injury, or worrisome clinical examination (see Cervical Spine Injury, later).[246]

THE PATIENT IN RESPIRATORY OR CARDIOPULMONARY ARREST: GRADES 5 AND 6

Approximately half of drowning resuscitations involve bystander CPR.[19,192] Initiation of immediate ventilatory support and early CPR, if indicated, results in a better prognosis and outcomes.[19,50,64,81,94,181,185,216] In an 11-year study of pediatric drowning incidents in Houston, Texas, the biggest predictor of survival was prompt CPR by lay responders.[249] Initiation of chest compressions while the patient remains in the water is ineffective, delays extrication, and may further endanger the patient and rescuer. Effective CPR cannot realistically be started until the patient is out of the water and on a solid surface. Alternatively, rescue breathing should be initiated as soon as the patient's airway can be opened, even if in the water; this intervention improved survival rates threefold in one series of cases[222] (see Figure 69-8). The success of this intervention often depends on a well-trained and skilled rescuer, but is very useful in the appropriate setting. An example would be a child who is being pulled unconscious toward a boat or land in calm water. The extra few seconds taken to provide rescue breaths can provide critical oxygenation that may delay otherwise imminent cardiac arrest caused by hypoxemia.

When the individual is out of the water, either mouth-to-mouth or mouth-to-nose ventilation can be used.[8] If it is available at the scene, supplemental oxygen should be initiated as soon as possible. If the patient is spontaneously breathing, a face mask with a reservoir bag of approximately 2.5 L can be used. If a

*References 42a, 87, 111, 134a, 148a, 171a, 178a, 181a, 187a, 198a, 206a, 221.

TABLE 69-2 Prehospital Management and Classification of Drowning Patients

Grade	The Asymptomatic Patient		The Symptomatic Patient			The Patient in Respiratory or Cardiopulmonary Arrest	
	0	**1**	**2**	**3**	4	5	6
Mortality (%)	0	0	0.6	5.2	19	44	93
Pulmonary Examination	No cough or dyspnea	Normal auscultation with cough	Rales, small amount of foam	Acute pulmonary edema	Acute pulmonary edema	Respiratory arrest	Cardiopulmonary arrest
Cardiovascular Findings	Radial pulses	Radial pulses	Radial pulses	Radial pulses	Hypotension	Hypotension	
On-scene Management	Release at scene with education	Rest, rewarm, reassure, and release	Oxygen via nasal cannula; observe for 6-24 hr	Oxygen via nonrebreathing mask; advanced cardiac life support	Oxygen via nonrebreathing mask; advanced cardiac life support	Load and go	
Transport	No	No	Transport or observation	Yes	Rapid	Rapid	
En Route Management			Vital signs	Vital signs	Possible endotracheal tube and manage pressure	Advanced cardiac life support	
Hospital Management			Emergency department or overnight observation	Admission for observation	Intensive care unit	Intensive care unit	Intensive care unit

Courtesy Justin Sempsrott, MD. Adapted from Szpilman D: Near-drowning and drowning classification: a proposal to stratify morality based on the analysis of 1,831 cases, *Chest* 112:660, 1997.

nonrebreathing mask, portable positive end-expiratory pressure (PEEP) valve, or portable continuous positive airway pressure (CPAP) device is available, oxygen should be delivered at a high flow rate (i.e., 10 to 15 L/min). Using CPAP and PEEP in the field remains an area of controversy. Multiple emergency medical services (EMS) systems have successfully used this modality for management of acute pulmonary edema in congestive heart failure.[227,245] To date, there are no randomized controlled studies of the application of either prehospital PEEP or CPAP for drowning patients, and only several case reports of CPAP application for drowning.[59] A CPAP mask should be used cautiously if there is any concern about vomiting or loss of airway protective reflexes.

Draining water from the lungs of submersion patients dates back to the 17th and 18th centuries, but is no longer recommended. The Dutch method consisted of rolling subjects over a barrel; another method involved flinging subjects over a horse, which was then made to trot. In 1975, the Heimlich maneuver was introduced for victims of choking, which was subsequently recommended as a treatment for drowning patients. This maneuver is no longer recommended for drowning.[202] For patients who have swallowed large amounts of water, gastric distention can interfere with ventilation by increasing intraabdominal pressure. In such instances, gastric decompression by nasogastric tube is recommended.[7,198,202] Digital or visual examination for foreign bodies should be done, and if one is present, it should be removed with a swipe or grasp of the fingers. Foreign bodies (e.g., sand, beach flotsam, seaweed) were reported in up to 54% of cases in one series of surf beach-related submersions.[140]

Vomiting during resuscitation has been reported in 86% of cases involving drowning patients receiving CPR.[140] If vomiting occurs, the patient should be rolled onto the side or have the head turned to the side and vomitus removed with a cloth or finger-sweep maneuver.[181] If spinal injury is of concern, the patient should be logrolled, maintaining linear integrity of the head, neck, and torso.[8] The risk of aspiration from vomiting may be reduced by application of cricoid pressure during endotracheal intubation.[178] Because most beaches, riverbanks, boat ramps, and other waterway access points are sloped, patients should be placed perpendicular to the incline so that the head and feet are at the same level.[20]

When the patient is out of the water and the airway and breathing have been addressed, the presence or absence of adequate circulation should be ascertained. In cases of hypothermia or hypotension, a pulse may be difficult to identify. If ventilation or cardiac function is impaired, chest compressions should be initiated as soon as the patient is removed from the water. For patients who are more than 1 year old, an automated external defibrillator (AED) may be used to evaluate the heart rhythm. AED electrode pads with pediatric attenuation should be used for children who are between 1 and 8 years old.[8] However, some believe that there is no major role for an AED in this setting[19,192] because ventricular fibrillation is rare after drowning in an otherwise healthy patient, and a priority should be placed on oxygenation and ventilation over defibrillation.[19] If the field rescue team is capable of advanced life support (ALS), then cardiac monitoring, intravenous or intraosseous access, fluids, and medications should be administered according to ALS protocols. Basic life support or advanced cardiac life support should continue until the patient's core body temperature is more than 30°C (86°F).[8] Table 69-2 shows a classification scheme for drowning field assessment and management.

CERVICAL SPINE INJURY

Cervical spine injury is rare in drowning except in high-impact accidents, such as high-speed boating accidents, surf-related injuries, and diving accidents.[107,246] Evaluation of the breathing pattern and breath sounds is critical, because abnormal respiratory patterns can indicate cervical spine injury unrelated to direct pulmonary injury from submersion. Unless a focal neurologic deficit, significant mechanism of injury, altered mental status/unreliable examination, or midline neck or back tenderness (or a distracting injury preventing patient identification of such tenderness) is present, routine spine immobilization is unnecessary and may delay lifesaving resuscitation measures.[107,246] In cases in which neck injury is likely based on the mechanism of injury or suggested by physical examination, appropriate immobilization should be considered.[246] In the wilderness water setting, surfboards, paddles, or small watercraft may be used to immobilize and transport the patient (Figure 69-9). Evaluation for additional fractures or internal or intracranial injuries should be considered for any patient with a cervical spine injury.[64,95,181]

FIGURE 69-9 Surfboards can be used for spinal immobilization. *(Courtesy Lifeguards Without Borders.)*

THE OBVIOUSLY DEAD OR STILL-SUBMERGED PATIENT

Some persons may appear to be dead when they are retrieved from the water. They are unresponsive with a normal temperature, demonstrate postmortem lividity, and may be asystolic and apneic. If CPR has been in progress for more than 25 to 30 minutes with no return of vital signs, persons who are retrieved from warm water may be considered dead at the scene.[45,67,194] However, because of case reports of neurologically intact survival after submersion for more than 60 minutes in icy cold (< 6°C) water,[43,63,75,105] rescue crews must start and continue life support measures for all persons who are not obviously dead or who are hypothermic. After 1 hour of submersion in warm water, scene rescue efforts should transition to body recovery efforts.[168] Many drowning deaths become medical examiner cases, because the possibility of foul play must be investigated, and appropriate local authorities should be contacted.

TERMINATING RESUSCITATION EFFORTS

The decision to end CPR in drowning cases is an emotionally charged issue. In remote wilderness settings, it may become necessary to terminate resuscitation efforts on scene; this decision should be based on the distance to definitive care, time the patient has been submerged, and condition of the patient. The standard of care in the field is to terminate resuscitation efforts if there is no return of spontaneous circulation after 25 to 30 minutes of chest compressions or if the patient was submerged for more than 1 hour[67,168] (see Prognosis and Termination of Resuscitation, later).

EN ROUTE TO DEFINITIVE CARE

During transport, the goal is optimization of oxygenation, ventilation, and perfusion. Supplemental oxygen should be delivered by a simple or nonrebreathing face mask or nasal cannula. A pulse oximeter should be used to monitor arterial oxygen saturation, but rescuers should recognize that pulse oximetry might be inaccurate as a result of hypothermia and peripheral vasoconstriction. Body heat loss should be minimized by keeping the patient dry, out of the wind, and covered with blankets. Although shivering is a good prognostic sign, it increases tissue oxygen demand and caloric expenditure, and should be avoided by

warming the patient, removing wet clothing, and protecting the patient from wind and weather. Detailed assessment of neurologic status with attention to fluctuations in the patient's Glasgow Coma Scale score should be made repeatedly. The use of ALS medications should follow standard protocols. The core body temperature and acid-base status of the patient may not be known in the field, making effects and metabolism of drugs unpredictable. Drugs should be used only when absolutely necessary, preferably in a hospital setting. If active rewarming techniques using cardiopulmonary bypass or extracorporeal membrane oxygenation are anticipated, EMS protocols dictating transport of the patient to appropriately equipped facilities are recommended.

EMERGENCY DEPARTMENT TREATMENT OF DROWNING PATIENTS

Upon arrival at the medical facility, the patient's airway, breathing, circulation, and mental status should be reassessed. Patients who initially presented with minimal symptoms may develop worsening pulmonary function over the next several hours.

Box 69-3 outlines the emergency department care of drowning patients. Rapid correction of hypoxemia and acidosis are the most important priorities. Although severe drowning cases are true medical emergencies, if patients reach the emergency department before cardiac arrest and are given timely and effective treatment, nearly all will recover without significant neurologic or pulmonary sequelae.[50,117,212,213]

Because of the potential delay in symptom manifestations, even asymptomatic patients should be observed for a minimum of 4 hours. For asymptomatic patients, if there is a history of significant or prolonged submersion, clinicians may choose to obtain arterial blood gas measurements to look for a widened alveolar-arterial gradient, or they may obtain chest radiographs to inspect for occult injury.[121,150,177,183] A patient with a normal chest radiograph taken on admission may have a markedly abnormal radiograph several hours later[143]; regardless of the patient's clinical appearance, a baseline chest radiograph should be obtained.[121] Asymptomatic patients may be safely discharged from the emergency department if they have a normal Glasgow Coma Scale score, respiratory effort, chest radiograph, and oxygen saturation on room air after 4 to 8 hours of observation.*

Patients may present with a range of respiratory symptoms, from mild cough to profound hypoxemia and respiratory distress. Symptomatic patients may complain of cough, sore throat, chest tightness, and/or shortness of breath. The first and most important step is to evaluate the airway and breathing to address hypoxemia and acidosis. Even if the patient is adequately breathing with spontaneous and unassisted ventilations, the patient should still be provided with supplemental oxygen.

If the patient is unable to maintain effective spontaneous breathing (to prevent hypoxemia) and ventilation, CPAP or endotracheal intubation should be considered. Severe hypoxemia and intubation are not unusual with drowning; in one series, 53% of drowning patients had severe hypoxemia, and 36% required mechanical ventilation.[153] Serial arterial blood gas measurements may be helpful to determine the degree of hypoxemia and whether patients are improving without advanced airway management. Arterial blood gas measurements will determine (1) alveolar ventilation (i.e., acceptable $PaCO_2$ and pH); (2) gas exchange (i.e., $PaO_2 > 60$ mmHg on supplemental oxygen); and (3) perfusion (i.e., no metabolic acidosis). CPAP can be used in a brief trial before proceeding to endotracheal intubation in awake patients with low PaO_2 levels despite administration of high-flow oxygen. If arterial blood gas parameters cannot be maintained and the patient is not improving with less invasive measures, including CPAP, mechanical ventilation should be initiated using the volume-ventilation mode and a high level of supplemental oxygen. Other criteria for endotracheal intubation include a patient who is unable to handle his or her secretions or who has a seriously altered mental status. After advanced airway management, consider nasogastric or orogastric tube placement to prevent aspiration of gastric contents. The core temperature should be monitored. All patients with abnormal vital signs, hypoxemia, abnormal chest radiographs, or any respiratory distress should be admitted to the hospital for, at a minimum, observation.

BOX 69-3 Emergency Department Management of Drowning Patients

Check Airway/Ventilation
Adequate ventilation
- Supplemental oxygen: nonrebreathing mask at 12-15 L/min or via demand valve

Inadequate ventilation
- Borderline patients: consider CPAP
- Comatose patients or those with $PaO_2 < 90$ mm Hg on 15 L/min nonrebreathing mask or $PaCO_2 > 45$ mm Hg: endotracheal intubation with PEEP as needed

Diagnostics
Arterial blood gas studies for mechanically ventilated patients, SpO_2 monitor for spontaneously breathing patients
Chest radiograph
Cardiac monitor
Cervical spine radiograph/trauma evaluation if indicated by mechanism
Assess for hypothermia, hypoglycemia, electrolyte abnormalities

Further Interventions
Intravenous access, hydration
Nasogastric tube if significant aspiration/ingestion of water

Disposition
Admit
- All patients with abnormal vital signs, abnormal radiologic findings, respiratory symptoms, or abnormal findings on blood gas measurements
- ICU admission preferred for all patients not being admitted for simple observation
- Rehydration and ventilatory stabilization should be underway prior to ICU admission

Criteria for discharge in 4-6 hours
- Asymptomatic (no cough or respiratory complaints)
- No vital sign or examination aberrancies (in particular, normal SpO_2 on room air, normal lung auscultation, normal Glasgow Coma Scale score)
- No diagnostic abnormalities (normal chest x-ray)

IN-HOSPITAL TREATMENT OF DROWNING

Although the complexity of patient management is as variable as the condition of the patient, clinicians facing complex management decisions should keep in mind that nearly all successful clinical interventions focus on correction of acidosis, hypoxemia, and hypoperfusion. The following paragraphs present a systems-based discussion of in-hospital management of drowning.

From an epidemiologic and medical systems standpoint, the 2002 First World Congress on Drowning in Amsterdam made the following recommendations regarding in-hospital treatment of drowning[73]:

- Registration of drowning patients and collection of clinical data regarding in-hospital resuscitation and treatment of complications of drowning are recommended.
- Development of a uniform reporting system to register and collect these data (similar to the Utstein-style set of guidelines established for the uniform reporting of cardiac arrest) is desirable. Lack of standard definition and outcome criteria hinder the exchange of data across medical and basic science specialties. In 1990, a group of researchers studying

*References 37, 42a, 87, 111, 134a, 148a, 171a, 178a, 181a, 187a, 198a, 206a, 213, 221.

out-of-hospital cardiac arrest met at the Utstein Abbey in Norway and set forth consensus criteria for uniform reporting of data related to cardiac arrest. A similar consensus was reached at the 2002 World Congress on Drowning, which set forth the criteria for reporting drowning data and recommended that only three outcomes be recognized: death, no morbidity, and morbidity (this was further categorized as moderately disabled, severely disabled, vegetative state/coma, and brain death).[111]

- Hospital treatment of severe drowning patients should be concentrated in specialized centers.

PULMONARY MANAGEMENT

Therapy usually begins in the field with oxygen given by nasal cannula or face mask. Progression to a nonrebreathing mask or to noninvasive positive-pressure ventilation is determined by the initial response to oxygen therapy, respiratory symptoms, arterial oxygen saturation, and arterial blood gas analysis. When the decision has been made to begin mechanical ventilation, low tidal volumes (i.e., 6 mL/kg) should be used initially, and PEEP started at a low level (i.e., 5 to 10 cm H_2O) if a PaO_2 of more than 60 mm Hg cannot be maintained with a fraction of inspired oxygen of 0.6. Positive-pressure strategies such as PEEP should be used judiciously, because higher intrathoracic pressure may impair venous return and result in hypotension, decreased cardiac output, and further compromise of systemic oxygen delivery. Caution should be used if there is any concern about vomiting before applying a CPAP mask. However, at appropriate levels and when needed, PEEP can be remarkably effective for recruiting edematous or collapsed alveoli, and can thus improve gas exchange and oxygenation in patients with atelectasis, substantial aspiration, and pulmonary edema. Volume ventilation is preferred to ensure that adequate alveolar ventilation (i.e., approximately 10 L/min in adults) is delivered, reflected by acceptable pH and $PaCO_2$ levels.

A feature of drowning is cough and respiratory irritation caused by inhaled water and particles in the tracheobronchial tree. Cough and bronchospasm can impair airflow, resulting in $\dot{V}/\dot{Q}$ mismatch and hypoxemia. Aerosolized albuterol, which is a relatively selective β_2-adrenergic agonist, is the initial treatment of choice for bronchospasm. It can be administered to spontaneously breathing patients via face mask or to intubated patients using a T-piece adaptor. Ipratropium via nebulizer may be added to decrease bronchial smooth muscle spasm.[17,172,200]

Discontinuation of ventilatory support should follow standard principles. The timing may be influenced by surfactant regeneration, which is a situation uncommon in other mechanically ventilated patients. Surfactant washout or functional inactivation caused by drowning requires 2 to 4 days of CPAP and PEEP to allow adequate surfactant regeneration.[73] There has been interest in administration of exogenous surfactant for drowning patients with acute respiratory failure.[244] Theoretical benefits include more rapid improvement in pulmonary function and decreased required duration of mechanical ventilation,[89] as well as reduced infection rates because of surfactant's role in pulmonary immune function.[90] Although published case reports generally suggest a benefit,[82,124,217,220] multipatient studies have shown more variable results.[9,118,243,248] Prophylactic antibiotics to prevent pneumonia after drowning are not recommended (see Infectious Diseases, later). Most patients without underlying pulmonary disease or prolonged resuscitation or mechanical ventilation times recover full pulmonary function, as shown in Figures 69-10 and 69-11.

CARDIOVASCULAR SYSTEM AND HEMODYNAMICS

Avoidance of hypoxemia and acidosis requires monitoring and support of cardiac output and peripheral perfusion. From a hemodynamic perspective, drowning patients are likely to have mild intravascular dehydration.[197] Mechanical ventilation can reduce the circulating blood volume through increased intrathoracic pressure and decreased central venous return. Controlled studies have shown that hydration and adequate systemic blood pressure are critical to successful resuscitation; without adequate volume resuscitation, neither inotropic support nor mechanical ventilation alone results in adequate tissue oxygenation or survival.[197,223]

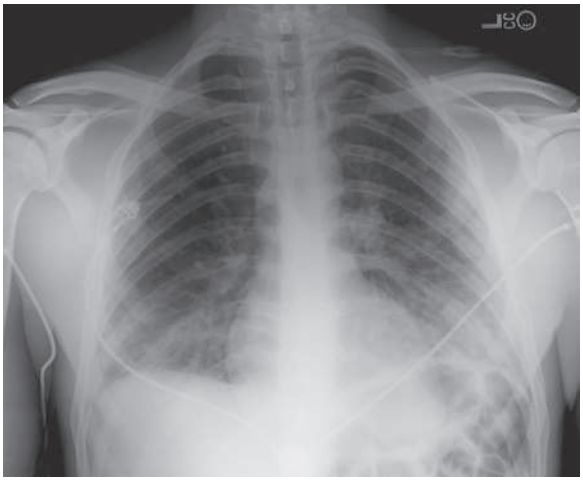

FIGURE 69-10 Submersion-related pulmonary edema. *(Courtesy U.S. Navy.)*

Dysrhythmias seen during resuscitation after drowning most likely result from hypoxemia, acidosis, and hypothermia rather than from a primary cardiovascular abnormality. Correction of the acid-base status and oxygenation may be the only treatments required. The dysrhythmias observed in controlled animal models of drowning and in human drownings include bradycardia, tachycardia, absent or decreased P waves, a widened PR interval, a widened QRS wave, ST segment elevation or depression, inverted or peaked T waves, atrioventricular dissociation, atrial fibrillation, premature ventricular contractions, and ventricular fibrillation.[158] The cause of dysrhythmias is variable, and parameters (e.g., comorbidity, ingestions, trauma, electrolyte disturbances, primary cardiac pathology [ischemia, heart failure]) other than any immediate drowning pathophysiology should be considered.

After initial cardiopulmonary and hemodynamic stabilization, further invasive monitoring should be considered to help guide therapy. An arterial catheter can be used to monitor the blood pressure and arterial blood gases. A pulmonary artery catheter may be useful for hemodynamically unstable patients or those requiring PEEP of 15 cm H_2O pressure or more to maintain oxygenation. Pulmonary capillary wedge pressure and cardiac output determinations allow a quantitative approach to fluid management in the hypotensive patient. Vasopressor agents (e.g.,

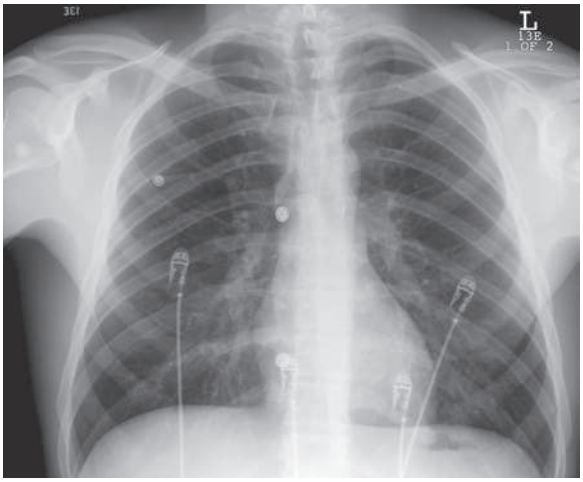

FIGURE 69-11 Resolution of the edema seen in Figure 69-10. *(Courtesy U.S. Navy.)*

dopamine, dobutamine, norepinephrine) may be required to maintain the mean arterial pressure adequate for tissue perfusion, but there is no evidence to suggest they improve the final outcome.[158] An ongoing need for vasopressors after the initial resuscitation and stabilization phases should prompt consideration of other causes of circulatory compromise, such as ongoing volume loss, cardiac failure or ischemia, traumatic injuries, or septic shock. Significant hypothermia can compromise cardiac function, cause dysrhythmias, and decrease the efficacy of electrical and pharmacologic interventions.[232,233]

CENTRAL NERVOUS SYSTEM

Global hypoxemia and hypoperfusion can result in cerebral anoxic injury with consequent focal or generalized neurologic deficits. Inflammatory damage from hypoxemia and reperfusion leads to loss of cell membrane integrity, resulting in extracellular fluid movement and cerebral edema, ultimately producing elevated intracranial pressure (ICP) and possible uncal herniation. The best preventive measure against this cascade of neurologic injury is correction of hypoxemia and hypoperfusion.

Accurate assessment of the neurologic status upon presentation is crucial to predicting the outcome. Serial neurologic examinations, brain imaging, and physiologic testing during the initial 24 to 72 hours of therapy may be helpful for assessing the patient's neurologic progress and projected outcome.[110] Measurements of ICP and cerebral oxygenation have been used as monitoring parameters, but a consensus recommendation from the Brain Resuscitation Task Force of the 2002 World Congress on Drowning states there is insufficient evidence that routine ICP monitoring and management alter outcomes.[245]

Certain neuroimaging modalities may be helpful for diagnosing conditions of particular concern, but routine imaging is rarely helpful for the drowning patient. CT scanning may detect severe brain injury if the scan is abnormal early during the patient's presentation[195]; however, in most cases, unless traumatic injury is suspected, the initial scan is normal and has limited usefulness.[201,224] Magnetic resonance imaging (MRI) and spectroscopy may each be more helpful than CT for showing early signs of injury and cerebral metabolic changes.[62] There is insufficient evidence to support the use of other neurologic diagnostic tools (e.g., brainstem auditory evoked potential response, somatosensory evoked potentials).[110]

Neurologic conditions of concern include seizures, metabolic disorders, and CNS infections. Seizures may occur as a result of cerebral edema or meningitis and should be treated appropriately. An electroencephalogram (EEG) may be helpful for detecting occult seizures; however, if seizures are not suspected, an EEG is of limited usefulness.[21,245] Diuretics and mannitol have been recommended if there is evidence of cerebral edema, but no studies have documented that osmotic diuretics improve the neurologic outcome of drowning patients. Critically ill and ventilated intensive care unit patients have shown improved outcomes if glucose levels are maintained in the range of 80 to 140 mg/dL. Although this study did not address drowning specifically, this should be considered part of the metabolic treatment of ventilated drowning patients.[239] Hyperglycemia has been linked to worse outcomes from acute brain injury.[11] Hypoglycemia should be prevented and can be seen in conjunction with hypothermia and alcohol use (both of which are common in drowning), and can cause direct brain damage and impaired cerebral functioning.[14,19,112]

In the past, controlled hyperventilation to a $PaCO_2$ of 30 mm Hg was advocated as a neuroprotective measure to prevent elevated ICP in the setting of trauma. It was believed that by decreasing cerebral $PaCO_2$, the resultant cerebral vasoconstriction would decrease the ICP. However, active hyperventilation is only indicated for brief periods of established elevated ICP, and metaanalyses suggest the evidence for this intervention is not as strong as for many other ICP interventions.[72,145] Hyperventilation for neuroprotection or ICP reduction in drowning patients is not recommended.

Corticosteroids for lung or cerebral injury were previously advocated for treatment of drowning. There is scant evidence to suggest a benefit to their administration. Steroids are not currently recommended because of their potential deleterious side effects (e.g., hyperglycemia, adrenal suppression, compromised immunity), which may complicate the clinical picture.[14,83,143,153]

As advancements are made in out-of-hospital resuscitation and EMS, patients increasingly survive incidents that would previously have been fatal. However, although cardiovascular functioning may be preserved, neurologic deficits may be profound and immediately call into question the benefit of "survival," or may be insidious and affect the quality of life in more subtle ways. Despite normal MRI and CT studies, longitudinal psychological monitoring may uncover deficits that are not apparent after initial imaging or presentation, because neuropsychiatric sequelae can develop many years after drowning. The importance of understanding these parameters to develop improvements for patients' future quality of life cannot be understated.[106]

HYPOTHERMIA

Hypothermia in the field can be metabolically and neurologically protective; there have been remarkable cases of survival after prolonged submersion and low body temperatures. Unless other obvious signs of death are present, patients should be rewarmed while resuscitation continues and until the body temperature reaches 30° to 35°C (86° to 95°F).[192] Resuscitation can be terminated if at that temperature there is no cardiac activity.[19] There are currently no formal guidelines beyond the recommendations of the 2002 World Congress regarding use of TH in drowning patients, so the current standard of care is rewarming. Guidelines of the American Heart Association and the European Resuscitation Council currently call for TH in comatose patients with return of spontaneous circulation after cardiac arrest.[171] Inducing mild hypothermia (32° to 34°C [90° to 93°F]) or only rewarming to that level and sustaining these temperatures for 12 to 24 hours may result in better neurologic outcomes for drowning patients with or without preceding cardiac arrest.[19,119,171] Drowning patients often arrive hypothermic at temperatures below the 32° to 34°C (90° to 93°F) range of TH. This is an area of active research, and in the absence of randomized control trials of TH in non–ventricular fibrillation cardiopulmonary arrest, the decision to initiate TH is based on expert consensus and theoretical benefit. Drowning patients with restoration of adequate spontaneous circulation who remain comatose should not be actively rewarmed to temperature values above 32° to 34°C (90° to 93°F). If the core temperature exceeds 34°C (93.2°F), hypothermia at 32° to 34°C (90° to 93°F) should be achieved as soon as possible and sustained for 12 to 24 hours. A more complete discussion of hypothermia and its management can be found in Chapters 7 and 8.

RENAL SYSTEM

Acute tubular necrosis and renal failure can result from hypoperfusion, acidosis, and hypoxemia. Hemolysis and myoglobulinuria may follow muscle trauma.[100,121] Creatine phosphokinase and urine myoglobin levels and urine output should be followed. Dialysis may be necessary in severe cases to correct volume overload from oliguria, hyperkalemia, or profound acidosis. With appropriate resuscitation with crystalloid fluids, rhabdomyolysis and acute tubular necrosis can be successfully treated.[100]

DECOMPRESSION ILLNESS

Posthypoxic encephalopathy and hypothermia may cloud the diagnosis of decompression sickness in a drowned scuba diver. If the clinical scenario suggests that decompression sickness is likely, hyperbaric oxygen therapy should be considered (see Chapter 71).

INFECTIOUS DISEASES

Infections from submersion, most often pulmonary in nature, are difficult to predict, and there is no evidence to support the use

of prophylactic antibiotics.[83,174] When infections occur, the mortality rate may be as high as 60%.[65] Bacterial, fungal, and amebic pathogens have been implicated. The primary sites of infection are the lungs and CNS, both of which create potential for systemic spread and septic shock.

Aspirated lake, pond, or canal water is more likely to be contaminated than swimming pool and hot tub water. Infections from *Pseudomonas* and *Vibrio* species occur from ocean water aspiration. Leptospirosis may result from aspiration of sewage or water contaminated by rats.[95] Vomiting with subsequent aspiration of gastric contents may lead to infection with gastrointestinal and oropharyngeal microorganisms. *Streptococcus pneumoniae* and *Staphylococcus aureus,* as well as aerobic gram-positive bacteria from the patient's oropharynx, have been found in submersion pulmonary infections, and gram-negative bacteria have been reported to be virulent pathogens following drowning.[19] *Aeromonas hydrophila* is a facultative, anaerobic, gram-negative bacteria found in both fresh water and saltwater, and has been reported to cause fatal pneumonia.[148]

Fungal infections after drowning are quite rare but can be overwhelming, even in immunocompetent individuals. They are often fatal, perhaps because of a delay in diagnosis. Infections from *Pseudallescheria boydii* and its anamorph *Scedoporium apiospermum,* found in water polluted with soil and sewage, have been reported weeks to months after drowning incidents.[19,120] CNS invasion and brain abscesses have been reported with these organisms,[116,120,144] and most reported cases have been fatal. Current treatment is with voriconazole, which has greater CNS penetration than do other antifungal medications. Other fungal infections include invasive *Aspergillus* species, resulting in invasive CNS infection subsequent to pulmonary aspiration pneumonia, particularly in warm climates with drowning in stagnant water. Severe cases should be treated with a combination of voriconazole and caspofungin.[129]

Amebic infection is a rare but potentially fatal complication of drowning. Primary amebic meningoencephalitis (PAM), an infection with a high mortality rate, is caused by *Naegleria fowleri,* a thermophilic ameba found in standing fresh water, such as lakes and ponds. One study in the southwestern United States found *Naegleria* in five out of six wells sampled and in 26.6% of groundwater samples near the location of two drowning deaths from primary amebic meningoencephalitis. The organism is prevalent particularly in warm climates.[126] It usually enters humans through the nasopharynx, with subsequent infection of the olfactory nerve and then the CNS. About 235 PAM cases worldwide have been described in the medical literature, with only a few reported cases of survival (the PAM mortality rate is approximately 95%).[55,99] Most cases have been reported in subtropical or temperate zones; underreporting in tropical regions has been suggested.[55] In the Americas, PAM has been reported in Venezuela, Brazil, Cuba, Mexico, and the United States.[1] Cases occur primarily during summer months (July to September) in southern and southwestern states, with the majority occurring in Texas and Florida.[258] Clinical features are similar to bacterial and viral meningitis and include fever, headache, neck stiffness, anorexia, vomiting, and altered mental status. Primary amebic meningoencephalitis is rapidly progressive, and death typically occurs within 3 to 7 days. The primary treatment is intravenous and intrathecal amphotericin B. Additional drugs reported to be helpful in combination with amphotericin B include rifampin, azithromycin, and the azole drugs.[39,92]

Despite the risks of infection associated with drowning, prophylactic antibiotics are not recommended.[83,174] Monitoring of temperature, tracheal aspirates (with Gram staining and culture), respiratory status, and chest radiographs should guide the clinician's use of antibiotics for established pneumonia. Mental status changes, headache, and fever without evidence of pulmonary infection should prompt consideration of CNS infection. The severity and high mortality rates of fungal and amebic infections are partly the result of the prolonged time for organisms to be grown and identified in culture in combination with the aggressiveness of the pathogens. The location and duration of submersion, water type and temperature, duration of hospital stay, and immune status of the patient help guide the infectious disease workup and direct initial antibiotic coverage until the regimen can be narrowed to organism-specific drugs.

PROGNOSIS AND TERMINATION OF RESUSCITATION

One of the most difficult tasks faced by physicians involves end-of-life decisions and counseling. These include declaration of a resuscitation as futile, and discussions regarding prognosis with families of comatose patients.

Declaring a patient dead as a result of drowning is complicated by the fact that many of the most dramatic and physiologically unexpected recoveries from cardiac arrest have been in young patients after cold-water drowning.[24,226] Because of this particular consideration with drowning and hypothermia patients, the duration of submersion, water temperature, patient core body temperature, and cardiac electrical or echocardiographic activity should be considered before declaration of death. If there is any uncertainty, resuscitation should be continued until the patient is rewarmed to 30° to 35°C (86° to 95°F).[192] Functional recovery with minimal neurologic impairment and the ability to independently perform activities of daily living occurs in approximately 17% of patients who require resuscitation in the emergency department.[194]

Similarly challenging are neurologically devastated patients who have survived emergency resuscitation. Because hypoxemia is the initial and primary insult with drowning and because the brain is one of the most sensitive organs to hypoxemia, this unfortunate scenario is not unusual. As one wilderness medicine specialist states, "It is clear that we are limited in our medical ability to 'treat' the global hypoxic-ischemic insult sustained by the victim of a significant submersion accident … preservation of patients in persistently vegetative states is a tragedy of our time."[57]

Factors known to be useful for predicting outcomes in drowning are listed in Box 69-4. In the absence of profound hypothermia, the neurologic status of a patient on admission to the emergency department is of paramount importance for predicting survival with intact neurologic function. Persons who are alert when admitted seldom die.[51,54,114,154,176] Age is included in many prognostic recommendations, but its significance has been contested. Some authorities consider an age of 3 years or younger as a favorable prognostic factor,[57,128,165] whereas others view it as unfavorable.[176,178,210] Prognostic indicators in pediatric patients include the Glasgow Coma Scale score[54] and the presence of spontaneous breathing.[62,85,114,122]

It is difficult to predict the ultimate outcome based on initial presentation, but several different classification systems help clinicians address questions regarding the potential for recovery.[45,50,114,123] Some persons have expressed concern that these systems may miss potential survivors. An ABC classification of patients based on presenting neurologic examination was established and refined by Modell, Conn, and Barker[50,151] (Table 69-3). This system categorizes patients on the basis of their condition within 1 hour of emergency department presentation and whether they were at that time A, *a*lert; B, *b*lunted in consciousness; or

BOX 69-4 Prognostic Signs in Submersion Incidents

Positive Signs
Alert on admission
Brief submersion time
On-scene basic or advanced life support
Good response to initial resuscitation measures

Negative Signs
Fixed, dilated pupils in emergency department
Submerged longer than 5 minutes
No resuscitation attempts for more than 10 minutes
Preexisting chronic disease
Arterial pH < 7.10
Coma on admission to emergency department

TABLE 69-3 Classification of Drowning Patients

Category	Description
A	Awake—fully oriented
B	Blunted—arousable; purposeful response to pain
C	Comatose—not arousable; abnormal response to pain
C_1	Flexor response to pain
C_2	Extensor response to pain
C_3	Flaccid
C_4	Arrested

C, comatose. Comatose patients were further categorized according to abnormal neurologic responses: C_1, flexor response; C_2, extensor response; C_3, flaccid; and C_4, cardiac arrest. In their series on children, Conn and coworkers[51,52] reported that all patients in category A survived and were neurologically normal, and all but one patient in category B survived. Patients in category C showed variable responses, with increasingly poor outcomes from C_1 to C_4. In another pediatric study, 37% of subjects who presented in the C_3 stage (i.e., no response to pain, fixed and dilated pupils, absence of spontaneous respiration, hypotension, and poor perfusion) had complete recovery despite predicted poor outcomes.[173] The classification system is therefore highly specific for predicting patients with good outcomes, but shows a significant amount of variability with regard to outcomes among patients with category C presentations.

Graf and colleagues[85] developed a prediction rule based on variables that predicted vegetative state or death: history of CPR, CPR longer than 25 minutes, hyperglycemia on arrival, or absence of pupillary light reflex on arrival. Those variables, plus male gender and an initial blood glucose level of more than 200 mg/dL, had the strongest associations with unfavorable outcomes in comatose children. Other studies have shown similar results, suggesting that poor outcomes are the rule rather than the exception in cases of cardiopulmonary arrest after drowning.[221]

Of special consideration with drowning patients is that most prediction rules, although good for predicting unfavorable outcomes, are not sufficiently sensitive to reliably predict favorable outcomes. As previously emphasized, there are multiple case reports of neurologically intact survival after prolonged resuscitation from drowning. It is therefore recommended that drowning patients be treated aggressively for 48 hours to determine if meaningful recovery is possible.[45,192] Withdrawing therapy from survivors who show no clinical improvement after 48 hours can then be considered.

Emergency care providers should understand that drowning incidents may be psychologically devastating for families. One study showed that 24% of parents separated after a drowning incident involving their child.[57] Siblings present during a pediatric drowning may suffer survivor's guilt. Families who have experienced a drowning incident have demonstrated posttraumatic stress syndrome, sometimes for years, and developed otherwise unexpected substance abuse patterns and sleep disturbances.[57] Counseling services should be offered to families of patients involved in a serious drowning incident or death.

DROWNING PREVENTION AND SURVIVAL

Prevention is a plausible strategy for reducing the incidence of permanent injury and death as a result of drowning. It can take the form of preimmersion interventions or postimmersion actions taken by individuals and groups to prevent complications and deaths related to drowning.

PREIMMERSION INTERVENTIONS

Drowning represents a serious public health problem worldwide, yet the majority of incidents are likely preventable.[19,141] Prevention strategies appear to be successful in avoiding fatal drownings,[81,98,138,167,216,257] and countries that have implemented prevention programs have reduced their drowning death rates.[164] Some authorities suggest that certain measures (e.g., fencing, lifeguard training, water safety training at a young age) shown to be helpful in high-income countries may not be relevant to curtailing drowning deaths in low- and middle-income countries because of geographic, social, cultural, and behavioral factors. They note a striking absence of adequate data regarding incidence rates, risk factors, intervention effectiveness, and cost-effectiveness in these settings.[108]

Preimmersion Interventions by Age

Strategies are broken down into four distinct age groups (Figure 69-12). For children who are 4 years old and younger, prevention is based on increased supervision. Children should swim only with "touch" supervision (i.e., within an arm's reach of an adult), and need to be protected from or closely supervised at all times around any bodies of water, including those as small as bathtubs, toilets, and buckets.[6,71,125,214] Children between the ages of 1 and 4 years may be enrolled in survival swimming courses. For children between the ages of 5 and 15 years, swim instruction and awareness of hazards have been shown to decrease mortality rates.[44,196] Even after swimming lessons, children of all ages

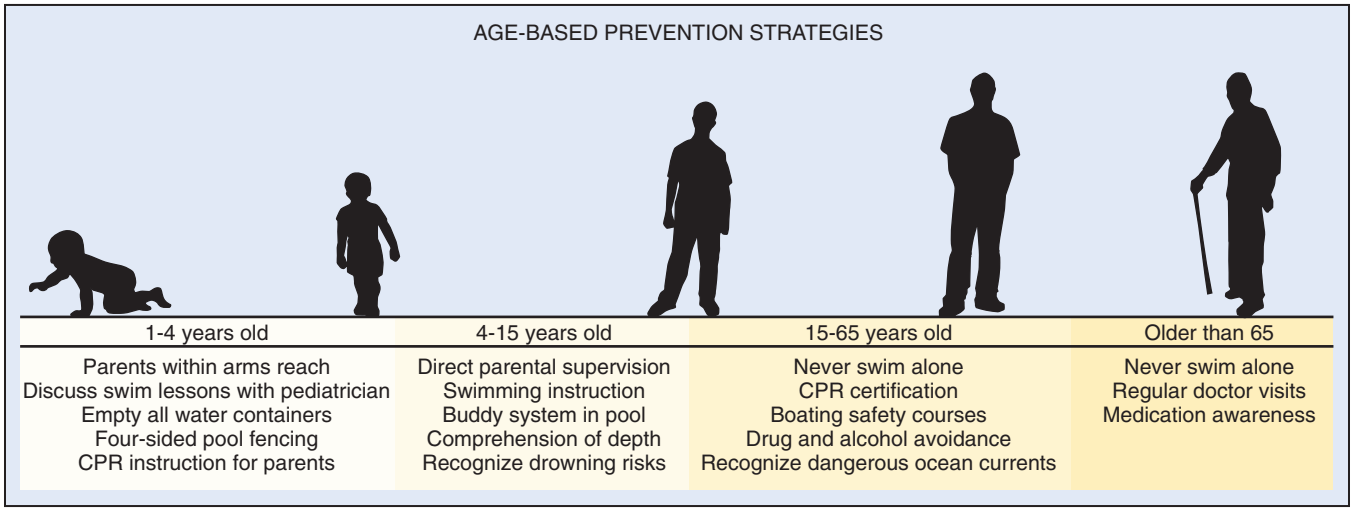

FIGURE 69-12 Age-based prevention strategies for drowning prevention. *(Courtesy Andrew Schmidt, DO, MPH.)*

BOX 69-5 Recommendations for Water Safety from the American Academy of Pediatrics

Infants and Children
Careful supervision
Empty all water containers, such as buckets and child pools
Do not allow swimming lessons to provide a false sense of security
Install four-sided fences around swimming pools
CPR instruction and 9-1-1 phone access
Use of flotation devices

Children 1-4 Years Old
Swimming instruction
Buddy swimming with supervision/lifeguard
Personal flotation devices
Knowing the depth of the water prior to entry
Recognizing drowning risks, such as skating on thin ice

Adolescents
Avoidance of alcohol and drug use
CPR instruction
Prohibiting alcohol use during boat operation
Proper training in scuba diving and water-sporting activities
Recognizing rip currents and other dangerous water situations

should wear PFDs as recommended during water sports and around aquatic environments.[6,71] For individuals who are 15 to 65 years old (especially toward the younger end), prevention strategies include risk-taking behavior reduction (especially use of alcohol and other performance-impairing drugs during aquatic activities) and increased understanding of potential hazards and mechanisms for survival and self-rescue should an incident occur. Adolescents in particular may benefit from counseling regarding swimming in remote locations or in areas not designated as approved for swimming.[71] Swimmers who are more than 65 years old may have comorbid conditions or be using medications that can affect their cognitive or physical abilities in the water, so they should be aware of potential medication effects before engaging in water activities. Water entry should be feet first any time the depth of water is unknown.[71] Box 69-5 outlines the formal recommendations for water safety from the AAP[4]; other agencies have developed similar formal guidelines.[38]

Swimming Pool Safety

Prevention involving swimming pools has been particularly successful. Legislation regarding residential pools and fences has been shown to reduce by 50% or more the incidence of drowning in communities with large numbers of swimming pools.[193,208] Inadequate enforcement of local ordinances and inadequate operation or maintenance of barrier equipment are contributing factors to childhood drowning.[161] Absence of fencing around pools increases the risk of drowning fourfold.[187] Studies have shown that pool fencing should be four-sided, with a minimum height of 1.5 m (5 feet) and a self-locking gate.[33,133] Proper gate closure should be emphasized, because a fence alone does not prevent drowning.[23] Even when locks are present, they are often nonfunctional or in an unlocked position; barrier equipment may be inadequately maintained or operated.[161,203] Ground pools should be filled so that the water level is as close as possible to the surrounding external ground, making self-extrication easier for a struggling person who reaches the side of the pool and does not have sufficient physical strength to pull up to gain exit from the water. Pool alarms and rigid pool covers may provide additional protection and should be used in addition to, not in place of, fencing.[30] Fish and garden ponds should be eliminated or fenced if small children can be near them.[133]

Supervision and Lifeguards

In almost 90% of drowning incidents, there is no supervising individual, or the individual is absent at the time of the incident.[193] This has been further validated in developing nations, such as Bangladesh, where one-third of children who die by drowning are unaccompanied. Of the remaining two-thirds, more than 40% are accompanied only by a child who is 10 years old or younger.[44,196] Young children should never be left alone, even momentarily, where there is accessible water.[5,34,98,214]

Figure 69-1 shows international drowning rates with territories sized in proportion to the absolute number of people who died from drowning over the course of 1 year. It is informative to compare results in regions that have long-standing traditions of lifeguarding, such as England, North America, and Australia, with regions that do not. Using its own surveillance data, the United States Lifesaving Association has calculated the chance that a person will drown while attending a beach protected by United States Lifesaving Association–affiliated lifeguards at 1 in 18 million (0.0000055%).[234] Other studies found that the presence of a lifeguard positively affects the outcome of drowning.[81,216] Observation by pool lifeguards is also associated with decreased rule violations.[93] Lifeguards not only perform actual drowning rescues, but are also active in swimming education and drowning prevention. CPR training and competency are not mandated for lifeguards in all international areas, so this remains a target for intervention.[193] Case fatality rates can be as high as 42% for individuals under the supervision of lifeguards, suggesting that in some regions, there is an opportunity to improve lifeguard efficacy.[193] International organizations such as the International Life Saving Federation (ilsf.org) and national affiliates such as the United States Lifesaving Association (usla.org) promote modern open-water and pool lifeguarding principles worldwide. A number of groups are involved with exporting lifeguard training and practice to countries with high drowning rates and low or nonexistent lifeguard and drowning prevention programs. These agencies (e.g., Nile Swimmers [NileSwimmers.org], International Surf Lifesaving Association [ISLASurf.org], Lifeguards Without Borders [lifeguardswithoutborders.org]) also provide oversight to private lifeguarding agencies; they offer preventive education and promote lifeguarding techniques in areas where such activities are absent. Other private-sector agencies have developed unique courses and curricula for water safety and supervision. One example is Landmark Learning, a wilderness medicine school in the southeastern United States that developed a Wilderness Starguard program in cooperation with the Starfish Aquatics Institute and the American Safety and Health Institute (landmarklearning.edu/courses/starfish-aquatics-institute/wilderness-starguard). This certification course trains students to serve as lifeguards and water safety personnel in wilderness settings.

Training for Out-of-Hospital Personnel

On-scene resuscitation is associated with improved outcomes. Persons designated to attend the scene of drowning incidents should have specialized training and triage ability to deliver the patient to the appropriate medical center.[98] Lifeguard agencies should conduct routine interagency training sessions with EMS workers, firefighters, and other personnel who may respond to drowning or rescue incidents to discuss management and extrication strategies. Specific training is available for open-water and swift-water rescuers, and personnel should seek certification appropriate to the aquatic environments in which they participate. In the United States, the *National Fire Protection Association 1670 Standard on Operations and Training for Technical Rescue*[166] regulates the components of swift-water rescue training, including operational and technical training.

Swimming Lessons and Boating Instruction

One possible intervention to prevent drowning is to promote swimming lessons. Although this seems intuitively sensible, some studies suggest there is no solid evidence that swimming lessons are an effective public health measure to reduce drowning.[216] Potential explanations for this finding are that swimming ability or lessons may lead to overconfidence in a water setting, swimming in hazardous situations, increased exposure to water, or reduced parental vigilance.[6,10,12,160,215] Others debate the validity of positions that argue against swimming lessons.[80,142] More recent studies suggest that lessons may reduce the incidence of drowning in young children,[257] and emerging data from developing nations show that swim instruction may have a lifelong protective effect and decrease mortality rates.[44] Swimming instruction may improve survival ability in water, but should not be

seen as a guarantee against drowning. Even in studies showing a protective benefit from swimming lessons, many persons in the drowning cohort were relatively skilled swimmers.[31]

The optimal time to begin swimming education remains controversial. Prehospital trauma life support (an expert consensus certification course) workers and other authors recommend swimming education for children who are younger than 3 years old.[165,178] Commercial training programs, such as Child Drowning Prevention and Infant Swimming Resources, offer specific floating training to children in this age group.[42,113] Multiple studies show that swimming skills can be taught and retained at a young age.[10,66,179] Two case-control studies suggest that participation in formal swimming education prevents 40% to 88% of drowning incidents among children who are 1 to 4 years old.[31,257] Based on recent research showing that drowning victims between the ages of 1 and 4 years were less likely to have had formal swimming instruction,[31,257] the AAP updated its recommendations to allow formal survival swimming lessons as early as 1 year of age.[5] Unless there is a cause for exception, all children who are 4 years of age or older should be enrolled in formal swim instruction. No level of swim training or instruction should replace direct, uninterrupted, and competent supervision of children in and around the aquatic environment. Some suggest that educational efforts to explain high-risk situations and avoidance activities may be more beneficial than actual swimming instruction.[6,98,216] Formal training in boating skills and self-rescue can prevent drowning and help boaters and paddlers manage the consequences if they enter the water.

Personal Flotation Devices

All individuals involved in water sports should be familiar with PFDs and particular devices that may be recommended or required for their specific activities. In 2013, 84% of persons who died by drowning aboard boats were not wearing PFDs. Air-filled swimming aids (e.g., water wings, floaties) should not replace PFDs.[30] The AAP further states that water wings and floaties are detrimental to safety and can allow a child to reach water beyond his or her depth and then easily fall off or be removed, resulting in drowning. Drowning survival, prevention, and self-rescue hinge on correct PFD selection. PFDs are categorized based on the amount of buoyancy they provide and type of activity in which they will be used (see Figure 69-2). As of September 22, 2014, the U.S. Coast Guard issued a ruling that it would abandon the "type" classification as presented in Figure 69-2. No new terminology has yet been approved or disseminated, and many manufacturers still use the type classification.[104]

Preventive Equipment

By selecting safety equipment appropriate to the conditions, many hazards can be controlled by individuals in the water setting. This includes activity-specific helmets, wetsuits or thermal protection, PFDs, and survival suits. Newer-generation whitewater helmets feature multilayer impact shells with specially designed retention systems to keep the helmets in place during water sports. Wetsuits or drysuits can be lifesaving in cold-water environments by preventing cognitive, motor, and other adverse physiologic consequences of hypothermia. Whistles that are lightweight, operate without a pea, and can be carried in pockets or attached to survival gear greatly increase the chance of a wet individual being located if he or she is lost. Beacon locator systems and other boating safety technology can reduce open-water boating-related fatal drownings by allowing rescuers to locate persons in a timely manner. Using boats that are correctly configured and equipped for the chosen environment is critical for all paddling sports.

Education About Alcohol and Drugs

Despite similar pathophysiology and injury patterns between motorized water- and land-based vehicular injuries, there is greater legal and societal acceptance of alcohol use during motorized water sports. There is also widespread social acceptance of alcohol ingestion while recreating in and around water, and a plethora of advertisements promote its appeal. Strategies similar to those used to curtail alcohol drinking while driving may have a similar effect on alcohol drinking while boating.[134] In addition to impaired cognition that may result in a collision while operating a vessel, impairment from drugs or alcohol can significantly hinder an individual's ability to self-rescue. Decreased situational awareness and/or physical stamina required for self-rescue and peer rescue in an aquatic environment are risky and should be discouraged.[33,98,192,216] This is an area amenable to law enforcement.

POSTIMMERSION ACTIONS

Individuals venturing into remote settings that include water exposure should be mentally and physically trained and prepared to act if an accident occurs. Preparation, education, and proper equipment are crucial factors for the prevention of and response to a drowning event. Ideally, all activities involving water exposure should include safety personnel either in or beside the water who have predetermined roles if a victim gets into trouble. This is not necessarily a formal lifeguard; many drowning rescues have been accomplished as a result of the astute situational awareness of bystanders. Rescuer safety should always be considered; there are numerous case reports of well-intentioned rescuers dying while attempting to perform rescues. One Australian study examined cases over a 6-year period where adults attempting to rescue children died by drowning. In 93% of cases when the adult died, the child they were attempting to rescue survived.[68] Having safety personnel in place can be as simple as having one team member downstream on the shore before everyone else runs a rapid, or as complex as rigging a safety line before making a stream crossing. It is critical that rescuers never exceed their level of training or abilities.

A key educational intervention is the principle of "reach, throw, row, go" (Figure 69-13). First, attempt to reach the victim with a rope, branch, oar, paddle, or other object while safely remaining on shore or in a boat. Second, throw any floating object to the victim; ideally, this is a buoy or PFD; improvised items include something like a soccer ball, empty cooler, or dry bag full of air. Third, row or paddle to the victim in a boat, kayak, canoe, surfboard, or any other large watercraft capable of safely simultaneously floating both the rescuer and victim. The final option, "go," should be reserved as a last resort for professional rescuers. An uncertified would-be rescuer has a high risk of becoming a second drowning victim, either by succumbing to the same environmental dangers as the original victim or by being dragged underwater by the panicked victim. If it becomes necessary for an untrained rescuer to enter the water to effect a rescue, it is critically important to never physically engage an actively drowning person. The rescuer should swim toward the victim while wearing a PFD and carrying an additional PFD for the victim. Stop a safe distance (i.e., greater than two arms' lengths) and throw or push the second PFD to the victim, avoiding direct contact. When the victim is wearing the given PFD and no longer is in a state of panic, he or she can be safely assisted to shore or a boat.

The person who is suddenly thrown into water must remain calm and make a plan based on the situation. For example, if a person wearing a backpack falls into moving water while crossing a stream, the backpack must be removed before the person attempts to swim, to avoid being pulled underwater by the pack. If a person falls into a calm lake or ocean, the person should attempt to float on the back and discard any items that might be weighing the person down. For rip currents (i.e., surface ocean currents that form as the result of channeled water returning seaward), a person should relax and swim parallel to shore instead of struggling against the current. All of these situations can be compounded by cold-water or prolonged immersion. The person must stay relaxed, remember to turn the head to breathe, and remain in the HELP position (Figures 69-14 and 69-15) while not swimming. Rhythmically turning the head to breathe as the face-down body bobs to the surface helps to promote controlled breathing, especially in open water or surf. Achieving adequate respiration with this maneuver becomes very difficult in rough water, currents, and tides or if there is concurrent injury; however, the chances for successful survival increase with practice.

RECOMMENDED RESCUE TECHNIQUES			
			
Reach	Throw	Row	Go
Lowest risk to rescuer	→ → → → → → → →		Highest risk to rescuer
Weak/injured victim, unable to swim, close to shore	Weak/injured victim, unable to swim, far from shore	Long distance, victims unable to grasp rope	Should be reserved for trained rescuers, swim floatable object to victim, never contact active victim
Branches, poles, paddles, clothing	Rope bags, ring buoys, coolers, soccer ball, PFD, air-filled dry bag	Kayak, canoe, surfboard	PFD, rescue buoy, ring buoy, cooler, soccer ball, air-filled dry bag

FIGURE 69-13 Reach, throw, row, go: recommended rescue techniques in submersion incidents. *(Courtesy Andrew Schmidt, DO, MPH.)*

FIGURE 69-14 Heat escape lessening posture (HELP). *(Courtesy Alan Steinman, MD.)*

FIGURE 69-15 Huddle technique. *(Courtesy Alan Steinman, MD.)*

If rescue is not immediate, a decision must be made by the victim as to whether swimming to safety or staying in place is the best course of action. Trying to swim a great distance in hope of rescue increases exposure of the body's surface area to cold water and thus heat loss by convection. It also increases loss of energy that might be better spent keeping the head above the water surface for a longer time. Decisions such as this are made easier by preventive planning before and during activities in and around water. Individuals anticipating recreation in a water environment should have a premeditated plan that includes appropriate equipment, as well as practiced swimming, rescue, lifesaving, and resuscitation skills. Boats, canoes, kayaks, and rafts should meet accepted standards and be kept in good condition. All boats and vessels should be equipped with U.S. Coast Guard–approved PFDs or other flotation devices in sufficient numbers to equip all participants. Courses on boating safety, rescue, and navigation are offered by the U.S. Coast Guard in most waterways and by numerous private companies for various boating sports.

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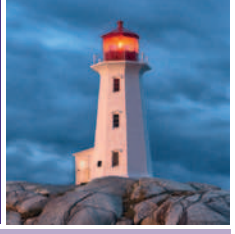

CHAPTER 70

Safety and Survival at Sea

CHARLES G. HAWLEY AND MICHAEL E. JACOBS

> It is difficult for us to grasp the idea that parts of our planet remain in an almost primordial state of wildness and isolation. There are only a few places left on Earth where merely getting across them is an achievement: Antarctica…the Sahara…the Southern Ocean…the wilderness of ice or sand or water or terrible places where nature retains power over humans to terrify and diminish. *Derek Lundy, Godforsaken Sea*

The greatest wilderness on Earth is the sea. Water covers two-thirds of the planet and, except for the sun, has the greatest influence on global weather patterns. Although it may take hours or days to succumb to exposure in most environments, death at sea can happen in under a minute. Compared with desert heat, high-altitude hypoxia, and polar subzero temperatures, water is the most hostile and life-threatening natural environment for inadequately equipped survivors.

The 1979 Fastnet Race distinguished itself as the worst disaster in the long history of ocean yachting. A surprise storm crossed the Irish Sea between southwest England and southern Ireland, and exploded without warning in the midst of the Fastnet racing fleet. Suddenly, 2700 men and women in 303 ocean-sailing yachts unwittingly became participants in hundreds of incidents of survival at sea. Winds of force 10 (55 knots) with much stronger gusts, and seas as high as 15 m (50 feet) knocked down 48% of the fleet until their masts paralleled the water; 33% of the fleet experienced knockdowns substantially beyond horizontal, including total inversions and full 360-degree rolls for at least 26 yachts. Despite a massive response of rescue personnel and equipment, 24 yachts were abandoned and 5 vessels sank. Fifteen sailors died and 136 were rescued from disabled yachts or the water. The official Fastnet Race Inquiry noted, "The common link among all 15 deaths was the violence of the sea, an unremitting danger faced by all who sail." It concluded, "The sea showed that it can be a deadly enemy and that those who go to sea for pleasure must do so in the full knowledge that they may encounter dangers of the highest order."[5] The Fastnet storm had two positive results. First, boats, safety gear, and safety procedures were improved dramatically. Second, sailors began to talk realistically about the risks of sailing and came to regard safety as a necessary component of their craft. The first public Safety at Sea Seminars were held in Annapolis and New York after the Fastnet shock. Since then, hundreds of safety and seamanship seminars have been held across the United States and other sailing locales (Figure 70-1).

Twenty years later, on the edge of the Southern Ocean, the Sydney to Hobart race between the southeast coast of Australia and the island of Tasmania became a terrifying ordeal for 115 yachts. Within a day of leaving harbor, an explosive low-pressure cell, a "southerly buster," formed over the fleet as it entered Bass Strait. In its aftermath, 6 men died, 55 men were rescued, and 12 boats either sank or were abandoned. One hundred participants were seriously injured and five drowned. Fractured ribs, lacerations, and head trauma were the most common injuries. The 330-page investigative report recommended strict guidelines for improved safety gear, life rafts, and communication equipment. In 2012, five sailors perished while participating in the Full Crew Farallones Race out of San Francisco. An independent review panel of experts under the auspices of the U.S. Sailing Association (often known as U.S. Sailing) subsequently issued a full and detailed report of this disaster. This report can be read in its entirety, together with other safety reports and reviews of sailing accidents, on the U.S. Sailing website.[11]

HOW DO PEOPLE DIE IN RECREATIONAL BOATING ACCIDENTS?

For ocean racing sailors and voyaging seafarers, most emergencies and accidents occur during extreme weather conditions created by violent ocean storms (Figure 70-2). In contrast, most recreational boating accidents in the United States occur in fair weather, with flat to 30-cm (1-foot) seas, light (0- to 10-km/hr [0- to 6-mph]) winds, and good daytime visibility. Most of these accidents happen close to home, on inland lakes, ponds, rivers, and coastal bays, which are the most common areas used for pleasure boating.

The most recent U.S. recreational boating statistics available from the Coast Guard from their annual report "Recreational Boating Statistics 2014"[8] indicate there were 610 boating fatalities (14% decrease from 2012 to 2013; 9% increase from 2013 to 2014). This Boating Accident Reporting Database (BARD) report is a compilation of accident data from individual states and trust territories, and it recounts the fascinating, if macabre, stories of how people die while boating. Capsizing and falls overboard from open motorboats, rowboats, canoes, and kayaks account for more than half of fatalities. Vessels involved in accidents with the greatest number of casualties are open motorboats (47%), personal watercraft (17%), cabin motorboats (15%), canoes (13%), and kayaks (10%). Eight of every 10 boaters who drown were in vessels less than 21 feet in length. The BARD reports an average of 5000 recreational boating accidents annually. Sixty percent of all accidents involve operator and passenger controllable factors, and 25% involve boat or environmental factors. Operator inattention, improper lookout, operator inexperience, excessive speed, and machinery failure rank as the top five primary contributing factors in accidents. Alcohol use is the leading known contributing factor in fatal boating accidents; where the primary cause is known, alcohol use is the leading factor in 21% of deaths.

Eighty percent of deaths occurred on boats where the operator received no boating safety instruction. However, this statistic is misleading, because the overall number of boaters who have received boating safety training is unknown. Collision with another vessel is the most common type of accident, followed by flooding/swamping, collision with a fixed object, grounding, and water-skier mishaps, accounting for 30% of fatalities. Open motorboats less than 8 m (26 feet) in length and personal watercraft make up two-thirds of the watercraft involved in collisions. Drowning is the cause of death in 63% (178 of 282) of open powerboat fatalities, but it is the cause of death in only 35% (12 of 34) of personal watercraft fatalities; this discrepancy suggests

FIGURE 70-1 U.S. Sailing Safety at Sea courses offered around the country are a great way to be exposed to expert opinions and hands-on training with safety gear.

FIGURE 70-3 Protection from sun, wind, and water, eye protection, and a harness and inflatable life jacket are just some of the "kit" for offshore sailors.

that use of life jackets (sometimes called personal flotation devices [PFDs]) on personal watercraft has a significant impact on mortality rates.

These statistics indicate that one of the greatest threats to a boater's safety in "home waters" is an inexperienced, inattentive operator motoring at excessive speed, unaware that another vessel or individual may be nearby. Constant vigilance is necessary in order to take evasive action and avoid collision. Sixty-nine percent of all fatalities in accidents on recreational boats described in the BARD report are from drowning, and 84% of victims were not wearing a life jacket. The full report may be reviewed at the website of the U.S. Coast Guard's Boating Safety Resource Center, along with the annual JSI study of life jacket wear rates.[9]

Fractures, lacerations, contusions, head injuries, and low back sprains are the most frequent injuries in boating. Burns, hypothermia, amputations, carbon monoxide poisoning, and dislocations are among the next most common problems. Open motorboats and personal watercraft are the vessels most frequently involved in passenger injuries from trauma, while canoes and rowboats account for 33% of boaters affected by hypothermia. Explosions from propane stoves and carbon monoxide poisoning from bad exhaust systems accounted for only two fatalities in 2014, or less than 1% of all deaths.

FIGURE 70-2 Sailors need to be able to "weather the storm they cannot avoid"; breaking entrances may force them to wait out the gale until a channel can be safely navigated.

PERSONAL SAFETY GEAR

LIFE JACKETS

Annual Coast Guard recreational boating fatality statistics reemphasize that boaters should wear life jackets. In 2014, for example, 418 (69%) of 610 recreational boating fatalities where the cause of death is known were due to drowning. Of drowning victims where the life jacket status is known, 84% were not wearing a life jacket.

The vast majority of these drowning victims were in boats under 7 m (21 feet), including open motorboats (43%), canoes (17%), and kayaks (12%). Each annual Coast Guard report estimates that at least 85% to 90% of small vessel drowning deaths could have been prevented if a life jacket had been worn. The most common causes of drowning include capsizing of a vessel, falls overboard, flooding/swamping, and collision with a fixed object. Life jackets help prevent drowning in water at any temperature and are crucial in combating the lethal effects of cold water. When boating in cold waters (< 70°F [21°C]), the cold shock response (see Chapter 8) is the primary cause of drowning. A high-buoyancy life jacket (Figures 70-3 and 70-4) is essential to survival; it keeps the head above water and maximizes the airway freeboard (distance from the water to the mouth), which prevents aspiration immediately following sudden immersion. Some states require everyone in boats 21 feet long or less to wear a life jacket between November 1 and May 1. Few boaters routinely wear life jackets, and delay in donning them until facing storm conditions, losing protection during both collisions and falls overboard. During the Dauphin Island Race in Mobile Bay, Louisiana, a fleet of 118 sailboats was overtaken by a fast-moving (but widely predicted) line of squalls. Videos and testimony from the sailors in that race indicate that many delayed putting on life jackets despite the ominous front that approached the fleet. Six sailors drowned in that race (some of whom were wearing low-buoyancy life jackets). The 2014 National Life Jacket Wear Rate Observational Study reported that from 2006 to 2014, usage rates for adults in open motorboats varied from 4.5% to 5.8%, with a 2013 figure of 5.8%.

Nonswimmers, children, and inexperienced crew should wear life jackets at all times whenever on deck or in an open boat. Everyone on deck should wear a life jacket in heavy weather, at night, when visibility is reduced, when the boat is traveling quickly, or while traversing cold waters. In warm waters, experienced boaters who are strong swimmers implicitly acknowledge a degree of risk by not wearing a life jacket.

When boating in cold weather, a foam-lined float coat can be worn either with or in lieu of a life jacket, although the combination of float coat and life jacket is demonstrably safer. In addition to providing 7 kg (15.5 lb) or more of flotation, the coat offers

FIGURE 70-4 Modern life jacket-harness combinations combine some features of climbing gear with personal flotation. While not approved by the U.S. Coast Guard, this model from Spinlock meets ISO standards for life jackets and is approved in much of the rest of the world.

excellent protection against hypothermia and cushions the ribs and thorax from injury during a fall on deck. Most models are approved as type III* flotation devices (for calm, inland waters, or where fast rescue is likely). For additional flotation or hypothermia protection, many float coats can be combined with bib overalls. Use of these gear items in tandem has the added benefit of extended survival time in very cold water.

The primary reasons cited by veteran sailors for not wearing life jackets include discomfort and inconvenience. Virtually all inherently buoyant (foam or kapok-filled) type I offshore life jackets and type II near-shore life buoyant vests are bulky, uncomfortable, awkward to wear, too warm in summer, and limit mobility. The common type III flotation aid and many type V vests are comfortable and wearable, but have significant limitations, including low total buoyancy (generally 70 N or 15.5 lbf), poor reserve buoyancy (as the wearer is immersed, buoyancy does not increase, because the life jacket is already immersed), low freeboard, inability to turn the unconscious victim face up (righting ability), and inability to support the head (and thus maintain an airway). These type III and V life vests are suitable only for calm water and should be worn in situations where a quick rescue is assured. Life jackets should be selected according to comfort and practicality in order to maximize compliance.

The Inflatable Advantage

Inflatable vests have a low profile and lightweight design that allow them to lie flush against the body so they do not restrict movement, resulting in ease of wear and superb flotation. For offshore sailors, vests provide 150 N (33.7 lbf) of buoyancy, compared with 100 N (22.5 lbf) in foam type I and 70 N (15.5 lbf) in foam types II and III. Incremental buoyancy enables a person to float high in the water, easing inspiration and expiration, and reduces risk of seawater aspiration in rough seas (see Figures 70-3 and 70-4). Avoiding mouth immersion is vital to prevent drowning. When possible, victims should face downwind and away from oncoming waves to avoid splash-over of water into the airway, although a natural tendency is to assume a face-into-the-waves attitude. By keeping the head, neck, and chest higher out of the water, it is easier to adopt the heat escape lessening position (HELP) (see Figure 69-14). With buoyancy high on the chest, there is also superior righting ability and head support. Inflatables with 150 N of buoyancy can be purchased with an integral safety harness, and all vests can be equipped with strobe or LED safety lights, whistles, crew overboard (COB) beacons, and other attention-attracting gear (see discussions of safety harnesses, as well as other equipment, below) (see Figure 70-3).

The U.S. Coast Guard has approved a wide variety of inflatable life jackets with varying buoyancies (70, 100, 150, and 275 N [16, 24, 35, and 60 lbf]). Water-activated inflatable vests should be worn at all times by nonswimmers because they might panic after falling overboard and not deploy their manual inflation ripcord; children must be older than 16 years and weigh more than 36 kg (80 lb) to have these types of vests count in a vessel's inventory. Even strong swimmers should consider wearing a water-activated model. Head injury from a surprise fall or an inadvertent head injury from the boom might render a person unconscious or stunned and leave the person face down in the sea with a deflated manual inflation vest. More importantly, any sudden, unexpected immersion can be very disorienting, especially when compounded by a cold shock response and reduced swimming ability due to clothing, footwear, and gear. When interviewed, survivors of boat accidents involving quick, unanticipated immersion consistently report that they might not have been able to find the "ripcord" and activate it, because of the disorientation that occurs with cold-water immersion. Wearing an inflatable vest that automatically responds to immersion negates having to locate the "jerk to inflate" lanyard.

Newer models of inflatable vests have 1F (water-activated) and 3F (manually activated) inflators that have a single point indicator to show if the vest is armed with an unused carbon dioxide (CO_2) cylinder, so-called cylinder seal indication. The indicator allows the user to determine whether the life jacket is properly armed and ready for use. Newer inflators will not deploy unless the wearer is immersed in water, and are highly resistant to incidental water contact, such as sea spray or rain. Older models suffered from premature and undesired inflation. All automatic vests have a manual backup ripcord for inflation, plus a backup oral inflation tube that can also be used to deflate the bladder routinely and in an emergency.

Like all mechanical systems on-board ship, inflatable vests require regular inspection and maintenance according to the manufacturer's instructions. Prior to each use, the CO_2 cylinder must be in place and unused. With newer 1F and 3F models, this can be verified by looking for the green indicator. Older models may require investigation under the protective shroud and unscrewing of the cylinder. Users should note whether the life jacket has a water-activated inflator. Every season, the CO_2 cylinder should be removed and the vest orally inflated as much

*In the fall of 2014, the U.S. Coast Guard announced that life jackets would no longer be classified by type as part of the process of harmonization with ISO (international) life jacket standards. A new North American standard, UL 12402, was in the process of being approved as this book went to print. This standard is based on the ISO 12402 standard and will use different levels to denote the lifesaving potential of the devices. Proposed levels are roughly equivalent to the buoyancy of the life jacket in Newtons, and will be level 50, 70, 100, 150, or 275. The new standard includes dramatically different labels that show the conditions under which the life jackets are to be used and, in some cases, not used, as well as the righting ability of the device and sizing recommendations. As of January 2016, two sections of UL 12402 were approved: -5 and -9. These cover the Level 50 and Level 70 devices (-5), and the means to test life jackets (-9). Life jackets made to this standard will be legal in both the United States and Canada, which will preclude the need to have separate products for each country. Level 150 (-3) and Level 100 (-4) portions of the standard were being worked on by the Standards Technical Panel as of the date of this writing (summer 2016). Boaters are advised to rely on the label instructions found inside every life jacket to assess the conditions for appropriate use of a given product.

as possible and left to stand for at least several hours to assess for leaks. If the vest becomes soft, it should be established that the oral inflation tube valve is seated. If the oral inflation tube is not the leak source, the vest should be destroyed or returned to the manufacturer for further analysis. The water-soluble bobbins should be inspected and replaced as scheduled (generally every 2 years). One must know how to rearm, deflate, and repack any inflatable vest according to instructions. A life jacket should be maintained as though one's life depends on it, because it certainly does.

Boaters who resist wearing any type of vest may tolerate an inflatable vest packed in a compact belt pack. This vest is carried in a belt pack around the waist, and is the least cumbersome of any flotation device. After inflation in water, the horseshoe-shaped device must be pulled over the head and secured with straps to the chest (similar to a regular inflatable life vest) while the swimmer works to stay afloat; it offers 100 N to 150 N (22 to 35 lbf) of buoyancy. This device is not suitable for nonswimmers or children. It is becoming more common due to the increasing popularity of stand-up paddleboards because of lack of interference when paddling.

An additional option is one of the increasingly lightweight, stole-type vests that fit close to the chest but do not look like the original suspenders-appearing vests (see Figure 70-4). They are more like athletic equipment, and are available as water-activated models that can be converted to manual-only operation when desired.

Testing a Life Jacket

One should test and wear any new vest in a pond or pool and practice the HELP position. The wearer should float in a slightly reclining position with 3 to 4 inches of freeboard. Life jackets should be close fitting with small arm holes, or at least grip the torso so that they stay in place. Life jackets that ride up on the wearer's chest allow the wearer to sink lower in the water, reducing the airway freeboard.

Children require a properly fitted model with leg straps to prevent the vest from sliding up over the arms and head (this is also effective for adults). Common size ranges for children's life jackets are 0 to 30 lb, 30 to 50 lb, and 50 to 90 lb. The best life jackets for children under 23 kg (50 lb) should have a bifold head support to right a face-down child and keep the child face-up in water. An additional option is to have children wear safety harnesses to keep them on deck. It is dangerous to buy a life jacket that is too large for a child (in anticipation of future growth); incorrect sizing of a life jacket compromises its usefulness and may have tragic consequences. The PFD should fit snugly with zippers closed and straps tightened. Test the fit by having the child raise his or her arms over the head while an adult lifts the PFD by the shoulder straps. If the PFD slips upward or slides up to touch the child's nose, further adjustment is necessary. Adults can also perform this test by jumping into the water wearing the PFD to see if it slips upward, or have someone pull forcefully on the shoulder straps from above. Falling overboard with the PFD rising up and covering the face and head is an alarming and life-threatening situation.

For young sailors, there are many styles, rated buoyancies, designs, and constructions. These were reviewed in the June 2013 issue of *Practical Sailor;* the following month's issue reviewed some newer designs for "active" adult sailors (racers) and kayakers.

IMMERSION (SURVIVAL) SUITS

Immersion (survival) suits are the ultimate protection from hypothermia and drowning. An immersion suit combines properties of a life raft, life jacket, and dry suit. Most models include a watertight full-length zipper, watertight hood, face seal for wind and water protection, detachable mitts, neoprene wrist seals, integral boots, inflatable head pillow for optimum flotation angle, integrated lifting harness, water-activated safety light (strobe or LED), whistle, and buddy line. Their general bulkiness (one size fits a 50- to 136-kg [110- to 300-lb] person) and built-in gloves make them impractical for continued wear while actively working aboard ship. The Coast Guard now requires personnel onboard their vessels to wear a dry suit (not an immersion suit) when the ocean water temperature is less than 50°F (10°C), and a less bulky antiexposure suit with insulating underwear and clothing when temperatures are between 50° and 59°F (10° and 15°C). A type III life jacket is still necessary to provide adequate flotation if the head pillow and flotation are not integral to the suit.

One potential disadvantage of survival suits (as with high-buoyancy life jackets) is that their buoyancy may impede escape from an overturned craft. The person may become trapped in the cabin, under the cockpit, or under the trampoline of a multihull. "When the trampoline is on top of you, buoyancy is your enemy," said one multihull sailor, who barely survived after capsizing his vessel while wearing his survival suit. One should always don the suit topside (never below decks or in the wheelhouse) and move away quickly from a rolling, unstable craft. In high latitudes (over 40 degrees), many experts recommend that a survival suit be carried for each person onboard, although the bulk and expense make this somewhat impractical for recreational boaters. Crew should read instructions for suit use because there is a specific technique and sequence for donning the suit, pulling the hood over the head, zipping, and closing the face flap. In practice, the suit should be closed and made watertight in less than 60 seconds. Suits should be inspected regularly for tears or deterioration, and zippers lubricated with Zipper-Ease or other lubricant. A partially closed immersion suit serves only to keep the victim afloat and alive until the victim dies of the cold shock response, immersion hypothermia, or drowning; there are cases of professional fishermen who died from these causes while wearing immersion suits when forced to abandon ship in cold waters.

Water-activated inflatable life vests also may lead to entrapment. In the 2012 Chicago-Mackinac race, two sailors drowned when their sailboat capsized in a powerful squall; they were unable to free themselves from underwater entanglement (see ussailing.org/racing/offshore-big-boats/big-boat-safety-at-sea/safety-incident-reports/ for incident reports regarding recent sailboat accidents).[1] In the incident report, survivors describe how inflated life jackets and attached tethers impeded their escape. Required skills for any sailor include safety equipment familiarity, PFD deflation and air bleeding, use of a quick-release clip on a tether, PFD, and underwater knife use and deployment.

If one has to enter cold water voluntarily (e.g., to free a fouled propeller) without protection of a specialized suit, one must acclimate gradually to frigid water in anticipation of the cold shock response. Upon entering, one must control one's breathing while wearing a safety harness with the tether held by an alert crew member. Once breathing is controlled, the person may proceed.

SAFETY HARNESSES

Safety harnesses (see Figure 70-4) are made from 50-cm (2-inch) nylon webbing or are integrated into inflatable life jackets, with a breaking strength exceeding 1450 kg (3300 lb), and attach the wearer to the vessel. Harnesses are worn when sailing in rough weather, at night, when on deck alone or out of sight of crew, or when both hands are occupied. In heavy weather, a harness should be worn at all times, even in the cockpit. A harness should fit snugly around the chest, 2 inches below the armpits. Tethers connect the harness to through-bolted deck fittings or dedicated jacklines, and are made from uncoated stainless steel wire, HMPE rope, or webbing running fore and aft to a through-bolted pad eye or cleat. Cockpit pad eyes or jacklines should be established within reach of the companionway, so crew can clip on before entering the cockpit. Many newly-awakened sailors, groggy with sleep and unaware of sea conditions, have been lost overboard transitioning from cabin to cockpit during a change of watch. Jacklines should be continuous, allowing crew to roam without having to unclip. Low-stretch webbing is preferable to wire and rope because webbing tends to lie flat on deck and does not roll underfoot. Wire jacklines should be inspected for broken strands, and webbing and rope for ultraviolet (UV) damage and weakening. Recently manufactured tethers have colored threads that will break if the tether has been overtensioned and stretched (also called an overload indicator or overstress indicator). Tethers should be no more than 2 m (6.6 feet) long with an elastic core

to keep them from dragging underfoot; a second tether 1 m (3.3 feet) long helps triangulate support and ensures continuous contact with the ship while changing positions. Tethers are best used in confined places, such as the cockpit and at the helm. Ideally, the tether should be attached to the boat such that it will not allow the wearer to be dragged in the water alongside or behind the boat. However, this may not be possible; sailors have drowned while being dragged alongside the vessel by their harness and tethers. The chest shackle should be a quick-release snap shackle that can be released under load if the wearer is trapped under a capsized boat or feels he or she might drown due to being dragged. The ship's end should have a locking snap hook, not one that will accidentally self-release from a pad eye. It should be possible to accomplish the clip on/off process with one hand, and never require a sailor to use both hands to connect or loosen the hook at the end of the harness tether. A double-action carabiner only opens when squeezed "between one hand" (between fingers and palm), and eliminates the possibility of accidental opening. Another option is the double-action locking safety hook, which can be opened with one hand.

The harness should be donned before leaving the cabin, and tether secured before leaving the companionway. While underway, crew should hook the tether onto the windward (uphill, upwind) jackline whenever possible; a crew person is more likely to fall to leeward, and the shorter tether length will keep a person on deck instead of dragging the person through the water. Women should not adjust the chest strap below their bust line, because breast injury may occur from upward force placed on the harness under sudden tension; there are female-specific harness designs. Inflatable life jackets are available with an integral safety harness; this convenient combination may be the most important piece of personal safety gear at sea. A harness keeps the crewmember aboard, prevents separation from the vessel if and when a crewmember falls overboard, and can guide escape from an overturned craft, especially when someone is disoriented.

CREW OVERBOARD

REMAINING ABOARD

Falling overboard (most often from collision) with subsequent drowning is the most common cause of a marine fatality in recreational boating and commercial shipping. In stormy weather, the safest location is inside the cabin. Following these rules can prevent virtually all crew-overboard (COB) incidents:

- Remain sober, especially if you expect to go on deck for any reason.
- Wear nonskid footwear that grips well on a rolling and wet deck, and have nonskid paint or pads in critical work areas.
- Walk in a crouched position with a low center of gravity and wide-based stance when the boat is rolling, heeling, or pitching.
- If the boat's motion is too violent for a person to stand, then crawl or slide along the deck.
- Use a safety harness and tether as a "third hand," secured to a strong attachment point.
- Use a safety harness whenever going aloft in the rigging or climbing any superstructure.
- Avoid leaning overboard with all your weight on a lifeline or stanchion, and use a strong adjustable boat hook to pick up a mooring and dock lines or extend to a COB.
- Know the location of secure handholds and grab rails so you can find them at night.
- Know safe routes to avoid tripping on deck hardware, vents, and hatches, especially at night; use spreader lights if necessary to illuminate the deck. Be aware, the helmsperson will lose night vision temporarily (Figure 70-5).
- Do not urinate from the afterdeck in rough weather unless you are kneeling and attached with a safety harness. In really rough weather, consider urinating into cockpit scuppers.
- Wear a safety harness whenever seasick; vomit into a bucket rather than leaning overboard.

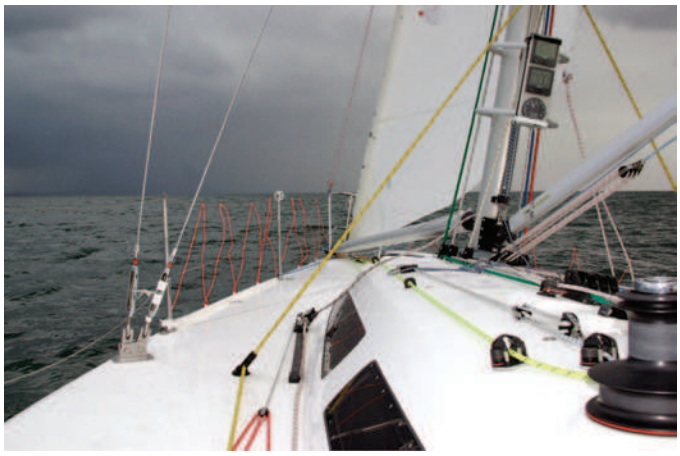

FIGURE 70-5 Clean side decks on this ocean racer provide fewer trip hazards, but not a great deal to hold on to. Toe rails on modern boats may only extend aft from the stem to abeam of the mast. Using jacklines would make going to the foredeck dramatically safer.

- In heavy weather, sleep in the harness and be ready to attach the tether to a cockpit pad eye before coming on deck.
- Check the integrity of pulpits, stern rails, handrails, stanchions, lifelines, and jacklines. Look for cracks on welds, corrosion of fittings at terminals, and telltale rust streaks on plastic coating. Current standards for lifelines do not allow vinyl coating, because it can hide corrosion. Inspect for broken strands on lifelines. Check for sun damage, chafe, and stretching on jacklines, and keep these lines taut with minimal slack.
- Clip on when sailing alone, when sailing short-handed, at night from dawn to dusk, in conditions with poor visibility, when reefed, and in heavy weather.
- Practice "boom awareness" and know the boom's position relative to your head and body while walking on deck, and while tacking and jibing. Walk forward facing the boom, and keep low when returning on the opposite side, with the boom always in sight. Offshore, use a boom preventer at all times, in all conditions, and on all points of sail (not just running downwind) to prevent being knocked overboard. A vessel's boom might be thought of as a deck-sweeping club designed to knock overboard anyone not paying close attention, with the added complication that he or she will probably be unconscious.
- Crew should maintain physical fitness, agility, and balance. This may involve extra time exercising and balance exercises in a gym, prior to a trip.

There is some debate in the Safety at Sea "world" about whether to have the engine on when rescuing a COB. Much of what we know about COBs came from studies done by the Sailing Foundation in Seattle. The researchers found that many victims became injured from propeller strikes, especially if visual contact with the victim was lost as the vessel approached. The conclusion was to maneuver under sail to avoid injuring the victim with the vessel's propeller. Other studies conducted by the Sailing Squadron at the Naval Academy in Annapolis, Maryland, were done with the engine on as a backup to sails.

If used, the engine can be started and left in neutral, ready to be used if needed in the final approach. Rescuers must ensure that all lines are aboard before engaging the engine, to avoid fouling the propeller. It is advisable to return to neutral when close to the victim. The main danger to the victim is being sucked under the stern while the propeller is turning and the boat is moving forward under power

RECOVERY OF CREW OVERBOARD

Time is the critical factor in recovering a crew overboard (COB). (We prefer the gender-neutral term *crew overboard [COB],* which is gaining popularity in the United States, but some product

FIGURE 70-6 This crew overboard module consists of an inflatable horseshoe-shaped buoyant device, 1.8-m (6-foot) inflatable locator pylon with a light, and self-opening sea anchor.

FIGURE 70-7 The MOB Dan Buoy is a ballasted, inflatable pylon with a light at the top, very visible streamer, drogue, and enough buoyancy to float the victim.

names, as well as global positioning system [GPS] functions, still use the term *man overboard [MOB].*) A well-rehearsed rescue under competent leadership with clear communication during rescue maneuvers is most likely to succeed. When someone is observed falling overboard, shout "crew overboard." The crew should then initiate the COB function to create a COB waypoint. Most GPS receivers will store a waypoint named COB, and will also make this the GOTO waypoint, thus providing guidance back to the geographic position of the victim at the time of the accident. A pan-pan should be broadcast on the very high frequency (VHF) radio to alert vessels in the area. Heavy winds, large seas, and strong currents decrease the relevance of the GPS waypoint in marking the COB, because the person may drift downwind and especially down-current while the boat returns to the scene; however, it is still better to have a last known point for a starting position than no position at all. One or more crew should be tasked with spotting and pointing at the victim continuously without losing sight of the victim. Floating objects should be thrown overboard, including buoyant cushions, horseshoe buoys, ring buoys, and extra life jackets, to "litter" the water surrounding the victim. Gear may provide extra flotation to the victim and provides visual cues for the spotter. Unfortunately, most of these objects will drift faster than a person can swim in winds over 10 knots, so the COB cannot be expected to retrieve a thrown life jacket after falling overboard.

Special equipment designed for locating and retrieving a COB should be deployed immediately. This gear should be ready for easy deployment and instant release. Too often, gear is protected against accidental loss by extra wraps of line to the stern pulpit or rigging, and a delay in releasing COB gear will leave it too far from the victim. A COB pole is a 4- to 5-m (12- to 15-foot) floating flagpole that is ballasted to remain upright in rough seas. Without a small drogue (a cone-shaped device to slow a vessel's drift downwind), it will quickly drift away from the designated area. When released from its canister, a crew overboard module (Figure 70-6) automatically deploys a CO_2-activated horseshoe buoy and a 2-m (6-foot) inflatable locator pylon equipped with a drogue and water-activated, lithium-powered light. The SOS Dan Buoy is a throwable pylon that inflates to 6 feet high, with 150 N (35 lbf) of buoyancy and a light on top. The swimmer can grab the pylon handles for support (Figure 70-7).

A variety of lights have been developed to serve as rescue beacons worn by the COB or thrown overboard. An overboard marker strobe marks the site, illuminates the scene for rescuers, and automatically activates when thrown into water. Waterproof personal rescue strobe lights attached to a life jacket can flash for 8 hours at 1-second intervals and are visible a mile away. A strobe light together with a proper whistle (with a special flat design to prevent the whistle body from holding water and dampening sound) should be attached to every life jacket.

In the last decade, a number of personal COB beacons have become available, which is fortuitous because the speed of offshore sailboats has greatly increased during the same period; faster sailboat speeds imply longer distances between a sailboat and the COB. There are several variations on a theme in current models:

- A beacon transmits a signal (commonly on the retired EPIRB frequency 121.5 Mhz) when immersed in water or activated; remaining crew uses a radio direction finder or automatic direction finder to home in on the victim.
- A device worn by all crew members ceases transmitting to an onboard base station when the wearer goes overboard. The station then sounds an alarm and stores a waypoint on a GPS so that the victim's initial location is known (Figure 70-8).
- A radio allows the victim to communicate by voice with the mother ship, using a small VHF transceiver, possibly equipped with a GPS and digital selective calling (DSC) capabilities.
- A personal locator beacon (PLB) may be carried and activated. It is designed to communicate with the Search and Rescue Satellite-Aided Tracking (SARSAT) system, a rescue coordination center, and rescue agencies (Figure 70-9). This system works reliably and worldwide, but it could take too long for the U.S. Coast Guard to arrive at the position. A

FIGURE 70-8 Cessation-of-transmission crew overboard devices alert the helmsman when a particular crewmember's pendant loses contact with the base station. The device stores a waypoint for immediate navigation back to the "swimmer."

FIGURE 70-9 A personal locator beacon (PLB) may be an excellent link back to the Coast Guard, but if you are too far offshore, you may not survive until the Coast Guard arrives. Consider a MOB beacon that alerts the crew on the mother ship, or better yet, both methods.

PLB broadcasts little information of use to the rescuing crew on the vessel or the fleet in which it is sailing.

The beacon contains a GPS and ability to transmit on automatic identification system (AIS) frequencies or both AIS and DSC frequencies, so the mother ship can determine both the position of the victim and that the victim is overboard. This requires that the mother ship have the ability to receive AIS and DSC signals; several modern VHF transceivers have this capability. Ideally, they are with a chart plotter so that the victim's position can be constantly plotted relative to the rescuing vessel.

With these new systems, it is essential that all components integrate seamlessly, because they function together to locate and retrieve the COB.

Crew Overboard Maneuvers

The goal in overboard recovery is to return as quickly as possible to the COB using the simplest maneuver. A boat traveling at a speed of 8 knots moves away from the COB at about 3.9 m (13 feet)/second or 244 m (800 feet)/minute. At that rate, one-half mile is traversed in 3 minutes. Motorboats should reduce speed, return in a simple circle, and approach the victim heading upwind, with the victim about 6 m (20 feet) to one side. Sailboats under power alone can return by simply circling back and approaching the COB in the same manner. Contact should be established with the COB by using a rescue throw rope (i.e., a floating polypropylene line in an easily throwable, high-visibility, nylon storage bag). If the boat is drifting downwind, the vessel should be advanced forward slowly to complete recovery over the leeward side. Sailboats under sail should approach on a close reach, which allows the vessel to speed up or slow down as needed by changing course or luffing the mainsail.

The "quick stop" recovery maneuver is designed for rapid COB recovery, especially with short-handed crews. This method enables the boat to reduce speed immediately by turning into the wind and allowing the headsail (jib) to back. Thereafter, the helmsman keeps the boat turning through the eye of the wind, until headed downwind. The jibe will be relatively gentle because the main is kept sheeted in during the maneuver. After passing abeam of the victim, the jib is dropped (or furled), and the boat heads up to the wind (on a close reach) to stop alongside the victim at an angle of about 60 degrees to the wind with the sails luffing.

The COB must be approached slowly, with the boat under good control, in order to avoid hitting or dragging the COB as soon as the COB is contacted and attached to a retrieval device. By sailing the final approach to the COB on a close reach, the sails can be fully luffing or trimmed to maintain forward movement if short of the mark. The technique is similar to picking up a mooring under sail. Communication with the victim is essential, providing instructions on the rescue plan, what is required of the victim, and reassuring the victim that recovery will occur. Eye and voice contact should be maintained until the COB is safely aboard (Box 70-1).

The final direction of approach used by the rescue boat involves factors that include conditions of the sea, wind strength, drift of the boat relative to the COB, maneuverability of the boat, and condition of the COB. If the seas are large, the approach should be to leeward (downwind) so the boat cannot fall off a wave and injure the COB. Low to flat seas allow an approach to windward (upwind) with a slow drift down to the victim. The boat will always drift faster than the person in the water, so retrieval gear should be ready at all times.

An injured, hypothermic, or unconscious person (not waving or looking at the rescue boat) requires assistance by a rescue swimmer, who should take steps to avoid the cold shock response. The rescue swimmer should be tethered to the boat during recovery, trained in water rescue and lifesaving techniques, and able to recognize warning signs of panic as the swimmer approaches the victim. If the COB is unconscious, possible head and neck injury should be considered; the cervical spine should be stabilized before hoisting the victim out of the water if injury is suspected. The life jacket itself may be used to control the head and neck if it is tightened in the upper chest area.

Recovering an unconscious person from the water is extremely difficult and puts a second person (at least) in a very precarious position. Unless the water is very calm, it is nearly impossible for the victim to avoid aspirating water, causing drowning if the head injury has not been fatal in the first place. The decision to risk a second life in recovering a body must be carefully considered.

Bringing the Victim Aboard

The goal is to bring the COB onboard as quickly as possible. Different rescue techniques should be practiced with the planned boat and crew prior to deciding which methods and modifications work best. The Lifesling pack was developed to enable a single individual to retrieve a person overboard. Stanchion-mounted, the Lifesling[11] pack contains a pliable center section horseshoe, 120 feet of three-eighths-inch floating polypropylene line, and an optional water-activated personal marker light. The flexible floating horseshoe collar can be used as a hoisting sling (Figure 70-10). The collar should be deployed from the stern pulpit and delivered by repeatedly circling the victim, just as a ski boat maneuvers to deliver the towrope to a fallen water-skier. After securing the horseshoe over the head and under the arms, pull the victim back to the boat and make the retrieval line fast to a cleat in the cockpit. At this point, the pace of rescue can be slowed, because the victim cannot be lost and is not likely to drown. The Lifesling can be used with a long halyard, capable of reaching to the bight of line at the Lifesling, or it can be used

BOX 70-1 Engine On or Engine Off?

There's some debate in the Safety at Sea "world" about whether to have the engine on when rescuing a crewmember overboard. Much of what we know about crewmembers overboard came from studies done by the Sailing Foundation in Seattle. The research found that many victims became injured due to propeller strikes, especially if visual contact with the victim was lost as the vessel approached. Their conclusion was to maneuver under sail to avoid injuring the victim with the vessel's propeller. Other studies conducted by the Sailing Squadron at the Naval Academy in Annapolis, Maryland, were done with the engine on as a backup to sails.

If used, the engine can be started and left in neutral, ready to be used if needed in the final approach. Rescuers must ensure all lines are aboard before engaging the engine, to avoid fouling the propeller. It is advisable to return to neutral when close to the victim. The main danger to the victim is being sucked under the stern while the propeller is turning and the boat is moving forward under power. Other experienced sailors have pointed out that sailing a boat back to the victim in light winds may be slow compared with firing up the engine and powering directly into the wind.

FIGURE 70-10 Getting the person in the water back onboard is not a simple task. The Lifesling provides flotation, connects the victim to the vessel, and serves as a lifting sling.

with a 3:1 tackle with 65 feet of line. On smaller sailboats (< 30-foot length overall) that have smaller winches, the 3:1 tackle reduces tension on the fall of the tackle and makes winch operation easier. Note that in Lifesling demonstrations and clinics, students frequently improvise the lifting tackle rigging and end up with suboptimal results. As with all rescue methods, practice with the planned boat and crew to increase familiarity with rescue systems and concomitant likelihood of success.

Lifelines are an obstacle to bringing the COB back on deck, but are an important source of protection for remaining crew. Lifelines may be secured at the stern (or transom) with lashing, rather than shackles or pins, so they can be easily cut and released in a recovery.

The most important factors for a successful rescue are the crew's familiarity with the boat and COB equipment, leadership of the captain, and expert teamwork developed during practice of rescue maneuvers. In 2005, a Crew Overboard Rescue Symposium[4] was conducted on San Francisco Bay during which hundreds of rescues were performed. The resulting publication reviews challenges for a successful recovery, required crew skills, preferred recovery maneuvers, and helpful equipment for locating and retrieving the victim. It is a must-read for every boater. It is also available in a slightly different format on the U.S. Sailing website.[12]

EMERGENCIES AT SEA

FIRE AT SEA

Causes

Uncontrolled fire is a disaster aboard ship. Fires aboard wood and fiberglass boats can double in size every 10 seconds. Approximately 7500 pleasure boat fires and explosions occur annually; of the boats affected, 10% are declared total losses. More than one-half of the 2700 fire-related injuries incurred each year occur on small, open motorboats. According to statistics compiled by Boat U.S. Marine Insurance claims investigations,[3] the leading causes of fires on boats (55%) are alternating current (AC) and direct current (DC) wiring faults. The most common electrical problem is related to chafed wires creating short circuits. Many fires are started by battery cables, bilge pump wires, or instrument wires chafing on hard objects, such as vibrating engines or sharp-edged bulkheads. The DC voltage regulator is responsible for 25% of electrical fires. Eleven percent of fires are started by the boat's AC system, frequently at the shore power inlet box. AC heaters and other household appliances that have been brought onboard cause a small number of fires. Twenty-four percent of fires are started by overheated propulsion systems. Frequently, an intake or exhaust cooling water passage is obstructed, causing the engine to overheat and melt down hoses and impellers. These fires tend to be less serious, but because of the amount of smoke and in areas with flammable fuels, they appear more threatening. Often the fires are simply smoldering rubber, until the engine compartment is opened, allowing fresh air to enter. In Florida, lightning is a major cause of boat fires in marinas. Box 70-2 lists ways to prevent fires aboard ship.

Fire from Fuels, Liquids, and Gases

The explosive potential of fuel depends on its chemical properties and where vapors accumulate in enclosed, unventilated spaces. Hazardous liquids are classified according to flash point, the lowest temperature at which a liquid releases sufficient vapor to sustain burning. "Flammable liquids," such as gasoline,

BOX 70-2 Fire Prevention at Sea

1. Sniff the engine compartment before starting a gasoline engine and before starting the electric bilge blower. Bilge blowers are ignition-protected, but it is still a good idea. Run the bilge blower for at least 4 minutes before starting the engine.
2. Use appropriately sized marine-grade wire. Periodically inspect the wiring for cracks, charring, and deteriorating insulation. Wiring connectors and terminals should be tightly set and periodically inspected for looseness. Each electrical circuit should have fuses or circuit breakers according to American Boat and Yacht Council Standard E-11.
3. Battery compartments must not be completely enclosed, and must allow hydrogen gas to escape through the lids of the battery boxes. Even sealed AGM (absorbed glass mat) and gel batteries can generate hydrogen gas if overcharged.
4. Consult a professional to develop an appropriate ship's grounding system. See westmarine.com/WestAdvisor/Marine-Grounding-Systems for the best article on the complicated topic of vessel grounding.
5. Observe all precautions when taking on fuel. Close all hatches and ports before filling the tanks, and extinguish all flames. Ventilate the boat after fueling.
6. Never leave the stove unattended while in use.
7. Store extra fuel on deck in approved plastic containers, or in a locker that is sealed from the main cabin and drains overboard.
8. Store outboard motor and fuel tank on deck in approved fuel containers.
9. Transfer gasoline and other flammable liquids from one container to another on deck or off the boat, not below decks.
10. Inboard engines require meticulous inspection and maintenance. Perform a routine visual inspection of any operating engine and the exhaust system. Keep seawater strainers for the engine cooling system clean. Become an expert in examining and repairing all components of the fuel system. On vessels with an engine room, consider having a mirror-finished piece of stainless steel that will make it obvious if you have a fine spray of fuel or other liquids in the air.
11. Install a heat-activated automatic fire-extinguishing system in the engine compartment, together with smoke and flame detectors.
12. Properly store fuels, solvents, paints, brushes, and combustibles in a deck storage locker. Keep all rags in metal containers with the lid tightly closed. Do not keep oily rags below decks or in the engine compartment.
13. Place fire extinguishers away from the intended area of use so that they are accessible, and have them inspected and maintained on a scheduled basis.
14. Read the extinguishers' instructions periodically, and practice on a controlled fire away from the boat.
15. Remove extinguishers from their brackets and invert them several times to ensure that the agent inside is loose and flowing.

turpentine, lacquer thinner, and acetone, have flash points below 38°C (100.4°F), meaning they release enough vapor at warm temperatures to form burnable and explosive mixtures. "Combustible liquids," such as diesel oil, kerosene, and hydraulic fluid, have flash points above 38°C (100.4°F).

Gasoline is the most hazardous fuel, causing 60% of fuel-related fires. Typical problem areas are fuel lines, engine connections, and leaking fuel tanks. The first warning sign is frequently the odor of leaking gasoline. Vapors can ignite from heat in the engine compartment and from liquid spills over hot engine parts. Five mL (1 tsp) of gasoline can vaporize and cause an explosion, and 237 mL (1 cup) of gasoline has the explosive potential of several sticks of dynamite. Vaporized gasoline is heavier than air, so it accumulates in the lowest part of any enclosed space, generally the bilge. It is therefore critical to run the bilge blower for at least 4 minutes before starting the engine. Diesel fuel is much less explosive than gasoline. However, pressurized diesel fuel spurting from a burst fuel line will ignite and burn when it strikes something hot, such as the exhaust manifold. Charging batteries generate hydrogen, which accumulates in the battery compartment or the compartment overhead; the gas is lighter than air, highly flammable, and potentially explosive. Sparks from a nearby electric motor may set off an explosion from excess hydrogen produced by overcharging batteries.

A popular galley stove fuel is liquefied petroleum gas (LPG), either propane or butane. Both are highly explosive, heavier than air, and may accumulate in the bilge. Like gasoline, free propane in the bilge is potentially explosive. Proper LPG tank installation requires a completely self-contained vapor-tight locker that opens only above decks. A drain should be located at least 51 cm (20 inches) from any opening to the boat's interior, and should not be submerged while the boat is underway. A pressure gauge connected to the LPG cylinder valve helps indicate a leak somewhere in the system. It is not, as many believe, intended to show the quantity of LPG in the tank. A regulator to reduce pressure in the gas line to the stove and an electric solenoid valve complete the delivery. Safety standards recommended by the American Boat and Yacht Council require an LPG sniffer in the system to continually monitor the air for LPG. The sniffer should be installed at a low point where gas is likely to accumulate; if any is detected, an alarm sounds, and the sensor shuts off the solenoid and gas flow to appliances. Newer LPG stoves have a built-in thermosensor to shut off the gas supply if a burner flame is accidentally extinguished by a draft or wind. The safest practice is to never leave any lighted galley stove unattended. Expert professional assistance is recommended when installing a complete fuel supply system. As of April 2002, propane cylinders must be equipped with an overfill protection valve, because overfilled cylinders may explode after overheating. If a LPG leak is suspected, the propane tank should be shut off using the manual valve, and the main battery switch should be used to kill electrical power to all electrical devices. All hatches and ports should be opened to ventilate the boat, and the ignition-protected bilge or engine room exhaust blower should be activated to remove any leaked gas.

The system should be periodically pressure-tested for leaks, especially after rough weather, or repairs or maintenance to the system using the LPG sniffer. In order to check for a leak properly, the cylinder valve should be opened with the solenoid switched on and all appliance valves closed, and the pressure gauge reading recorded. The cylinder hand valve should be closed in order to assess the pressure drop over intervals of 3 to 5 minutes; if the pressure drops, there is a leak. Soapy water may be used to look for the leak source, because leaking gas will generate bubbles.

To avoid accidents after cooking, switch off the solenoid first, leave the burner ignited until the line is cleared of gas, and then turn the burner off. When appliances are not in use or the boat is unattended, the cylinder valve must be closed. Most importantly, a stove should never be used as a cabin heater; open combustion and flames can deplete a cabin of oxygen and release carbon monoxide, asphyxiating the sleeping crew. Carbon monoxide is a colorless and odorless gaseous byproduct of incomplete combustion; severe exposure can be lethal. Early exposure symptoms consist of fatigue, sleepiness, headache, malaise, nausea, vomiting, and ataxia; these are also symptoms of seasickness, so consider carbon monoxide exposure when several crewmembers exhibit these symptoms. Carbon monoxide detectors should be installed below decks. LPG appliances, such as cabin heaters, manufactured for boats have a sealed combustion chamber, which is not in direct contact with the atmosphere aboard. This is different from recreational vehicle equipment, which is not safe for use aboard a boat.

Stove alcohol is another hazardous fuel, especially if one burner is accidentally extinguished and liquid alcohol used in priming the burner pours onto an adjacent flaming burner, causing a flare-up. A nonpressurized burner can also reignite if refilled with alcohol while still hot. Alcohol fires can be extinguished with fire blankets or wet towels. A stove grease fire can be extinguished with these items or by liberally sprinkling the fire with baking soda. A good precaution is to place a kettle of water on the burner before lighting it; this helps contain any high flames arising from excess alcohol used in priming the burner.

Charcoal grills are popular on boats; however, they should not be used on boats berthed in marinas or when wind can blow hot ashes onto the surface of the boat or a nearby craft.

FIGHTING FIRES

The best way to manage a fire in the engine compartment is to install a properly sized, automatic discharging extinguisher system, which interrupts combustion with chemical materials or gases. Fire-suppressing alternatives to Halon gas (now banned because it breaks down atmospheric ozone) are fluoropropane and fluoroethane (FM-200 for occupied spaces, and FE-241 for unoccupied spaces, respectively). These fire suppressants can be automatically discharged from extinguishers by devices that sense ultraviolet radiation or temperature above 79°C (174.2°F). Automatic systems should also have a manual trigger for activation. Portable fire extinguishers should be discharged into the engine compartment through fire ports (Figure 70-11), which minimize the amount of fresh air allowed into the compartment with fire. Shut down the engine either automatically or manually when fighting a fire in the compartment. Automatic shutoff for diesel engines, generators, and engine room blowers is now required in the event of an extinguisher discharge. Diesel engines consume large volumes of air when running and can quickly deplete the extinguishing agent. The area should be allowed to cool before opening hatches or inspection ports, because fresh air may rekindle the fire, either by diluting the concentration of extinguishing agent or by introducing oxygen.

FIGURE 70-11 Portable fire extinguishers should be discharged into the engine compartment through fire ports.

FIGURE 70-12 Dry chemical fire extinguishers should be inspected to ensure the pressure gauge indicates a full charge, and should be inverted regularly to unpack the extinguishing agent.

In the United States, fires are categorized by letter designations. Class A fires involve common combustible products such as wood, canvas, and plastic (items that leave an Ash). Class B fires are flammable liquids such as kerosene, diesel, gasoline, and alcohol (items that Boil). Class C fires result from an electrical fault, but generally involve class A or B materials once the circuit is deenergized (think of electrical Charge). Class D fires involve burning metals such as lithium batteries or possible flare contents (think of Don't, because these fires are extremely difficult to deal with). Fire classes in countries using ISO standards will find that the letter designations are slightly different, but are defined on the extinguisher's label.

The Coast Guard requires all recreational vessels to have portable fire extinguishers (Figure 70-12). Because many boaters have never used a fire extinguisher, it is important to read instructions and become familiar with use before a fire extinguisher is actually required. The standard multipurpose dry chemical extinguisher (filled with monoammonium phosphate) and newer dry chemicals can be used on all types of fires. Dry chemical fire extinguishers work by preventing access to oxygen and interrupting the chemical reaction of fire. ABC dry chemical extinguishers are painted red. The chief disadvantage of ABC extinguishers is that the monoammonium phosphate powder is difficult to clean up and damages electronic equipment. Halotron 1 is a relatively new, clean, "no residue" agent for portable extinguishers (also painted red) that discharges as a rapidly evaporating liquid. It is safe for electronics and computers, and is a good alternative to older dry chemical powders. A major drawback of all portable extinguishers is the extremely brief window of opportunity provided to put out the fire. The common B-1 extinguisher discharges completely in just 10 to 13 seconds. Units with greater capacity simply deliver more chemical over the same period. Because of a short discharge time, more than one fire extinguisher should be available to cope with a large blaze.

The acronym "PASS" is used to remind users of how to use a portable extinguisher. P stands for Pull; the pin on the extinguisher's head must be pulled for activation. A is for Aim, because there is a limited amount of discharge time. S stands for Squeeze; release the extinguishing agent. Finally, S stands for Sweep; the most effective use of the agent is to sweep across the base of the flames. Fires extinguished with dry chemicals should be considered hazardous until cooled to room temperature. A single portable extinguisher should be dedicated to the engine compartment, a second to the galley (where it should be reachable even if the stove is on fire), a third to the area under the fore hatch, and the fourth (and largest size practical to stow) in a cockpit locker. It is also recommended to have one in each stateroom. The crew should know all locations. One should never have to walk more than half the boat's length to reach an extinguisher. The ideal location for a portable extinguisher in a closed compartment is next to the exit door. Fires involving common combustible solid material can be brought under control by cooling with large amounts of water. Fires involving flammable or combustible liquids can be smothered to remove oxygen by using a fire blanket (required for many ocean races on sailboats). Electrical fires can be difficult to extinguish because the heat source (a shorted wire) can reignite fire even after a fire extinguisher has been used. Deenergizing the electrical circuit can often control electrical fires, especially when a short circuit is generating sufficient heat to cause other materials to combust. Every boat must have a main battery switch and/or AC breaker to turn off the entire electrical system. Water does not extinguish electrical fires, but may be effective on the resulting class A fire after the electrical circuit has been disconnected. See Box 70-3 for fire-fighting guidelines.

FLOODING

Flooding, with potential for sinking, is a threat to every boater. Boat U.S. Marine Insurance examined 50 claims[2] from recreational boats that sank while underway, ranging from a tiny personal watercraft to a 16.5-m (54-foot) ocean-going sailboat. Thirty-four percent of boats sank because of leaks at through-hull fittings, outdrive boots, or raw water cooling system/exhaust. The single most critical reason that small motorboats flood in open water relates to transom height. Engine transom cutouts may be only inches above the waves, and the motor well may not protect the cockpit. Often, weight distribution of passengers and gear to the stern contributes to the problem. Weight distribution problems may be exacerbated by heavier four-stroke outboards when used with boats that are not designed for the incremental weight.

Flooding may occur from system failure or construction (6% of boats sank after coming down hard off of waves and then splitting open), or structural damage from collision and extreme

BOX 70-3 Guidelines for Fighting Fire at Sea

1. Attack fire immediately at the source. Detection and reaction time must be immediate, before the fire burns out of control. Prepare a plan that has been shared with the crew so that everyone knows their responsibilities and the location of equipment.
2. Initiate a MAYDAY call immediately. The purpose is to alert ships in the area in which the crew may have to abandon ship. Always state your position clearly.
3. When fire is discovered, all crew should report on deck as quickly as possible with life jackets and fire extinguishers in hand. Ensure that the life raft is away from the fire. This is another good reason to store life rafts on deck or in a deck-accessible locker.
4. Slow the boat to reduce relative wind, and steer to keep smoke and flames clear of the crew and vessel. Keep the fire on the downwind side of the ship, exposing the smallest amount of the boat's structure to the flames.
5. Crew should always have a clear escape route when fighting a fire.
6. Cut off the source(s) of the fire (e.g., fuel supply, electric current, and ventilation system). Turn off blowers, and stop the engine if fire is in engine compartment.
7. Shut off the air supply to the fire. Close hatches, doors, and vents to all compartments free of people.
8. If you must open a hatch to discharge a portable extinguisher, beware of burning your hands or face. As fresh air enters the compartment, the fire will rise to the air source and flare up. The safest way to open a hatch is to wear gloves and stand on the hinged side of the hatch while it is opened. A "fire port" that allows a fire extinguisher to be discharged without opening an engine compartment can easily be retrofitted to a boat.
9. If the fire is too large or out of control, abandon ship before the fuel tanks explode.
10. Check the engine compartment frequently to detect smoke/fire.

BOX 70-4 Sources of Flooding

Failure of through-hull fittings involving the following systems: head, galley, wash basins, shower sump, bilge pumps, centerboard pins and cables, engine exhaust, engine cooling, deck and cockpit drains, bait box, drain plugs (for small boats), knot meter sensor, depth sounder sensor, propeller shaft stuffing box, shaft struts, shaft log, rudder post, and keel bolts
Failure of hose connections, clamps, pipes, and fittings
Open hatches, companionways, portholes, and ventilators
Siphoning of seawater back into the boat because of poor system design or failure of a check valve
Collision with large floating or submerged objects (e.g., vessel, container, whale, rock, reef)
Structural failure in hull, deck, or rigging
Punctured hull from overboard broken spars (e.g., after dismasting)
Clogged scuppers or cockpit drains
Waves entering the cockpit of an outboard-powered motorboat through the transom engine cutout

BOX 70-5 Damage Control Kit

Assorted hose clamps and tools to adjust them
Conical wood or foam plugs, preferably secured at each through-the-hull opening, and spares
3M Fast Cure 5200 cartridges, or epoxy putty sticks
Caulking gun, extra cartridges
Triangular collision mat, spare plywood for patches
Water-activated fiberglass repair fabric (Syntho-Glass)
Pruning saw with spare blades, bolt, and rigging cutters
Drifts matching the size of clevis pins
Duct tape, electrical tape, and self-amalgamating tape
Self-tapping stainless steel screws, threaded rod, bolts, nuts, and large washers
Extra buckets

weather (Box 70-4). Before abandoning ship, quickly assess damage; time is the limiting factor. Proper tools and repair supplies (Figure 70-13) must be stocked in a damage control kit (Box 70-5), and the crew must know how to use them effectively. Before departure, specific duties are assigned to crewmembers in case of emergency, so they know what to do in the event of flooding, COB, fire, grounding, and dismasting. Duties include damage control, radio transmission of a MAYDAY message (which can be cancelled later if necessary), and preparation to abandon ship.

FIGURE 70-13 Damage control kits are not suitable for making fine furniture; in fact, one may have to remove the furniture in the vessel's saloon to get to the source of the leak. Saws, hammers, rivet tools, Band-It tools, and a rechargeable drill are just some of the items one needs to carry.

Flood Mitigation

Early detection of flooding is crucial for an effective response. Visual inspections of the bilge, engine room, galley, and head while underway should be performed frequently, to maintain watertight integrity at all times. Port lights, hatches for the main companionway, engine room, lazarettes, cockpit lockers, fish holds, and other potential breaches require gaskets and proper dogging (locking) devices to ensure watertight seals. All of these seals should be closed while underway, especially in heavy weather, no matter the temperature below decks. Place storm boards over large windows to protect them during long ocean crossings. Flooding may occur either from top down or bottom up. After a knockdown or wave breaking over the cockpit, water may go straight below decks if the companionway drop boards are not in place. These boards require a means to secure them that is operable from both sides, or a lanyard that can be tied securely to keep trapezoidal boards from upward movement. Severe flooding, with damage to batteries, electronics, navigation station, and engine, usually results from top-down flooding. Loss of electrical and engine-mounted bilge pumps prevents dewatering the ship and reduces the amount of time available for the crew to identify the source and stem the flow.

Discharge plumbing requires seacocks. Regularly inspect through-hull fittings, the engine drive shaft stuffing box, clamps, and hoses. Avoid polyvinyl chloride pipes or any other domestic plastic plumbing fittings for through-hull fittings below waterline; these materials are prone to fracture if struck by shifting stores. Preferred materials are Marelon (reinforced noncorrosive plastic) or silicon bronze, together with two stainless steel hose clamps over hose barbs. Post a diagram showing locations of all through-hull fittings and routes of connecting hoses in the cabin. Seacocks should be readily accessible and unobstructed, and easy to find even in conditions of darkness or reduced visibility. Install U-shaped antisiphon loops above the highest waterline, because the waterline changes as the boat heels in. Without these loops, water can siphon back through the hose and into the bilge.

Reliable manual bilge pumps may mitigate mild flooding (Figure 70-14). A high-capacity pump or multiple pumps can buy time for locating and plugging the leak. However, no pump can keep up with even a modest-sized hull breach. If a boat is equipped with an automatic bilge pump(s), install a cycle counter on the pump and an "on" light to alert crew when the pump is activated. A second emergency pump mounted above the first, using a separate float switch, can provide added pumping capability if the first pump cannot keep up with the leak. In this case, an alarm installed in the circuit will alert crew to flooding in excess of what the first pump can handle.

FIGURE 70-14 To be effective, a manual bilge pump has to allow the user to operate it for an extended period of time. A long handle and convenient location, along with a high-capacity pump, make a tremendous difference.

Bilges should be kept clean and free of debris to avoid clogging the pump strainer. New boats frequently have debris (sawdust, masking tape, wire ties) remaining from construction that can clog pumps. Regular inspection and maintenance of the entire pumping system is strongly recommended. Aluminum-body bilge pumps may corrode from the inside out, especially while retaining saltwater. They may appear to be in perfect condition, when in fact they may be useless. Hoses crack with age, rubber components become dry and brittle, valves jam, and moving parts deteriorate through wear and corrosion. To guarantee reliability, manual bilge pumps should annually be disassembled, inspected, and cleaned. Bilge pump handles should be easily accessible and secured with a lanyard near the pump to avoid loss after a knockdown or rollover. Offshore boats require at least two manual bilge pumps, one operable from above decks and one from below decks. A captain should have thorough knowledge of the capacities of a boat's compartments and a means to pump them out if flooded. Test the pump's capabilities by intentionally flooding the bilges (preferably with fresh water) and timing how long it takes to pump out the water.

The volume of water entering through a defect in the boat's hull depends on the breach size and its depth below waterline. A hose that is 2.5 cm (1 inch) in diameter disconnected from a seacock 30 cm (1 foot) below waterline allows 75 L (20 gal) of seawater per minute into the cabin, which is about as much as the best manual bilge pump and a fit operator can handle. A disconnected open seacock of the same diameter just 60 cm (2 feet) below waterline admits three times that amount of water. A boat equipped with the largest manual diaphragm bilge pump can pump a maximum of 1 gallon per stroke if fitted with a 2-inch hose together with an able and inexhaustible crew member; such a pump is rated at 30 gpm based upon a 30-stroke per minute rate. It is therefore critically important to locate and stop the leak, rather than fight what is likely a losing battle by pumping to prevent sinking. There are, however, emergency large-capacity hydraulic bilge pumps capable of pumping hundreds of gallons per minute. Large-capacity pumps may be installed on larger boats with sizeable engine rooms and are capable of saving vessels when pumping large amounts of water is required. Engine-driven front-mounted/clutched centrifugal pumps are the best solution if there is room for the installation and hoses. An alternative is to use one or more high-capacity DC centrifugal pumps.

As water rises in the cabin, a leak becomes more difficult to locate. The inflow rate decreases as the depth of water inside increases, because the pressure gradient is reduced. At a critical level of flooding, the inflow rate may slow sufficiently to allow pumps to handle the volume, so one is best advised to keep pumping. A point of zero net flooding may be reached from inherent buoyancy of the boat as it settles in the water, which is another reason the ship should not be abandoned unless it continues to flood. Many small wood and fiberglass boats float when fully flooded or swamped. Boats less than 6 m (20 feet) in length constructed in the United States after July 1972 are required to have sufficient built-in flotation to remain afloat when swamped. Small boats (e.g., day sailors, small open motorboats) can obtain additional buoyancy by lashing down unused life jackets, cushions, and fenders.

A tapered, soft, and dry wood plug sized to fit a leaking through-hull fitting (including propeller shaft) can serve indefinitely as an emergency seal; the plug will absorb water and swell to seal the fitting or any small puncture in the hull (Figure 70-15). Plugs should be removed from the damage control kit at the beginning of the voyage and attached to their respective through-hull fittings with lanyards; this makes them instantly available and helps crew find them in the dark. Forespar's Sta-Plug is a new damage control product. It is a tapered circular cone-shaped plug about 23 cm (9 inches) tall and 12 cm (4.75 inches) across at the base, made of soft foam that is a spongy but firm cellular material, coated with a flexible sealer that adds strength and color. The TruPlug can be used as a temporary or emergency plug in boating applications where water would enter a circular, oval, or irregular hole caused by mechanical failure or hull breach due to impact.

FIGURE 70-15 A damage control kit focused on flooding. Tapered wooden (or foam) plugs, underwater epoxy, saws, and hose clamps provide a variety of tools to stem the flow. *(Courtesy WheelHouse Technologies, Inc, Hudson, MA.)*

Large holes can be overlaid with a collision mat (Figure 70-16) placed outside the hull to supplement a temporary interior patch. The mat is a triangular-shaped piece of heavy canvas or vinyl-coated fabric with grommets and lines that enable it to be positioned and secured on the exterior surface. The mat is held in place by water pressure and lines. Collision mats can be purchased or improvised by using a small sail or awning material, although it is a mistake to make the mat too large; generally 1.2 m (4 feet) on an edge of the triangular shape is adequate. Water pressure automatically spreads the patch over the hole to form an effective seal and holds it in place. A commercial hull repair kit features flexible oval concave sheet metal plates with rubber gaskets. A bolt is welded to the intended exterior piece. The exterior oval plate is passed through the hull breach; the second plate is used to reinforce it from the inside. Finally, the plates are tightened together with a thumbscrew.

In the absence of specialized repair equipment, any soft, pliable material, such as a life jacket, mattress, blanket, or foam pad, can be used to slow water rushing through a break in the hull. When placed against the exterior hull, the suction effect created by flow and hydrostatic pressure will generally provide clamping pressure to the plug or patch. If a plug is positioned from inside the cabin, it should be shored up with a board and braced with a metal or wood pole (e.g., oar, mop handle, boat hook, whisker pole, strut, bunk rail). The eventual solution to a hull repair may start with a crude internal patch to slow the flow of water, followed by an external patch, followed by an improved internal patch with plywood and bracing. The vessel's pumps may then be able to handle the remaining leaks.

FIGURE 70-16 A collision mat consists of a triangular piece of fabric that is held in place with lines attached to each corner. Water pressure and the lines hold it in place.

FIGURE 70-17 Coast Guard gasoline-powered dewatering pumps are extremely effective, but are also a bit cryptic in their use. Note that she is reading the waterproof instructions, while he ponders the controls. Some are self-priming, while others may have a hand pump to draw water into the pump body.

Underwater patching compounds can be used to bond a solid plate over the hole or to impregnate expandable material or packing to serve as plugging material. Patching compounds vary in cure speed, ease of mixing, mixed viscosity, and adhesiveness. Some products work only on specific hull materials (e.g., wood, fiberglass, or aluminum). Direct repairs should be supplemented with additional measures to help slow water inflow. The vessel should be heeled away from the area of damage to decrease hydrostatic water pressure. This is relatively easy to accomplish under sail. Crews of powerboats should shift items to the side opposite the leak and slow the forward speed if water is entering a hole in the bow.

When ingenuity and improvisation fail to stop flooding, the U.S. Coast Guard can supply a portable gasoline-powered dewatering pump to assist a sinking vessel. The unit is simple to operate and comes in a waterproof barrel with hoses, gasoline, and illustrated operating instructions (Figure 70-17). When dropped from an aircraft into the sea, a retrieving line will be dropped to the deck crew. Two people are required to lift the pump from the sea onto the deck. The standard CG P-1B dewatering pump can pump 450 Lpm (120 gpm) at 3 m (10 feet) lift, and is capable of running 4 to 5 hours on a tank of gas. The P-1B pump, known as the drop pump, is carried by helicopter and by any of the Coast Guard rescue craft. A larger pump, the CG P-6 (classified as a dewatering/fire-fighting pump) is carried by Coast Guard search and rescue (SAR) ships and is placed either onboard or passed via lines, depending on weather conditions (Figure 70-18). Pump capacity is 950 Lpm (250 gpm) at 3.6 m (12 feet) lift; it runs 4 to 5 hours on a tank of gas. Both pumps are dramatically more effective than even the most robust manual or electric bilge pump. However, 950 Lpm (250 gpm) is equivalent to the inflow of water from a 3-inch hole (a relatively small breach) only 60 cm (2 feet) below waterline; any larger hole at this depth will exceed the capacity of even the P-6. Whatever pump is used should be run outside, not in an enclosed space where carbon monoxide may accumulate. Pumps should be refueled only after the engine is stopped. As with all safety equipment, these pumps can be confusing to operate for nonprofessionals, especially at night while a ship is swamping or sinking. Attendance at a safety at sea seminar or other survival training is highly recommended to become familiar with infrequently used and new safety gear.

Some oceangoing cruising vessels are built with watertight compartments to confine flooding to a limited area. Crew should know locations of watertight doors and have knowledge of their operation. If flooding and sinking are inevitable, the captain should consider running the boat onto shore.

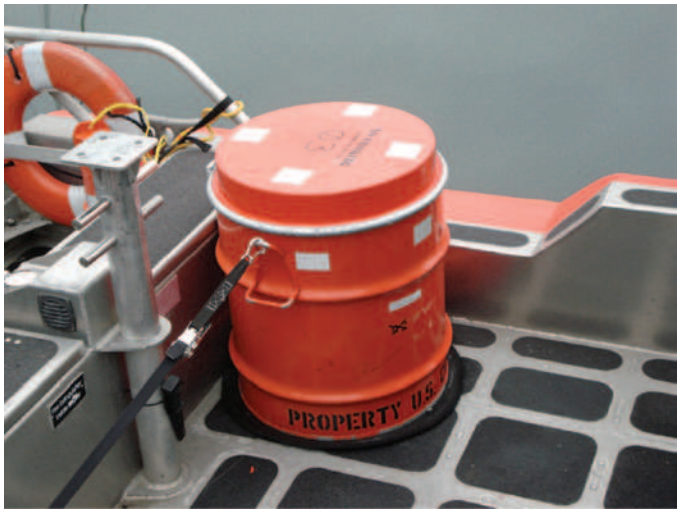

FIGURE 70-18 Larger P-6 pump carried on a Coast Guard 29-foot response boat.

COLLISIONS WITH OTHER VESSELS

International regulations for preventing collisions at sea are referred to as COLREGS. They define the responsibility of ships when collision is possible between two boats, as when crossing each other, overtaking, or meeting head on. Although the *stand on* vessel, which has the right of way over the *give way* vessel, is permitted to hold course and speed, rules require that both ships take any actions necessary to avoid an imminent collision. In the presence of large ships, small boats must be especially vigilant. All tow vessels or large craft, such as freighters or tankers operating on inshore waters, should be considered restricted in maneuverability. Rules 3 and 18 of the Inland Navigational Rules make it clear that a sailing vessel does *not* have the right of way over these vessels. In commercial traffic areas, a deck watch must be maintained at all times, and approaching ships should be hailed on VHF channel 13 (the vessel's pilothouse), or channel 16 if no response, if the ship's course or intentions are uncertain. If two vessels are in a crossing situation and the bearing (angle) between them remains constant, collision is inevitable. Visibility from the pilothouse of a large vessel may be partially obstructed by containers, fishing gear, or other items on deck; small craft may not be seen by the ship's lookout or pilot, show up on radar (Figure 70-19), or be granted the right of way. Reduced visibility

FIGURE 70-19 Although radar reflectors make a vessel incrementally more visible on a ship's radar, it is a lot easier for you to see them than vice versa. Keeping a radar watch in shipping channels makes sense, as it does here in the Strait of Juan de Fuca.

is a major cause of collisions along shipping lanes for commercial and recreational small craft. Every crew should be prepared for defensive action.

Automatic Identification System

Although radar, visual observations, and plotting are the anticollision tools for most mariners, a relatively new tool for avoiding collisions is the automatic identification system (AIS), which is required on a variety of commercial vessels and is also available for use by smaller craft. AIS transceivers consist of automated VHF radios that transmit information about the vessel's position, speed, and course, as well as name, classification, call sign, and maritime mobile service identity (MMSI) number, while also receiving the same information from similarly equipped vessels. The transceivers have a dedicated GPS to obtain position data, and use two VHF channels to send and receive data. Three categories of AIS transceivers are in use:

- Large ship AIS receivers are class A (for commercial ships), which transmit more information, more frequently, and with more transmission power.
- Class B transceivers are used on vessels not rated as Safety of Life at Sea (SOLAS) vessels and on recreational boats; they transmit fewer data fields, less frequently, and at lower power.
- Receive-only models "eavesdrop" on transmissions of other vessels but do not send out information about the vessel on which they are installed.

The two transmitting systems are compatible, but the class A system is more robust. As of 2014, some domestic commercial vessels, including self-propelled vessels of 20 m (65 feet) or more in length and those engaged in commercial service, are required to have onboard a properly installed, operational, Coast Guard type–approved AIS.

All AIS class A transceivers and some class B models incorporate a display that allows other vessels to be plotted and provides a list of vessels and their threat potential, or provides this information to a chart plotter, network display, or radar. Alarms can be set to warn if another vessel will pass within a certain distance, so that vessels posing the greatest threat are highlighted.

Comparisons between benefits of radar and AIS are common, but the best solution is to have and use both technologies. Radar can detect large vessels, buoys, non-AIS vessels, shorelines, harbor configurations, and rocks. Radar requires training and experience to operate and interpret. AIS provides critical information about vessels and aids navigation on vessels equipped with transceivers, but no information about other threats.

Using Radar to Avoid Collisions

Radar is invaluable in conditions of poor visibility (Figure 70-20). At night or in fog, radar can be used to identify other ships, hazards to navigation, squalls, and other local weather that may endanger a vessel.

Small, affordable radar units are available for recreational boaters. Most are simple to operate, but require practice and patience for mastery. The radar horizon is a function of radar antenna height and target height; the higher each is located above sea level, the better the range of visibility. Modern units integrate a variety of data, including electronic bearing lines, rings that give a fast reference point to a target, and the variable range mark, which marks the range to a target at a particular time. These data are invaluable for avoiding collision with another vessel. Many units can be integrated with electronic chart displays and interfaced with computers for precision course plotting and tracking during navigation in narrow and complicated passages. A big advance in collision avoidance is the automatic radar plotting aid (ARPA), which works in conjunction with the main radar and is required (under COLREGS rule 7) to be used whenever a risk of collision exists. ARPA provides automated long-range scanning, sounds an alarm when a vessel comes within a predetermined distance, calculates the speed and bearing of other vessels, and automatically calculates course alternatives to avoid collision. Radar's interface with AIS provides the most comprehensive data set for collision avoidance.

FIGURE 70-20 Plotting aids, such as ARPA and MARPA, remove much of the doubt about whether or not the other vessel is going to pass safely.

One problem common to radar units on small boats is that during storm conditions, large commercial ships and yachts may be lost on the radar screen because of echoes from nearby tall waves. Intense reflection of radar signals from the sea is called *sea clutter*. Fiberglass and wooden boats with wood spars are nearly invisible to radar. Regardless of construction, all boats should have a radar reflector mounted at all times while in shipping lanes, on the open sea, or under conditions of reduced visibility.

Search and rescue transponders (SARTs) render the object to which the SART is mounted significantly more conspicuous on a nearby vessel's radar screen. The radar-SART is used to locate a survival craft or distressed vessel by creating a series of dots on a rescuing ship's radar display. Essentially, it functions as an active radar reflector. SART is an excellent aid for life rafts and boats in distress, which usually do not return noticeable radar reflections on their own. A SART will only respond when interrogated by 9 GHz X-band (3 cm wavelength) radar, which is standard on all SAR craft but not on all commercial craft, which often use 3 GHz S band radar. New SARTs respond to both bands.

An important function of radar is the ability to maintain an electronic guard zone. The radar scans this zone, preset as the circle formed by a given radius, and sounds an alarm when a new target enters the guard zone for a certain number of sweeps. Unfortunately, commercial ships and solo ocean racers may rely too much on radar while they run on autopilot, and fail to post a lookout on deck. This is a clear violation of rule 5 of the COLREGS: "Every vessel shall at all times maintain a proper lookout by sight and hearing."

Visual Means of Avoiding Collisions

For collision avoidance, many cruising sailboats install a masthead strobe light. Although these are highly visible, masthead strobes are not accepted internationally as legal running lights, and the Coast Guard does not recommend them. A flashing strobe light may be interpreted as a distress signal, paradoxically inviting an unwelcome convergence of ships. Shining a powerful spotlight on the mainsail, turning on spreader lights, or igniting a white "anticollision" flare can be an effective alternative means of alerting a ship to the danger of collision. It is recommended that cruising sailboats have a masthead tricolor light for coastal and offshore cruising when sailing (to extend the range of visibility), in addition to deck-level lights for meeting, powering,

crossing, and passing maneuvers when vessels are nearby. The LED tricolor masthead light combines sidelights, sternlight, and all-round white light (for anchoring) and is notable for its energy efficiency, durability, longevity, and brightness. The U.S. Coast Guard Navigation Rules,[10] International-Inland, specifies light requirements for every description of watercraft.

Mariners should recognize the different patterns of lights displayed by commercial ships. Beyond U.S. coastal waters, many ships do not always show appropriate lights, and the risk of collision is increased. Box 70-6 reviews tips to prevent collisions at sea.

Sailors crossing the North Atlantic in summer along a great circle route to Europe need to beware of colliding with icebergs. Every year from February to July, more than 10,000 icebergs are separated from Greenland glaciers. Global warming has had a profound effect on the number of icebergs; it increases local production of icebergs because ice shelves fragment in warming coastal waters. In August 2010, a giant ice sheet measuring 260 km^2 (100 mi^2) broke off from the floating portion of Petermann Glacier. By 2012, splintered fragments had drifted southwest, and on July 15, 2012, a 130-km^2 (50-mi^2) piece calved from the northern tip of the glacier and drifted into Baffin Bay. At least 1000 icebergs drift in the Labrador Current south and east of Newfoundland and are a hazard to mariners above 41 degrees north latitude. Navigation around icebergs is complicated by dense fog often present over the Grand Banks region, and the difficulty of detecting icebergs with radar. U.S. and Canadian Coast Guards broadcast iceberg reports twice daily. The International Ice Patrol[7] broadcast times and frequencies can be obtained from the International Ice Patrol in New London, Connecticut.

BOX 70-6 Preventing Collisions at Sea

1. Post a lookout with a 360-degree view of the horizon.
2. Know the rules of the road and right of way.
3. Use radar (if available) and running lights. A tricolor masthead light is most visible and cannot be obstructed by sails or other gear when sailing in the open ocean.
4. An automatic information system (AIS) allows one to plot large vessels on the chart plotter or AIS display, and helps prevent collisions at night and in busy traffic lanes.
5. Mount radar reflectors at least 4 m (13 feet) above the waterline.
6. Do not assume that other boats have operational or unobstructed running lights. Be aware that outside of U.S. waters, navigation lights on foreign ships may not match standard configurations.
7. Observe Rule 7 of the COLREGS: "When a vessel has any doubt as to whether a risk of collision exists, she shall assume it does and avoid it." Be prepared to change course and speed.
8. Ask yourself, "Can I be seen?" If you cannot see the wheelhouse windows of a ship, they probably cannot see you. Deck cargo, cranes, and containers can make it impossible for watchkeepers on a ship to see you up to 3 miles in front of the vessel. Do not assume you are seen, even in daylight.
9. If you hear a foghorn, stop and let the other ship steer around you.
10. If a collision threatens, sound the danger signal (five or more short blasts on the horn), and take whatever actions are necessary to save your boat.
11. Maintain radio contact with other ships in the area. VHF channel 13 is the bridge-to-bridge channel. Use VHF channel 16 if there is no response.
12. Avoid congested shipping routes if possible, and avoid navigating in the harbor's incoming and outgoing traffic separation lanes designated for commercial traffic. If you have to cross the traffic lanes, do so at right angles and remain in them for the shortest possible amount of time.
13. When your boat is in the way of another vessel, the right of way does not automatically grant you safe passage. Regardless of the COLREGS, be prepared to take evasive action. Caution is the primary rule of the sea to avoid collision.
14. To avoid collision with another vessel, make early and obvious changes in course and/or speed. Port-to-port is a good rule for most head-on passing situations (sound one whistle). In overtaking situations, sounding one whistle means you are overtaking on the starboard side of the overtaken vessel, and sounding two whistles means the port side.
15. Give large ships a wide berth. Whenever possible, avoid meeting or overtaking tugboats towing vessels or barges near river bends, bridges, and narrow channels.
16. Observe the bearing of an approaching vessel (the direction relative to you). If the bearing does not change, and if the distance between the two boats is decreasing, you are on a collision course.

HEALTH MAINTENANCE AT SEA

THE FEARSOME FIVE

The "Fearsome Five" are health issues that must be addressed to maintain optimal physical and mental performance: food (calorie depletion), fluid (dehydration), Fahrenheit (hypothermia), fatigue (sleep deprivation), and fitness (injury, illness, infection).

Food

To keep the crew well fed, meals on a long sea voyage should be simple, high calorie, and easy to digest. Some meals should be prepared in advance to minimize the risk of seasickness for the cook, who would otherwise be required to spend extended periods below decks. Snacks should be readily available. Energy bars, trail mix, crackers, and fresh or dried fruit are good choices. Sips of water help speed absorption and digestion of snacks. The goal is to avoid hypoglycemia with depletion of reserve muscle and liver glycogen stores.

Fluid

Common causes of dehydration include excessive sweating from fever or vigorous exercise (especially in hot weather), profuse/protracted diarrhea/vomiting, and restricted use of potable water. Seasickness and gastroenteritis may cause dehydration. Fluid loss at rest in a thermoneutral environment (28° to 30°C [82° to 86°F] and 50% relative humidity) is via skin, lungs, and kidneys. Each of these organs has an obligatory daily fluid loss of approximately 500 mL (1 pint). Minimal daily body water loss is therefore 1500 mL (1.6 quarts). Headache, nausea, lethargy, apathy, lightheadedness, and hypotension can develop with a deficit of 1 to 2 L (2 to 4 pints; 3% to 5% of total body water); these symptoms can mimic those of seasickness and heat exhaustion. Boaters often fail to appreciate both heat absorption and the drying effects of warm breeze on skin. Continuous evaporation of sweat removes the visible reminder of fluid loss, and quick-drying fabrics accentuate this invisible evaporative loss. Signs of overheating also may be less obvious if the body absorbs radiant heat from the sun while a breeze cools the skin. Exposure to sunny, hot, breezy, and dry conditions promotes increased fluid loss from the lungs, because the air we inhale and exhale is humidified in the nose and bronchial tree, increasing "insensible loss." Boaters are more susceptible to dehydration during this "ideal boating weather." On an average summer day, sailors should consume a minimum of approximately 2 quarts of fluid. Feeling thirsty is not always a reliable early sign of dehydration. However, the sensation of thirst should not be ignored, and one should drink fluids until thirst is satisfied. A good way to determine the hydration level is to monitor the color of urine. Clear to pale-yellow urine indicates sufficient fluid consumption. Dark yellow or tan-colored urine indicates dehydration.

There are a variety of causes of and remedies for limited fluid intake onboard. Poor taste of a boat's tank water can reduce voluntary fluid intake; powdered drink mixes or fresh citrus fruits can counteract bad-tasting water. Seasick sailors often suffer from dehydration because of recurrent nausea and vomiting. When rehydrating seasick crew, frequent sips of small volumes of water are often more effective than drinking large volumes at a single sitting. Self-imposed water restriction is sometimes practiced to reduce urinary frequency, especially in rough weather, when it is often difficult for crew to go below to use the head. In these circumstances, having the helmsman make a brief change of

course allows the crew to safely go below decks. Men suffering from prostate enlargement may also voluntarily restrict fluids in order to urinate less frequently. Some drugs for seasickness accentuate urinary retention in men with enlarged prostates; these medications should be tried before going to sea to evaluate for potential side effects.

Fahrenheit

Hypothermia (see Chapters 7 and 8) may develop acutely when crew falls overboard and remains immersed in cold water (< 25° C [77° F]) or over a period of hours to days during prolonged exposure to the elements, as when wind, rain, and seawater inundate the crew on deck in cold ambient temperatures. For sailors, staying warm requires staying dry. Good foul-weather gear (including sea boots, hat, and waterproof gloves), combined with insulating layers of clothing made with modern synthetic fabrics (highly breathable, moisture-wicking, and fast drying), are essential. Mild hypothermia, defined as a core temperature above 32° C (90° F), is the only level of hypothermia that can be treated onboard. More severe hypothermia requires evacuation to a medical facility. Sustained uncontrollable shivering is the most reliable and earliest sign of drop in core temperature, and usually begins in earnest at 35° C (95° F). Other clues are alterations in motor skills and changes in mental status. As blood is diverted from muscles and nerves, there is loss of manual dexterity, large muscle coordination, and strength. Clumsiness occurs while performing simple tasks, such as adjusting binoculars or using navigational instruments. Walking safely on deck and working with lines and gear become hazardous. Subtle changes in mental status cause impaired judgment, confusion, and disorientation. Initial treatment of a conscious and shivering mildly hypothermic person (core temperature above 32° C [90° F]) is to prevent further cooling and heat loss. This person is capable of rewarming himself or herself and does not require evacuation. The victim should be sheltered from wind and water, and wet clothing replaced with multiple layers of dry insulating garments after the skin is completely dried. If dry clothing is not available, an external vapor barrier should be added with additional foul weather gear. A windproof layer minimizes convective and evaporative heat loss. When practical, the victim should be wrapped in blankets, a sleeping bag, sails, or sail bags. Provide calories with simple carbohydrate foods and sugar-based liquid drinks, and allow vigorous shivering to generate rewarming heat. Warm liquids are psychologically beneficial but do not significantly increase rewarming rates.

Avoid warming skin directly because this inhibits shivering. Warm showers will not warm the core; instead, they may cause vasodilation and severe hypotension (circumrescue collapse).

Fatigue

Sleep deprivation and fatigue impair physical and mental performance, and foster cognitive errors, poor judgment, mood changes, and even hallucinations. Sailors often have irregular sleep schedules, prolonged watches, and difficult sleeping conditions. The crew should sleep in secure sea berths that are narrow with lee cloths, so they do not roll around, fall out, or battle to stay in their berths. The challenge is to improve sleep efficiency. Sleep cycles have light and deep stages of rest. A 1-hour nap can quickly bring about deeper restorative sleep. Regular watch and sleep schedules, and napping to reduce fatigue are recommended. Sleep deprivation increases susceptibility to seasickness, and many medications for seasickness cause drowsiness, which may further disrupt regular sleep schedules and patterns.

Fitness

Seasickness, sunburn, and dehydration are the most frequent ailments, and together with infections, are the most common illnesses for otherwise healthy cruising sailors. Sailors are constantly exposed to solar radiation (see Chapter 16). Sunlight is reflected off water, especially from choppy seas; reflected rays can burn eyes and undersides of the mouth, nose, and chin. Overcast days afford little relief, because most radiation is transmitted and scattered through haze and high clouds. Boaters especially suffer from sunburn on cloudy days, because they neglect to take measures for sun protection, such as using sunscreen or wearing protective clothing, hats, and sunglasses. Certain medications increase the skin's sensitivity to sun exposure; medications should be reviewed for this possibility and extra precautions taken.

Soft tissue extremity injuries are common, usually caused by trips and falls during sailing maneuvers, especially in heavy weather. Being caught in lines or struck by objects, including sheets, blocks, lines, and hardware, contributes to most injuries. Typical injuries are contusions, lacerations (most often on hands), sprains, and strains. Severe injuries, such as fractures, concussions, and dislocations, are not common. Most injuries occur in the cockpit or on the foredeck.

In the enclosed and tight quarters of a boat, upper respiratory infections are easily spread by crew coughing and sneezing airborne droplets; advise ill crew to sneeze/cough into the crook of the arm to avoid contaminating hands and surrounding surfaces. Viral and bacterial gastroenteritis can quickly spread by improper hand washing before leaving the head and prior to food preparation and eating. Poor hand washing facilitates spread of intestinal disease by contaminating cookware, dishes, utensils, and food. All crew should wash hands carefully with soap and water, followed by application of an alcohol-based hand sanitizer and air-drying the hands.

SEASICKNESS

Seasickness is the most prevalent medical illness for mariners at sea, and causes a significant number of maritime rescue operations, putting crew and rescue personnel at unnecessary risk. During stormy weather, mariners frequently consider seasickness a medical emergency and justification for medical evacuation. Each year, seaworthy yachts are abandoned because their exhausted and despondent crews have lost the collective will to persevere. In the words of a professional mariner, "They are wet, seasick, scared, and want to go home."

Anyone can develop seasickness with sufficient stimuli; however, individual susceptibility is variable. Only persons without a functioning vestibular system are fully immune. Pregnant women are highly susceptible, especially in the first trimester.

Seasickness involves a conflict of sensory input processed by the brain to orient the body's position. Someone positioned in the cabin of a heeling or rolling boat is inviting seasickness. Below decks, eyes oriented to cabin sole and ceiling detect no tilt from vertical, while fluid in the inner ear's vestibular system (semicircular canals and otolith organs) constantly shifts, sending neural messages that the head and body are not vertical. Position sensors (proprioceptors) in the neck, muscles, and joints send additional signals, depending on how a person shifts and secures himself or herself from falling. This mix of sensory data from eyes, inner ear, and position sensors arrives in complex and conflicting combinations, creating a "sensory conflict" that activates the emetic center in the brainstem. According to Dr. Charles Oman, Director of the Man Vehicle Laboratory at the Massachusetts Institute of Technology and an authority on motion sickness, the sensory conflict is also a sensory cue and "expectancy conflict." The expectancy conflict occurs when signals from the inner ear do not match expectations based on one's commanded self-movement, or concurrent visual or proprioceptive cues. When the motor cortex generates motor signals for movement, it also generates predicted sensory feedback of our motion. If there is a conflict in expected sensory input with actual movement (e.g., the effect of a rolling boat on our motion), an expectancy conflict develops that activates the autonomic and emetic centers, and symptoms of seasickness develop. If one eliminates these conflicts, one can prevent seasickness: if the eyes are seeing what the ears are feeling and what the brain is expecting, one has a good chance of experiencing a great day at sea.

Seasickness often presents with nausea and vomiting. Another facet of seasickness not often recognized is the *sopite syndrome,* which refers to profound drowsiness and persistent fatigue following provocative motion stimulation. Yawning has been shown to be a behavioral marker of the sopite syndrome; additional signs include boredom, sighing, pallor, dry mouth or salivating,

headache, dizziness, and lethargy. With sustained exposure to the stimulus, gastric emptying is inhibited. Subsequently, the hands and face sweat, becoming cold and clammy; belching, salivation, nausea, retching, and vomiting ensue. Some people experience headache, apathy, and depression. Moreover, seasickness impairs cognitive function. Sailors often lose the ability to multitask, making it difficult to analyze and integrate complex data, leading to poor reasoning, impaired judgment, and faulty decisions. Cognitive failure may also present as short-term memory loss. As seasickness becomes more severe, symptoms worsen, and include rapid mental, emotional, and physical deterioration marked by progressive dehydration, loss of manual dexterity, and ataxia.

Preventing and Treating Seasickness

Medication is more effective in preventing symptoms than in reversing them. Therefore, antiseasickness medication should be taken well in advance, before leaving port, or the night prior to departure. See discussion of medications below.

All voyagers should begin their journey well hydrated, well rested, and free of aftereffects of alcohol, which impairs vestibular function by sensitizing the vestibular apparatus to motion. Bland foods, such as crackers, fruit, trail mix, and popcorn, should be eaten throughout the day, even if one is not hungry, to maintain energy levels until meals are regularly tolerated. Drink small amounts of fluid frequently to avoid dehydration. Many sailors believe drinks with high amounts of vitamin C prevent seasickness; however, there are no clinical data to support this notion. Ginger is available in 250-mg capsules and sold in marine stores as Sailors' Secret. The suggested dose is 1 g every 4 to 6 hours. Foods containing lower concentrations of ginger, such as gingersnap cookies, ginger ale or tea, and candied ginger may be helpful. However, ginger may cause heartburn and even biliary colic in the setting of gallstones.

Both field and laboratory experiments have documented efficacy of acupressure in preventing seasickness. However, some experts consider acupressure no better than placebo. One sea trial showed that acustimulation suppressed symptoms of motion sickness. Pressure is applied on the Neiguan P6 acupuncture point on the forearm over the median nerve, found two to three fingerbreadths proximal to the wrist joint between the two prominent finger flexor tendons. There are commercially available elastic wrist straps with plastic studs that create pressure over the P6 point. The ReliefBand is an electric stimulator that operates under the same principle.

After departure, one can mitigate the symptoms of seasickness using the following multifaceted approach. Limit time below decks while underway to minimize sensory conflict. Stay on deck amidships (center) or aft (toward the stern), where motion is less severe. Maintain a broad view of the horizon, using both direct and peripheral vision; this provides a stable and level point of reference. Avoid close-focused visual tasks, such as prolonged reading and writing. Exposure to fumes (especially diesel) and odors may stimulate nausea. Sleep deprivation also increases susceptibility to seasickness. Take seasickness medication at the suggested intervals, and taper the dose after the first or second day.

At the first sign of seasickness, a direct remedy for many is to take the helm and steer, standing and feeling the waves, using clouds, the horizon, and distant marks as references, anticipating the boat's motion by "riding" the waves. "Wave riding" synchronizes sensory input and expectations of motion. One should keep the head, shoulders, and hips aligned to stay in balance and gracefully gain postural control. Sitting in the cockpit, one can still ride the waves and watch the horizon. "Postural anticipation of the boat's motion is the natural cure for seasickness," states Chuck Oman, who developed the concept of wave riding.

Seasick crew can easily fall or be washed overboard. They should always wear a safety harness on deck and be closely monitored. Seasick crew should not be allowed to move to lifelines to vomit overboard; a bucket should be readily available. In storm conditions, or if symptoms progress, the safest place to be secured is in the cabin, resting in a well-ventilated bunk, face-up with eyes closed and head still.

The antihistamines meclizine (Bonine) and dimenhydrinate (Dramamine) are available over the counter (OTC) without prescription. They are effective for preventing seasickness, as are the other prescription medications listed in Table 70-1. The popular antihistamine cinnarizine (Stugeron) is not sold in the United States but is available OTC in Europe, Bermuda, Mexico, and Canada (and can be obtained legally from online Canadian pharmacies). Many sailors favor it because it is less sedating than other antihistamines and has fewer reported side effects (described below).

Side effects of OTC antihistamines include drowsiness, dry mouth, blurred vision, irritability, urinary retention, dizziness, and headache. Meclizine (Bonine) causes less drowsiness and confusion. Antihistamines cause thickened bronchial secretions, and should be used cautiously in people with asthma and chronic obstructive pulmonary disease. An effective nonprescription drug for drowsiness is the decongestant pseudoephedrine, which is available in doses of 30 to 100 mg (immediate release, adult: 30-60 mg; sustained release, adult: 120 mg; maximum, 240 mg/day); caffeine 200 mg is also useful. Newer-generation nonsedating antihistamines are ineffective at preventing seasickness.

Parenteral antinausea medications include the phenothiazine-derivative promethazine hydrochloride (Phenergan). Promethazine is useful for prophylactic and active treatment of seasickness and can be administered as a suppository, by intramuscular injection, and orally as a tablet or syrup. Anticholinergic side effects include constipation, xerostomia, blurred vision, and urinary retention. Promethazine should be used with caution in persons with decreased gastrointestinal motility, urinary retention or obstruction, benign prostatic hypertrophy, xerostomia, or visual problems. Rare but serious adverse effects of promethazine include extrapyramidal reactions. The oral disintegrating tablet ondansetron (Zofran) is extremely effective for vomiting; however, it does not treat or prevent other symptoms of seasickness. Transdermal scopolamine hydrobromide (Transderm Scōp patch) is the most popular anticholinergic agent used for motion sickness prevention. Scopolamine prevents motion-induced nausea by inhibiting vestibular input to the central nervous system, inhibiting the vomiting reflex. The drug is delivered via an adhesive patch placed behind the ear at least 4 hours before departure; the patch will last for up to 3 days, often with minimal side effects. The scopolamine disk's integrity should be maintained, and not be cut or torn. Apply only one patch at a time. The administering person should be sure to wash his/her hands

TABLE 70-1 Medications for Seasickness

Medication	Dose	Interval
Diphenhydramine (Benadryl) (OTC)	25- or 50-mg tablet	6 to 8 hr
Dimenhydrinate (Dramamine) (OTC)	50- or 100-mg tablet (maximum 400 mg/day)	4 to 6 hr
Meclizine (OTC)	12.5- or 25-mg tablet (maximum 100 mg/day)	6 to 8 hr
Bonine (Meclizine) (OTC)	25-mg chewable tablet	6 to 8 hr
Cinnarizine (Stugeron)	15-mg tablet (maximum 100 mg/day)	6 to 12 hr
Scopolamine (Transderm Scōp)	1.5-mg skin patch	72 hr
Promethazine (Phenergan)	12.5-, 25-, or 50-mg tablet, suppository, deep intramuscular injection	Variable intervals, depending on dose/preparation

thoroughly after application; there may be temporary blurring of vision and pupillary dilation if drug residuum on hands contacts the eyes. The most common adverse effects of anticholinergic medications are dry mouth (66%) and drowsiness (17%). Other undesirable side effects include blurred vision (which may persist for weeks), dry mucous membranes, and short-term memory loss. Scopolamine is contraindicated in children, men with prostatic hypertrophy, and people with narrow-angle glaucoma; remove the patch immediately if a patient complains of the sudden onset of eye pain. Long-term use may produce withdrawal symptoms such as nausea, dizziness, headache, and equilibrium disturbances. Scopolamine in pill form (Scopace) is no longer available in the United States or Canada.

All therapies are subject to placebo effect, and there are no well-controlled trials comparing and evaluating different treatments for seasickness. It is not uncommon for one drug in a category (e.g., antihistamine) to be effective and a related drug to provide no benefit; the same is true for side effects. Medication side effects should be evaluated by trying different drugs while on shore. If all else fails, follow Samuel Johnson's 18th-century advice: "To cure seasickness, find a good big oak tree and wrap your arms around it."

MARINE WEATHER

THUNDERSTORMS AND SQUALL LINES

A single thunderstorm generally encompasses an area less than 3 km (2 mi) in diameter. The storm typically lasts approximately 30 minutes, with marked fluctuations in wind, temperature, and barometric pressure. The National Weather Service (NWS) considers a thunderstorm severe if it produces hail at least 2 cm (0.75 inch) in diameter, winds 93 km/hr (58 mph) or stronger, or a tornado. Of the estimated 100,000 thunderstorms that occur on inland and coastal areas in the United States, 10% are classified as severe. Eighteen hundred thunderstorms occur at any moment around the world, totaling 16 million a year. A squall line is a fast-moving row of violent thunderstorms, often more than 160 km (100 mi) long. An intense cold front 64 to 483 km (40 to 300 mi) behind the squall line contains most of the gusting winds and rain normally found in the front. A squall line is visible on both radar and to the naked eye, where it appears as a wall of boiling black clouds arising from the water.

Rapid growth of cumulus clouds is the primary indicator of a forming thunderstorm. The faster the clouds build, the more violent the resulting storm, because steep pressure gradients within the cloud generate high winds. Within the thunderhead are columns of rapidly sinking air called downdrafts. Downdrafts along the leading edge of the thunderstorm form the gust front. This zone of advancing cold air is characterized by a sudden increase in wind speed. Strong and highly localized downdrafts are called downbursts, the smallest of which are microbursts. Airplane pilots refer to these as wind shear. Downbursts are extremely intense concentrations of sinking air. On reaching the surface, they fan out radially in all directions, often generating winds at the gust front in excess of 150 knots. They are short-lived, typically lasting less than 15 minutes. A single thunderstorm can produce a series of downdrafts affecting an area several miles long, and persist an hour or more. Blowing spray under or slightly ahead of the storm may be the only indicator of its presence. A gust front often precedes a microburst. The combination of these two extremely strong and shifting wind systems can blow equipment and personnel off the deck and can easily capsize small craft and large sailboats.

In the United States, squalls occur predominantly during spring and summer in association with thunderstorms generated by towering cumulus clouds. The larger the cloud's size (and radar echo), the more wind potential within the cloud; the taller the cloud (especially above 6096 m [20,000 feet]), the more energy potential for stronger winds. Most squall formations occur at night when cloud tops radiate heat back into space, enhancing their ability to grow. Squall-generated winds rarely strike without warning. A rapid fall in temperature is the precursor of a local storm. The bigger the drop, the stronger the winds. Rain preceding wind suggests that stronger winds are coming. Occasionally, thunderstorms may form with the building of the cumulus cloud and not be associated with typical rain, thunder, and lightning. These violent winds are called white squalls. The only warning may be the sudden appearance of a cold, shifting wind with an increase in velocity.[2]

The inquiry into the 2011 Chicago Yacht Club Race to Mackinac reviewed the impact of a thunderstorm with powerful winds on the capsize of the 35-foot, ultralight, low-stability sailing vessel *WingNuts*. (The full report is available at ussailing.org/racing/offshore-big-boats/big-boat-safety-at-sea/safety-incident-reports/). When the *WingNuts* encountered a "wall of wind" with a speed of over 50 knots, it was blown over, capsized, and turtled. Six of the crew members, including one who was below decks at the time, were able to free themselves from the vessel; two who were unable to do so died as a result of head injuries and drowning. Weather and the boat's design were the dominant factors. The report issued the following weather description: "As they ran north at speeds in the high teens, the *WingNuts* team tracked the weather with several tools, including NOAA weather radio forecasts and WX weather overlays on the boat's GPS and radar. The forecast for Northern Lake Michigan area degraded throughout Sunday. A Severe Thunderstorm Watch was established at 1925 CDT, and subsequently the National Weather Service at Gaylord, MI, issued ever more urgent warnings." In a report produced after the race at the request of the Independent Review Panel, the Gaylord office described the developments this way: "During the late evening of July 17, a disorganized cluster of thunderstorms over Wisconsin and Upper Michigan moved into Lake Michigan and eventually evolved into a line of thunderstorms that crossed northern Lower Michigan. From a radar perspective, the storms were initially somewhat disorganized and marginally severe. As the cluster of storms progressed into Lake Michigan, however, one particular cell rapidly developed and intensified just prior to midnight EDT [2300 CDT]." During this intense thunderstorm, 37 other boats were knocked down but recovered upright.

Large sailboats, such as the clipper topsail schooner *The Pride of Baltimore,* have also been knocked down and sunk by these unexpected powerful winds. In May 1986, a sudden storm rumbled across the Atlantic and unleashed its strength over a small patch of the Bermuda Triangle. With furious precision, its 70-knot winds overwhelmed the 97-ton, 34-m (110-foot) clipper ship heading home from Europe; survivors reported that the ship immediately barrel-rolled in the heavy wind and sank. Daniel S. Parrott's book *Tall Ships Down* provides more information on the loss of *The Pride of Baltimore,* as well as four other traditionally rigged vessels, the *Pamir, Albatross, Marques,* and *Maria Asumpta*.

Sources of Marine Weather Information

To avoid or prepare for a thunderstorm, sailors must monitor local weather conditions. The National Oceanographic and Atmospheric Administration (NOAA) transmits on VHF-FM radio recorded messages, which are repeated every 4 to 6 minutes, and updated every 2 to 3 hours with the latest local information. Broadcasts usually can be received 20 to 60 miles from transmitting antennas. Stations are identified on the radio channel display as WX-1, WX-2, and WX-3. Nationwide, more than 860 transmitters provide coverage to all 50 states and adjacent marine areas. In many areas, the Coast Guard broadcasts weather information on VHF channel 22. Listings of schedules and frequencies for coastal and offshore weather broadcasts are available in a number of publications, including the *Admiralty List of Radio Signals,* volume III, and *Reeds Nautical Almanac*. Box 70-7 lists the definitions of storm warnings.

Many newer VHF radios have a weather-alert function known as Weather Watch. When the radio receives a warning signal from NOAA, it sounds a special alerting tone, signifying an urgent NOAA weather forecast. Some radios automatically tune to the active weather channel, whereas others require manual tuning. NOAA improved their severe weather warnings by encoding both the area affected by severe weather and the nature of the severe weather (e.g., hurricane, winter storm, high wind, severe

BOX 70-7 Storm Warnings

1. Small craft advisory: generally associated with sustained winds of 18 to 33 knots (30.5 to 61.2 km/hr [19 to 38 mph]) and/or sea conditions dangerous to small boats.
2. Gale warning: sustained winds of 34 to 47 knots (62.8 to 86.9 km/hr [39 to 54 mph])
3. Storm warning: sustained winds of over 48 knots (88.5 km/hr [55 mph]). If winds are associated with a tropical cyclone, the storm warning display indicates forecast winds of 48 to 63 knots (88.5 to 117.5 km/hr [55 to 73 mph]).
4. Hurricane warning: sustained winds of 64 knots (119.1 km/hr [74 mph]) and greater as the result of a tropical cyclone
5. Special marine warning: winds of 35 knots or more generally lasting less than 2 hours. These are usually associated with an individual thunderstorm or an organized series of thunderstorms, as in a squall line.

thunderstorm). Called Specific Area Message Encoding, this feature is intended to make weather warnings more customized so that users will not tune out possibly lifesaving warnings. Offshore sailors can receive a variety of high-seas marine weather broadcasts with single-sideband (SSB) high-frequency (HF) radios. The U.S. Coast Guard broadcasts NWS high-seas forecasts and storm warnings from each coast. All broadcasts are upper single-sideband HF, with additional broadcasts on medium frequency (MF). The time schedules and frequencies for these transmissions can be found in the National Geospatial-Intelligence Agency (NGA) Publication 117, titled *Radio Navigation Aids*. A less comprehensive list for specific locations can also be found in *Reeds Nautical Almanac*. For schedules and much more information on NWS marine products, visit NOAA's National Weather Service Marine Forecasts at web page nws.noaa.gov.

The U.S. Coast Guard sends a facsimile of a weather chart (weatherfax) from their East, Gulf, and West Coast communication centers. These marine weather charts are updated every 6 hours and available within 3.5 hours of the valid update time. Weatherfaxes contain graphic charts and forecasts compiled by the NWS; they are broadcast on HF radio bands between 3.5 and 30.0 MHz. Lists of transmitters, frequencies, and times can be found in *Reeds Nautical Almanac* or online at NOAA's page listed above. This site contains text forecasts, ocean current and surface analysis charts, wave charts, buoy reports, and prognosis charts (500-millibar [mb] charts) for weather outlooks of 12, 24, 36, 48, and 96 hours.

Software can integrate weather forecasting with optimal routing for safety and sailing performance. Weather files (called GRIB files) can be downloaded and overlaid onto an electronic chart, and routing formulas can then be applied incorporating the boat's known performance characteristics. Mariners can obtain sea state and weather data directly from NOAA buoys moored offshore by using the Internet. The National Data Buoy Center website is ndbc.noaa.gov; the website lists more than 1200 buoys around the world that one can find on the site's world map to obtain real-time observation of weather conditions.

NAVTEX is a worldwide land-based radio navigation warning service, transmitting text-only messages (in English) to a dedicated onboard receiver. The unit can be programmed to receive both specific stations and message categories; it can print out area weather forecasts, gale warnings, navigation warnings, ice warnings, and relayed distress messages on the assigned frequency of 518 kHz. Vital messages (e.g., gale warnings, SAR information) activate an alarm in the receiver as the message is being printed.

Traditional Weather Forecasting Methods

Even without expensive electronic equipment, mariners can predict changes in local weather by using their own observations. All crewmembers should learn the cloud types or augment their knowledge base through use of a photo atlas of cloud formations, to identify the variety of clouds and their significance. During the day, a sailor can learn to recognize a squall line, growth of a cumulus to a cumulonimbus cloud with its characteristic anvil head on the downwind side (a thunderhead), and the cloud sequence and wind changes of an approaching cold and warm front. "Mare's tails and mackerel scales; soon it's time to shorten sails," refers to cirrus and cirrocumulus formations (long puffy clouds at high level, commonly known as a mackerel sky). This cloud formation signifies unsettled weather, with the approach of a warm front. Two weather concepts can be helpful for predicting changes in weather: the crossed-winds rule, popularized by the meteorologist Alan Watts, and Buys Ballot's law. The crossed-winds rule states that whenever the upper-level wind flow (determined by observing the direction of the cirrus clouds at 6096 to 9144 m [20,000 to 30,000 feet]) and lower-level surface winds are crossed, the weather is going to change. Buys Ballot's law states that in the Northern Hemisphere, if you stand with the wind blowing at your back, a high pressure will be at 90 degrees to your right, and a low pressure 90 degrees to your left (remember "low-left"). If application of the crossed-winds rule and Buy Ballot's law indicates a low is approaching, the weather will certainly deteriorate into a warm front, with increasing winds and rain. Once past, a cold front follows. If the upper-level flow either matches or directly opposes the surface wind, weather is likely to remain stable for a while.

During daylight, a band of low, dark, and smooth tubular roll clouds can often be seen at the leading edge of a squall, preceding a cold front. The faster a roll cloud approaches, the stronger the wind is likely to be, and the more agitated the sea appears under it. When the sea is observed beneath the roll cloud, the cloud is about 2 miles away; because clouds typically move at about 40 km/hr (25 mph), the face will arrive in about 5 minutes.

A barometer is one of the most important weather instruments onboard a small boat. By 1 to 4 hours before an approaching thunderstorm, there is a sharp drop in barometric pressure of about 1.5 mb. If the atmospheric pressure is fluctuating very little, it means weather is likely to remain stable. A pronounced rise in pressure heralds fair weather.

The sea state is a valuable clue to approaching weather patterns. Large ocean swells precede a heavy weather system, whereas chop without swells often reflects a local, more isolated, and temporary disturbance. An alert observer can calculate the distance between a vessel and a thunderstorm within earshot. The distance of lightning from an observer can be determined by noting the time between the flash and the bang of associated thunder. For each 5-second count from flash to bang, lightning is 1.6 km (1 mi) away. Lightning is discussed in more detail in the following section and Chapter 5.

In order to meet the challenge of finding the most reliable, accurate weather information and mastering the art and science of weather forecasting, a sailor departing on an extended coastal cruise or ocean passage anywhere in the world can contract with a professional weather routing company; these services provide assistance in determining a favorable weather window for departure, and continued support for weather forecasting and routing during passage. Such consultation provides an invaluable additional layer of safety and confidence.

LIGHTNING

Lightning is one of nature's most destructive phenomena (see Chapter 5). Boating, fishing, and swimming rank second only to playing sports on an open field as the most dangerous activities associated with lightning strike. A cruising sailor in Florida, the lightning capital of America, can expect at least one strike to his or her boat in its lifetime.

Lightning protection systems do not prevent lightning strikes. They may, in fact, increase the possibility of the boat being struck. The purpose of lightning protection is to reduce the damage to the boat and the possibility of injuries or death to crew from lightning.

Tall and narrow objects, with highly charged and focused electric fields, are likely to attract lightning. Metal itself does not attract lightning. A sailboat mast, radio antenna, fishing outrigger, fishing rod, and even crew standing on deck are all good targets.

In a marina or anchorage, the boat with the tallest mast is usually the most vulnerable to a strike.

The most critical factor initiating the lightning streamer emanating from the boat is not only the mast height but also its electric potential. The crackling bluish-green light sometimes seen in a ship's rigging at night during thunderstorms is not lighting but a type of electrical discharge (corona discharge) called St Elmo's fire. It may even appear like a stream of fire as it trails from the mast. Magellan's storm-battered crew regarded the "fire" as a sign of divine protection by St Elmo, the patron saint of mariners. Captain Ahab saw St Elmo's fire and reassured his ill-fated crew aboard the whaler *Pequod*, "The white flame but marks the way to the white whale."

St Elmo's fire occurs when there is a large difference in electrical charge between the mast and surrounding air. This causes air molecules to be split apart by the voltage streaming off the mast, and resulting gas begins to glow. Do not climb the rigging for closer inspection, because there are 30,000 volts per centimeter of space surrounding the masthead.

Lightning is too erratic and unpredictable for full protection to be possible. Many experts advocate providing an adequate conductive path from the masthead through to the water by the shortest, most direct route possible, using an elaborate bonding system. All wire rigging and large metal objects should also be connected to an underwater ground plate. This is especially important for boats constructed from wood, composites, and fiberglass; these nonconductive hulls impede the passage of electrical charge to the water. Steel and aluminum hulls with traditional aluminum spars are excellent conductors of electricity and can easily carry electrical charges to the ground. The objective of bonding is to prevent injury to the crew, catastrophic damage to the boat, and severe damage to electrical systems and electronic equipment. Even with the best system, this is not always accomplished. Without grounding, a bolt of lightning will find its way to the sea from the base of the mast, usually through some part of the hull.

Mitigating the Damage from Lightning

The best advice on preventing and mitigating lightning strikes is to follow the practices recommended by the American Boat and Yacht Council for both lightning protection and grounding. At a minimum, the mast should be fitted with an air terminal. This consists of a solid 0.375-inch copper or 0.5-inch aluminum rod attached to the top of and extending at least 6 inches above the vessel's mast. Its skyward tip should be rounded. The path to the ground must be a highly conductive material of low resistance so that current passing through will not create heat sufficient to melt the conductor. The bonding system must be complete. Half measures may invite massive electrical charges into the boat and then fail to provide a safe path to the water; it would be preferable to remove the half measures and sail without a bonding system (other than one exclusively for electronics). Copper wire with a minimum of 4 American wire gauge (AWG), not 8 AWG as previously suggested, is required in saltwater, and a ground plate (with sharp rather than round edges), ideally made of solid copper or bronze with a dimension of at least 930 cm^2 (1 $foot^2$), is recommended. The ground plate should be located as close to the mast as possible on the bottom exterior of the underwater hull.

Lead keels on sailboats make excellent ground plates only when properly connected to the base of the mast and only if not encapsulated with fiberglass. Some motor and auxiliary sailboats use the exposed surface of the engine prop and shaft as a ground plate. With engine grounding, damage may occur from the heat generated by a powerful strike. All masts constructed of wood or other nonconductive materials (e.g., carbon fiber with epoxy resins) require wire or a solid copper strap from the masthead to the ground plate.

Lightning can also generate a side flash, which is the secondary flow of current from the charged area to some object near the path of the strike to the water, especially dangerous to crew who are accidentally nearby. Simple grounding of the mast to the ground plate prevents major hull damage but does not prevent side flashes. As current follows a designated path to the ground, another electric potential is created between the ground system and the objects surrounding it. The entire boat becomes high voltage, and the secondary electric current, called the side flash, is created.

The oft-quoted wisdom that a 45-degree cone-shaped zone of protection is created under the mast is false. Lightning has been known to directly penetrate this supposed area of safety, and a person can still be electrocuted by voltage along the deck surface or by side flash if the mast is struck. The key to preventing secondary current flows is to equalize the voltage of all metallic objects onboard by establishing a common electrical ground for the entire boat, that is, a complete bonding system. Any area capable of collecting a large static charge, including each piece of metal equipment onboard, all electronic instruments, and radio equipment, must be bonded to the same discharge system used to protect the boat from effects of the initial lightning strike. This is accomplished by connecting No. 6-gauge copper cable from all metal objects to the common ground. This includes shrouds, stays, tanks, the rudder, engine blocks, electric winches, pulpits, pedestals, arches, radar masts, and seacocks. Switching off or disconnecting electronic equipment is advised, but will not necessarily protect it. Portable electronics, computers, handheld GPS units, and radios can be placed in the galley oven, which acts like a Faraday cage, or in a designated grounded metallic box. Everything electronic that uses modern microprocessors is vulnerable in an electrical storm. A new way of bonding the boat recommends grounding the mast at deck level to a continuous loop outside the cabin, connected to multiple cables down to the grounding plate and supplemental electrodes near the water line to further disperse electrical charge. This method is still in early stages of development.

Crewmembers may become part of the current path if they are in contact with, or come between, two different metal objects that are not connected (e.g., by grasping the stanchion or rigging while holding an aluminum steering wheel). The best protection in the event of a direct lightning strike, even if the boat is grounded and bonded, is to remain low in the boat (preferably in a cabin and dry) and away from shrouds, the mast and other metal objects, wiring, and electric conductors. Crew should refrain from using all electrical equipment (Box 70-8).

OTHER WEATHER PHENOMENA AT SEA

Waterspouts

Waterspouts are maritime tornadoes. Although less common than lightning and downbursts, they are generated by the same dynamic forces found in the squall line at the leading edge of an advancing cold front or in a rapidly building summer afternoon thunderstorm. The danger of a waterspout lies in the powerful revolving winds, which may exceed 400 km/hr (250 mph), and the very low pressure at its center, which may cause tightly enclosed spaces to explode. Waterspouts are visible during the day, when most of them occur, and can also be located and tracked by radar. The average forward speed is 50 km/hr (30 mph) but may vary from nearly stationary to 120 km/hr (70 mph). An area of turbulent water in the distance is the earliest visible sign of a waterspout; as it approaches, spray rises upward and joins the funnel cloud with its characteristic snakelike, gyrating appendage. Waterspouts usually last only 30 to 60 minutes. Preparation of the boat and crew is similar to that for a thunderstorm. Because a waterspout is relatively narrow, steering a course perpendicular to its projected path (the direction the clouds are going) is a logical avoidance maneuver.

Hurricanes and Cyclones

A hurricane is a type of tropical cyclone—an organized rotating weather system that develops in the tropics (see Chapter 107). Tropical cyclones are classified as a:

- Tropical depression: an organized system of thunderstorms with closed low-level circulation and maximum sustained winds of 33 knots (38 mph) or less.
- Tropical storm: an organized system of strong thunderstorms with well-defined circulation and maximum sustained winds of 34 to 63 knots (39 to 73 mph).

BOX 70-8 Protecting Crew from Lightning Injury

1. Get out of the water. If possible, get off the water and away from the water.
2. Get off the beach. If stranded, squat down and place legs and ankles together. Keep the surface area of the body in contact with the ground to a minimum. Spread people out to maximize the possibility that some will survive a lightning strike.
3. If possible, get off the boat, including a boat at anchor or in a marina.
4. Remove wetsuits and other wet clothing. Put on dry clothing and foul weather gear, hat, boots, and a personal flotation device. Wet bodies make good electrical conductors, but wet foul weather gear may provide protection by lowering surface resistance and guiding the current around the body.
5. Remove all metal articles, especially jewelry, scuba tanks, and weight belts.
6. Avoid direct contact with all metal objects, including handrails, engine, stove, rigging, and spars.
7. Do not touch any of the boat's installed electrical equipment, including navigation instruments and radios. Use a handheld VHF radio for emergency communications during the storm.
8. Do not simultaneously touch two metal objects, such as the engine throttle and spotlight handle.
9. Stay away from the mast, stays, shrouds, and wet sails.
10. Stay out of areas where bridging between two highly charged areas, or side flashes, may occur. These areas include the foredeck, between the mast and the head stay, or a seat between an outboard engine and the portable metal fuel tank or steering wheel.
11. If caught in a storm while on a sailboard, lower the sail and mast and sit down on the board.
12. If fishing or trawling, stop and lay the rods and trawls horizontal in the boat.
13. Put nonessential crew and passengers below decks, in the center of the cabin. Stay out of the companionway, engine room, and head. If there is no cabin, stay low in the boat.
14. If possible, put the boat on autopilot to minimize contact with a metal wheel.

- Hurricane: an intense tropical weather system with well-defined circulation and sustained winds of 64 knots (74 mph) or higher.

The Saffir-Simpson Hurricane Wind Scale is a 1 to 5 rating based on the hurricane's intensity. Category 1 has sustained winds up to 153 km/hr (95 mph), and category 5, sustained winds in excess of 250 km/hr (155 mph). In the western North Pacific Ocean, hurricanes are called *typhoons* (super typhoons have sustained winds exceeding 241 km/hr [150 mph]) and in the Indian Ocean, *cyclones*. On average each year, 10 tropical storms, 6 of which become hurricanes, develop in the Atlantic Ocean, Caribbean Sea, or Gulf of Mexico.

The center (eye) of a hurricane is relatively calm. The most violent winds and rainfall are found in the eye wall, a ring of thunderstorms 15,240 m (50,000 feet) high. Coastal sailors seeking harbors of refuge from the destructive wind and sea still have to prepare for the greater threat of the storm surge; this is a large dome of water, often 80 to 160 km (50 to 100 mi) wide, that sweeps across the coastline where the hurricane makes landfall. The surge of high water, topped by huge waves, is devastating. In August 2005, Hurricane Katrina flooded and destroyed cities of the Gulf Coast with its 6-m (20-foot) storm surge. In October 2012, Hurricane Sandy became the largest Atlantic hurricane on record (as measured by diameter, with winds spanning 1800 km (1100 miles). Its storm surge hit New York City on October 29, flooding streets, tunnels, and subway lines and cutting power in and around the city.

When given the choice, some sailors head out to sea, risking their lives to save their boats. This may be a reasonable strategy for a battleship but is rarely a prudent decision for an offshore sailing craft. The best tactic for dealing with the fury of hurricanes at sea is to avoid them. The HMS *Bounty* (originally built for the 1962 film *Mutiny on the Bounty*) sank in the Atlantic Ocean off the coast of North Carolina after attempting to outrun or navigate around a massive hurricane. The vessel had started taking on water, its engines failed, and the crew of the stately historic craft had to abandon ship as it went down in the immense seas. The captain and one crewmember perished, and the remaining crew were rescued from life rafts by helicopters. The ship left New England just prior to the arrival of the storm. Ironically, one of the last posted messages on Facebook said: "Rest assured that the *Bounty* is safe and in very capable hands. *Bounty*'s current voyage is a calculated decision … NOT AT ALL … irresponsible or with a lack of foresight as some have suggested. The fact of the matter is … A SHIP IS SAFER AT SEA THAN IN PORT!"

In the Northern Hemisphere, wind blows counterclockwise around the eye of a hurricane. Facing into the wind and stretching the right arm back 120 degrees will point at the eye. In the Southern Hemisphere, wind blows clockwise around the eye. Facing into the wind and stretching the left arm back 120 degrees will point to the eye. In the Northern Hemisphere, the strongest winds are to the right side of the hurricane's path, where the forward speed over the water adds to the local wind speed; this is the more dangerous semicircle. In the Southern Hemisphere, the strongest winds are on the left side of the path. Although recommendations include placing the boat in the so-called safe or navigable semicircle and avoiding the strongest winds surrounding the eye of the hurricane, the safest course is to stay out of its path entirely.

Hurricane track forecasting has acknowledged limits and errors, and the U.S. National Hurricane Center is not infallible. Hurricanes are inherently unpredictable, even when the best computer models are used to predict the path. In October 1998, when category 5 Hurricane Mitch (the fourth-strongest Atlantic basin hurricane in recorded history) slammed into Honduras, the 82-m (286-foot), four-masted, steel-hulled *Fantome* was doomed. Despite every evasive action taken by the experienced captain (based on the updated forecast track), the massive hurricane swirled menacingly into his path and eventually sank the cruise ship with loss of the entire crew. The plots of the ship's daily locations off the coast of Central America and those of the hurricane exactly overlap each other, as though Mitch were actively chasing the ship before finally devouring it.

The expected track error for a hurricane is 160 km (100 mi) on either side of the predicted track for each 24-hour forecast period. For a 72-hour forecast, an error of 480 km (300 mi) to the left or right of the predicted forecast track can be expected, and for a 96-hour forecast, 650 km (400 mi) to either side is applied. That would make the storm's potential swath for destruction 800 miles wide, a considerable area to avoid if a vessel can only travel at 6 to 7 knots in storm conditions. In order to take meaningful evasive action, a hurricane needs to be monitored at least every 6 hours, the official forecast interval for the NOAA/NWS Storm Prediction Center. The National Hurricane Center in Miami, Florida, provides advisory updates on developing tropical storms and hurricanes 24 hours a day online at the National Hurricane Center website: nhc.noaa.gov.

SEA CONDITIONS AND BREAKING SEAS

The U.S. Sailing Farallones Panel Report of *Low Speed Chase* Capsize issued in July 2012 explores the danger of sea conditions in shoal waters (Figure 70-21) (see ussailing.org/racing/offshore-big-boats/big-boat-safety-at-sea/safety-incident-reports/ for the *Low Speed Chase* reports and other incident reports involving sailboats). "On April 14, 2012, at 14:36:40 PDT, while racing in the Full Crew Farallones Race out of San Francisco, CA, the sailing vessel *Low Speed Chase,* with eight crew aboard, encountered breaking waves when rounding Maintop Island, the northwest point of SE Farallon Island. The vessel, a Sydney 38, was less than 0.2 nautical miles (400 yards) from the point, crossing a 4-fathom shoal at near-low tide in a 25-knot northwest wind on a heading of approximately 235° magnetic. The morning forecast predicted 'wind waves 3 to 7 feet, NW swell 12 to 15 feet at 13 seconds.' A set of larger than average waves capsized

FIGURE 70-21 Breaking seas can occur when the depth of the water is less than 2.5 times the significant wave height. Waves may break very infrequently, lulling skippers into a false sense of security. Know wave heights and depths.

the boat and drove it onto the rocky shore. Seven of the eight crew members were thrown into the water. Two of those in the water made it to shore and survived, but five did not. One of the survivors rode the boat to shore. Whether or not a wave will break in shallow water depends on the size and steepness of the wave, water depth, and shape of the bottom contour. Wavelength and period are related, and the wavelength (L) of a wave with a 14-second period is 1000 feet. Depths over half of that (L/2 or 500 feet) are considered "deep water," and depths of less than L/20 (50 feet) are considered "shallow water" for wave dynamics.

Waves slow down as they reach shallow water and "feel the bottom," but the period is unchanged, so the kinetic energy is converted into the potential energy of a slower, taller wave. This increases as the depth decreases until the wave becomes unstable and breaks. If this process happens gradually (e.g., a gently sloping beach), then the waves will form "spilling" breakers, with the water tumbling down the sloping face of the wave. If the breaker forms quickly (e.g.., a faster-moving wave and a more steeply sloping shoreline), then a "plunging" breaker forms, with the face becoming vertical, curling, and then collapsing into the trough.

In storm-tossed seas, expect larger, more dangerous waves; the height varies, and some can be double the height of average waves in a given area. Strong tidal currents, common near inter-island passages, inlets, canal exits, and river mouths, can interact with wind-driven waves to produce high waves and perilous conditions. The same concept applies to adverse currents. When a current's speed approaches or exceeds 25% of the speed of oncoming wind-driven waves, the current stops the wave energy from moving forward; the wave energy builds vertically until the steep waves begin to break, endangering small craft. Current and tide tables should be checked in the regional *Nautical Almanac* in order to calculate the optimal time for safe passage by coordinating local weather with the predicted currents. Sailing parallel (beam-to) to high breaking seas (see Figure 70-21) should be avoided; the curl of a plunging breaker can easily capsize a boat. Capsizing may occur in seas that are not exceptionally high. On December 21, 2004, the 23-m (75-foot) *Northern Edge,* with six fishermen aboard, capsized off the New England coast in a snow squall with 5-m (15-foot) seas and 56-km/hr (35-mph) winds (an average North Atlantic winter storm). The lone survivor said, "We were dredging (for scallops) … then came a wave and the boat was hit broadside, and it just flipped." The bodies of the other five fishermen were never recovered from the 4°C (39°F) water after the U.S. Coast Guard searched a 4791-km^2 (1850-mi^2) area for more than 40 hours. None of the fishermen was wearing a survival suit. According to a preliminary investigation, the watertight door to the engine room and forward compartment was open, but scuppers to the main deck (openings along the deck that allow water to escape) were closed.

BOX 70-9 Visual and Sound Distress Signals at Sea

1. If other vessels are in sight, stand in an unobstructed area and slowly and repeatedly raise and lower outstretched arms as though you are flapping wings.
2. Sound a foghorn (SOS: 3 short blasts, then 3 long, then 3 short), ring a ship's bell, or blow a whistle continuously.
3. At night, point a flashlight at another boat and flash SOS (dot dot dot, dash dash dash, dot dot dot) repeatedly.
4. Fly the ship's ensign upside down, or fly the recognized distress flag: an orange flag with a black square under a black ball. Any square flag with a ball shape above or below is also a recognized distress signal.
5. Wave any brightly colored clothing or foul weather gear attached to a paddle or pole.

EMERGENCY COMMUNICATIONS AND DISTRESS SIGNALS

VISUAL AND SOUND DISTRESS SIGNALS

The simplest signaling strategies and devices are often overlooked. Box 70-9 lists some simple signaling techniques. Pyrotechnic distress signals have a replacement interval of 42 months from date of manufacture and are labeled with the expiration date. Expired flares can be kept onboard as spares, but will not be counted in the vessel's required inventory if boarded. The U.S. Coast Guard requires all boats over 16 feet on U.S. waters to carry three visual distress signals for day use and three for night use.

Handheld flares have varying effectiveness for attracting attention during daylight (Figure 70-22). Luminosity ratings range from 500 to 15,000 candlepower. The lowest-rated flares are virtually invisible in daylight at 0.4 km (0.25 mi), and the highest-rated flares are only slightly more visible. For daytime use, orange smoke devices are the most effective way to attract attention. SOLAS-graded smoke canisters are superior to handheld smoke flares. They float and emit orange smoke for 3 to 4 minutes. Immediately after ignition, the canister should be hurled in the water downwind of the craft. All orange smoke devices have a few seconds of delay in activation; direct inspection of the pyrotechnic device should never be performed after ignition. Smoke signals have a visible range of 1.6 to 5 km (1 to 3 mi) in daylight, depending on wind. Helicopter pilots find smoke signals readily visible, and in addition, smoke often conveys the strength and direction of the wind at sea level (Figure 70-23).

FIGURE 70-22 SOLAS red hand flare during the day. The smoke may attract more attention, but at night the red light is unmistakable.

FIGURE 70-23 Coast Guard rescue swimmer with a small handheld smoke flare. This instantly shows the helicopter crew the wind direction.

After sunset, a flare's luminosity, burn time, and attained altitude are all considerations relevant to use. An aerial flare (meteor or parachute) should be used to attract attention or alert a ship or an aircraft at night, followed by a handheld flare to guide rescue craft to the distressed boat's location (Figure 70-24). Because of the curvature of the Earth, sighting distances are limited. A high-altitude flare can alert a ship over the horizon from the crippled vessel. The greater the height, the longer and farther the signal can be seen by a distant viewer. A parachute flare at 305 m (1000 feet) is seen as a brief flash of light on the horizon from 64 km (40 mi) away, but from 32 km (20 mi), it appears to be 152 m (500 feet) above the horizon and is visible longer. Luminosity and burn time are more important than altitude in helping craft to home in on the ship's location. The rated visible range assumes a ship in the area has an alert lookout standing on the bridge watching for and anticipating the signal, on a clear night with calm seas.

Whenever possible, flares should be chosen that meet SOLAS (International Convention for Safety of Life at Sea) standards. There are three SOLAS pyrotechnic types: a red hand flare that burns for 60 seconds at 15,000 candela (Figure 70-25); a red rocket parachute flare that soars to 305 m (1000 feet) and lasts for 40 seconds at 30,000 candela; and a 4-minute smoke canister

FIGURE 70-24 SOLAS pyrotechnics do not require a launcher, but some recreational flares do. Do not drop it overboard, or it could render the rest of your signals irrelevant.

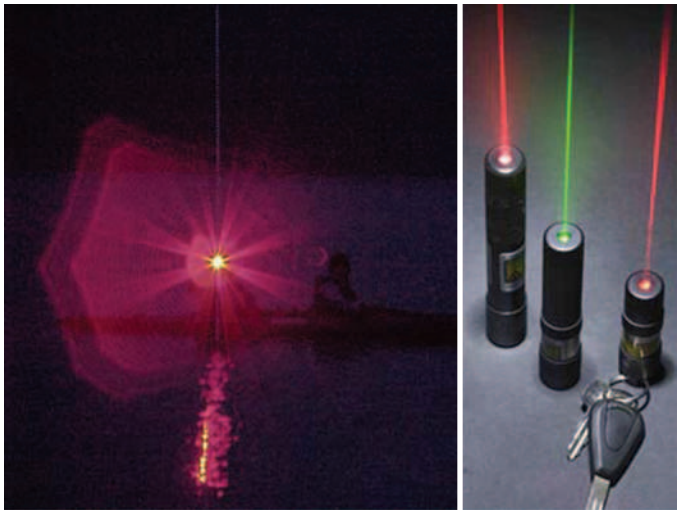

FIGURE 70-25 Although using a laser is exceedingly effective, Coast Guard members will abort a mission if they determine that their pilots have been "illuminated" by a laser. This is a shame, because laser flares are compact and have an exceedingly long range. However, it is not worth the risk to pilots if they are "targeted" with a laser.

(see Figure 70-23). All three are self-contained, waterproof, and found on commercial ships and life rafts, as well as on racing sailboats participating in offshore races.

All pyrotechnics are hazardous, capable of melting life-raft rubber and burning skin. Handheld red flares may drip considerable ash and slag, and should be held high, at arm's length, away from the raft or boat deck, and over the water. Crewmembers should be in a stable position when igniting a flare. All adhesive fastenings and covers on the flare should be removed and the device properly oriented after identifying the rocket or flare exit end. Some flares are activated by pulling a lanyard from the "business" end, while others are activated by pulling a lanyard from the "handle" end. After the signal is activated, shield the user's eyes. If a rocket or flare fails to ignite immediately, it may not ignite at all or there may be a lag time before firing. The flare should be held in a safe position for 60 seconds; if there is still no ignition, the flare should be thrown into the sea. Once activated, the flare should be treated as a live explosive, and should not be visually inspected or put back into the raft.

Red rocket parachute flares are designed to attract attention of potential rescuers out of sight over the horizon. They are the brightest, longest-burning aerial distress signals. For practical purposes, a parachute flare has a useful range of about 16 km (10 mi), regardless of its rated range or altitude. These flares drop slowly beneath a nonflammable parachute and burn for 40 seconds. The SOLAS models burn four times brighter and up to 50% longer than Coast Guard–approved models. Red is the standard distress signal, but a white parachute flare provides far more illumination than does a red one, especially if a collision is imminent. The main body of the rocket is contained within the launch tube. After the firing mechanism is activated, the flare tube should be held in both hands using gloves or a cloth to prevent the tube from slipping because of its powerful recoil.

Red meteor (aerial) hand-launched or pistol-launched flares (see Figure 70-23) should also be launched downwind approximately 80 degrees above the horizon. These flares are less effective than parachute flares because the burn time is only 5 to 9 seconds, and they are best used by firing in sequence. The first flare is fired to attract attention and the second about 10 seconds later to confirm distress and general position. Because of their lower altitude and brightness, visibility is limited to 5 to 8 km (3 to 5 mi) at night, and with these limitations, these flares are best suited for guiding nearby rescuers to the boat after it has already been spotted, or in inland waters where the distances are shorter.

NONPYROTECHNIC SIGNALS

The fullest effort should be made to use inexhaustible signal resources, such as signal mirrors, flashlights, or kites, before using flares. Rescue lights are no longer recommended because of hazard to airline pilots and rescue personal. The bright beam of the laser light (see Figure 70-25) may force a pilot to head back to base, unable to finish the search, and poses a potential hazard to injuring the pilot's eyes and vision. Pilots and aircrew require an eye examination before returning to duty after laser exposure. This is unfortunate because laser "flares" are visible over a long distance, have excellent duration, and are very compact. Pyrotechnic flares should not be fired in the direction of high-flying commercial aircraft; pilots and crew cannot see low-altitude flares and other signals when they are flying at 9144 m (30,000 feet). Flares should be used sparingly, and not all expended in an attempt to signal the first passing ship or low-flying aircraft, which might pass close by without seeing the signals.

Signal mirrors are inexhaustible devices and more effective than flares in daylight. Mirrors are especially useful when trying to attract the attention of aircraft, even those flying at high altitudes. The signal should be directed at the airplane when it is still in the distance and approaching the vessel. Signal mirror flash can be seen at a distance of up to 64 km (40 mi) from the air on a clear day. Mirror signaling should start as soon as a plane is audible, because flashing light may be visible to passing aircraft before a plane comes into view. Mirrors are also useful at night, where they can be used to increase vessel conspicuity by reflecting back a ship's searchlight, making the raft or ship easier to locate in the dark. Improvised reflective surfaces can be fashioned from materials such as compact discs, foil space blankets, jewelry, eating utensils, fishing lures, and credit cards. See Box 70-9 for other signals.

Coast Guard rules state that every boat must carry a device capable of producing a 4-second blast audible from 800 m (0.5 mi). This requirement can be met with chemical propellant–powered horns, lung-powered horns, or whistles. Plastic horns threaded into a metal canister often malfunction; spare parts and propellant should be kept onboard. Most handheld horns are barely audible at half a mile, and are inaudible at three-quarters of a mile. The same is true for lung-powered horns and whistles, which are barely audible at 400 m (0.25 mi). In the absence of a working sound device aboard ship, percussion of metal objects such as a spoon or winch handle against a pot can be used to sustain a continuous, albeit crude, bell-like sound.

Rescue ships and aircraft use radar to locate a life raft or vessel in distress. Commercial life rafts and lifeboats increase their visibility on the radar screen by using an electronic radar reflector known as a search and rescue transponder (SART). Once activated by an incoming radar signal (the interrogating radar), the SART is capable of sending back an enhanced electronic signal to any commercial radar located on a ship within a 16-km (10-mi) radius and up to 80 km (50 mi) on the radar screen of an aircraft flying at 914 m (3000 feet). It is much more effective than a simple radar reflector. Carbon sails also make excellent radar targets for planes and rescue craft, and should be left up at least partially, or deeply reefed, for as long as possible.

Commercially available yacht radar reflectors have variable performance.* Sailors have tales of having been seen at great distances when using their favorite radar reflector, but it is more likely that a small vessel with radar will see a ship than that a ship will see a small vessel with a radar reflector. The effectiveness of many radar reflectors varies with the fourth power of their radius, so larger reflectors are dramatically more effective. Small aerodynamic radar reflectors have virtually no effectiveness, even if mounted near the masthead.

Fluorescent dye marker can be seen by airplanes and has a daytime visibility of 3 to 8 km (2 to 5 mi). In moderate weather, dye lasts for approximately 30 minutes. It dissipates far more quickly in rough seas. The ideal time for using dye is just prior to an anticipated aircraft arrival or in a known flight path. A sea anchor will keep the drift rate of the boat similar to that of the dye.

Handheld waterproof personal safety lights offer portable, compact signaling and can flash for hours on a single replaceable battery. New LED models last far longer on a set of AA batteries and are less expensive than the xenon strobes they have replaced. Although not an internationally recognized distress signal, they are effective when supplemented with other recognized distress signals.

CELLULAR TELEPHONES

According to the U.S. Coast Guard, sailors use cellular phones more often than marine radios to call for assistance simply because more boaters carry cellular phones than VHF radios aboard their craft. Although sailors should be prepared to use any form of communication in an emergency, a cellular phone has several disadvantages associated with its use that make a marine VHF radio a better choice in many circumstances.

- Currently, 16 to 32 km (10 to 20 mi) offshore is the average effective range of cellular phones; range is determined by line of sight to the cellular antenna. Therefore, use is restricted to populated areas where there is coastal cellular coverage. Gaps in coverage make them unreliable, even for coastal use.
- Cellular communication is "narrowcast" between two parties, in contrast to the more public "broadcast" over VHF radio; this excludes potential assistance from boats that might be in the immediate vicinity.
- Cellular phone reliance excludes the possibility of avoiding a collision with a large ship because of inability to contact the pilothouse of the ship by cellular phone, as one could with a VHF radio (Table 70-2).
- In SAR operations, no practical way exists to maintain continuous communication with a number of rescue craft via cellular phone.
- The Coast Guard is unable to use radio direction-finding equipment to locate the vessel in distress if the vessel is

TABLE 70-2 Useful Marine Channels (VHF Radio)

06	Intership safety communications. Frequently used by boats engaged in towing.
09	Boater calling. Use to initiate contact with other boats, and ship to shore (hailing channel). Commercial and noncommercial.
12	Port operations, traffic advisories.
13	Intership navigation safety (bridge to bridge). Use this channel to contact a ship when there is danger of collision, or other emergencies near to both vessels.
14	Port operations and some Coast Guard shore stations. Used by Vessel Traffic Service in some harbors.
16	International distress, safety, and calling
22A	Working Coast Guard ships and shore stations, Coast Guard marine information broadcasts. Switch to 22A after contacting the Coast Guard on 16.
68, 69, 71	Noncommercial intership and ship to coast.
70	Digital selective calling (voice communications not allowed).
72	Noncommercial intership only.
78A	Noncommercial intership and ship to coast.
87A, B	Reserved for AIS digital communications.
WX 1 to 9	NOAA (National Oceanic and Atmospheric Administration) weather radio broadcasts on 162 MHz.

*See the 1995 radar reflector study performed by Stan Honey, Jim Corenman, and Chuck Hawley at SRI Labs in Menlo Park, California: ussailing.org/racing/offshore-big-boats/big-boat-safety-at-sea/safety-incident-reports

using a cellular phone, as it can with the VHF-FM signal. (New features of Rescue 21 may allow cellular triangulation in the future, but so far the cellular providers generally provide the positional information to the Coast Guard.)
- A cellular telephone has limited battery power and longevity, and cellular telephones in general have poor durability and water resistance.

If a cellular phone is the only communications link onboard, it can be made more robust as a communications tool with a 12V charger and waterproof case. A comprehensive list of emergency phone numbers should include local hospitals and physicians, regional Coast Guard rescue coordination centers, harbormasters, and maritime towing services.

SATELLITE PHONES

Portable satellite telephones are a versatile and useful tool for offshore sailors. Depending on the carrier, they can provide either coastal or worldwide voice and data communications for a reasonable initial, per-minute, and per-megabyte cost. (Prices are subject to change; few areas of consumer electronics change faster than the options available for satellite communications; both voice and data options are expanding continuously, and prices for both hardware and airtime are falling precipitously.) Service resellers can also rent phones or data terminals on a weekly or monthly basis, which may make sense for a single voyage or a summer of sailing. Due to the high cost of data, traditional web browsing is impractical, especially given slow data transfer rates. Generally, an email-forwarding service, such as Sailmail, will be used to strip away undesired images and spam. For small data files, such as GRIB weather charts, the cost is not excessive and speed not prohibitively slow. (The excellent, informative website sailmail.com has a variety of services for long-distance communication, including an SSB-based network of stations providing email service around the world.)

Satellite telephones require a clear view of the sky to operate, because the antenna must be directly visible to the satellite, or they may be used with a docking station and remote external antenna (which in turn can see the sky/satellite). Satellite phones are the primary high-seas communication alternative to SSB transceivers, although SSB transceivers have distinct advantages. When the mast comes down and antennas for the SSB and VHF are lost, a satellite phone may be lifesaving, especially when help is out of range of the handheld VHF radio. A satellite phone is an invaluable addition to the abandon-ship bag. When not in use, the satellite phone should be stored in a waterproof container together with a list of the critical phone numbers of the rescue coordination centers and Coast Guard communications centers in the area. Newer models can be preprogramed in the SOS function. A push of a button alerts the rescue service of choice and helps establish two-way communications to assist in their response. These models also are GPS enabled, which allows users to text message their location. Distress alerts and position data continue to transmit every 5 minutes until the user cancels the SOS; a fully charged battery will transmit for 26 hours in this mode.

Iridium is a popular phone providing reliable worldwide voice coverage without gaps. These portable phones can be used for text messages, voice transmissions, sending and receiving email and Internet communications, and serving as a WiFi connection. The Iridium communication system uses low earth–orbiting satellite constellations (see information on pricing, above).

Globalstar offers portable phones under the SPOT Global Phone and the Globalstar brands. Coverage for the Globalstar system is largely continental land areas and coastal waters out to about 200 miles. For many voyagers, this provides necessary coverage. The voice quality is very good, and data speeds are better than with Iridium.

Inmarsat, known for large ship terminals, offers the IsatPhone Pro and ruggedized IsatPhone 2, which uses geostationary satellites and provides nearly worldwide coverage (except at very high latitudes). The Fleet One system has been designed for small recreational and fishing boats, and provides global coverage for voice transmission, data transmission, Internet access, and text messages. Fleet One also supports Inmarsat's free 505 safety service, which in an emergency directs a call straight through to a Maritime Rescue Co-ordination Centre.

Each of these companies offers a wide array of payment plans for voice, short message service, and data. Each service offers the ability to call virtually any phone number in the world to get weather routing, medical advice, or miscellaneous information.

GLOBAL MARITIME DISTRESS AND SAFETY SYSTEM AND DIGITAL SELECTIVE CALLING

Until the new Global Maritime Distress and Safety System (GMDSS) is fully implemented, the Coast Guard and many recreational and commercial ships continue a radio watch on VHF channel 16. The GMDSS is a worldwide infrastructure, controlled from a shore-based communications center, to coordinate assistance to vessels in distress. This fully automated system uses satellite and digital communication techniques that require upgraded radio equipment and communication protocols. GMDSS simplifies routine communications at sea and facilitates regular weather forecasts, navigation warnings, and distress relays in the form of maritime safety information. Digital selective calling (DSC) technology permits a VHF radio (with DSC capability) to call another radio selectively using digital messages, similar to the modem on a computer. As with a direct-dial telephone call on land, only the vessel called receives the initial message. Every vessel has its own unique MMSI number. The radio must therefore be registered in order to be properly identified in an emergency or to be called directly by another boat using a DSC radio.* The vessel's MMSI and information about the boat are registered in the Coast Guard's national distress database. For VHF radios, channel 70 (156.525 MHz) is used exclusively for digital distress alerts, safety announcements, and calling using DSC techniques. No other uses are permitted. Distress messages can be sent automatically with DSC radios. The vessel's identity is permanently coded into the unit, and its position can be determined from the data output of a GPS receiver linked to the radio. On a DSC-equipped radio, the receiver sounds an alarm if it receives an "all ships call" (distress or otherwise), group call, or call specifically to that vessel.

A distress alert (equivalent to MAYDAY) can be sent to a shore-based rescue coordination center covering the area. Once the alert is sent, the radio will automatically repeat the call at intervals of between 3 and 4 minutes until the center acknowledges receipt of the message on channel 70. Subsequent communication should continue on channel 16 or another selected frequency for voice transmission. A verbal MAYDAY can also be sent immediately on channel 16 after the first alert. Most DSC-equipped transceivers can monitor channel 70 for DSC and channel 16 or other channels for voice transmission.

VHF-FM MARINE RADIOS

The VHF-FM radio transceiver is among the most popular marine communication systems, because it is a user-friendly, inexpensive, and reliable form of communication. It allows communication with those in the marine community of greatest utility: the U.S. Coast Guard, bridge tenders, fuel docks, yacht clubs, race committees, recreational boats, and even large ships. VHF radios specialize in ship-to-ship and ship-to-shore communication. VHF may also be used to check weather reports. Best of all, marine VHF radios are easy to use, even for crew with minimal experience.

The VHF signal range is limited to the line of sight and therefore depends on the height of transmitting and receiving antennas. A transmission range of 25 to 50 km (15 to 30 mi), and frequently farther when communicating to the Coast Guard, can be expected between boats having masthead-mounted antennas.

*To obtain and register a MMSI number, see the Boat U.S. website at http://www.boatus.com/mmsi, or the SeaTow website at http://www.seatow.com/boating_safety/mmsi.

FIGURE 70-26 A waterproof handheld VHF radio allows a person to call not only his boat but other boats in the fleet, should he go over the side. The low height-of-eye requires that one call early and often before the receiving party is over the horizon.

Communication is not private, which is a distinct advantage in maritime emergencies and SAR operations. VHF is the open party line connecting all vessels within range of the signal. Should a vessel broadcast a MAYDAY call, all boats in the area monitoring distress channel 16 will likely receive the call.

Offshore boats should have both a fixed mount radio, wired to the ship's electrical system with a masthead sailboat antenna, as well as a portable handheld VHF radio (Figure 70-26). Fixed mount radios not only have a higher antenna but also transmit at 25 W, and can therefore be heard for longer distances and over weaker signals. During an emergency, use the fixed mount radio for the initial distress call. The vessel should carry a spare antenna that can be substituted for the masthead antenna in the event of the boat being dismasted; this applies to owners of powerboats as well, because the antenna itself can be broken. In an emergency, a VHF antenna may be improvised by extracting 17.9 inches of center conductor from a piece of coaxial cable, while leaving the shield to one side.

After a knockdown or capsize, a handheld radio can operate independently of the ship's radio antenna (the mast may have been lost or antenna damaged), and can operate if the electrical system of the mother ship has been compromised, which is a common occurrence during a variety of emergencies at sea (e.g., flooding, fire). Consequently, the handheld radio is virtually indispensable during SAR operations and helicopter evacuation, when one is required to be mobile and on deck. Handheld radios can also be used to communicate with the mother ship from a dinghy, and are essential for communicating with rescue personnel from a life raft.

The range of handheld units is up to 5 km (3 mi), or further if communicating with a Coast Guard shore station. Transmit power is limited to 6 W, and all radios have the option to transmit at 1 W. Transmitting uses 15 times more battery power than does receiving; to maximize battery life, transmission should be used sparingly and initial contact should always be attempted first using lower 1-W transmit power. Higher transmission powers should be used only if contact cannot be established at lower power. Although the range of handheld radios is less than that of high-powered fixed-installation VHF radios, reliable communication can generally be established with any visible aircraft or vessel. When quoted, battery life is based on the 90/5/5 standard, meaning that the radio is on standby (no noise) for 90% of the time, receiving (audible signals) for 5% of the time, and transmitting for 5% of the time.

Digital selective calling (DSC) has affected handheld VHF and fixed mount radios. Several manufacturers offer handheld radios with built-in GPS receivers and ability to send the same DSC distress calls that a fixed mount radio can send. The combination of GPS and VHF radio simplifies their use considerably. An MMSI number should be requested* and assigned to the handheld radio–GPS unit so that it can function properly.

The most important VHF channel is channel 16, the distress and safety frequency (156.8 MHz). This frequency is used to initiate contact between two vessels and is the only frequency continuously monitored by the Coast Guard. When a radio is not active on another channel, it should be left monitoring channel 16, eavesdropping for distress and hailing calls. This can be accomplished by using the DualWatch or TriWatch functions on the radio (the radio switches quickly between channel 16 and one or two other channels), or by using a programmable scan feature. Table 70-2 lists other VHF-FM channels often used by pleasure craft.

A VHF radio is one of the best ways to summon help from the Coast Guard and other vessels. Prior to DSC, emergency procedure words were used to indicate the severity of distress:

MAYDAY: when there is a likelihood of losing the vessel or someone's life; distress.

Pan-Pan: when the vessel has a breakdown or there is a medical issue, but it is not life-threatening; urgency.

Securité: when there is an important message for other ships, such as describing a floating hazard or a buoy that is out of position; safety.

These words are still effective when requesting assistance. An example of a MAYDAY call is as follows:

"MAYDAY, MAYDAY, MAYDAY. This is the vessel Surprise, Surprise, Surprise." (Urgency word and vessel name three times.)

"We are located at 36 degrees 52.2 minutes north, 122 degrees, 19.7 minutes west." (Location from either the GPS or any display that shows position.)

"Surprise is a 38-foot yawl with a blue hull and a tan deck." (Description of vessel.)

"We are taking on water, and we cannot find the leak. We request immediate assistance." (Nature of the emergency.)

"There are six souls onboard. We have a life raft, and EPIRB, and life jackets." (Number of crew and information on safety equipment.)

"This is the vessel Surprise standing by on channel 16." (Complete the call and let potential responders know that you are standing by.)

DSC and the completion of the Rescue 21 network have made many of these techniques and terms less important, although sailors should continue to know procedure words and how to broadcast a MAYDAY. If the ship's radio has DSC capability (all radios sold for more than a decade have DSC built in), the process of transmitting a MAYDAY consists of lifting a small red distress flap and pressing the uncovered red button. This sends the digital equivalent of a MAYDAY, with the unique vessel ID (MMSI number) and precise position of the signaling vessel. This information will be displayed and stored on any vessel within range that has a DSC radio, as well as by the Coast Guard's Rescue 21 network. On many boats, the transmitted position will be transferred to a chart plotter as well. In this situation, the voice transmission of the VHF serves as a complement, allowing the crew in distress to add details to the distress call on channel 16 or whatever channel has been used, as directed by the Coast Guard.

Unfortunately, the Federal Communications Commission (FCC) and Coast Guard report that around 50% of the radios in use are not set up to use DSC and Rescue 21, because the radios predate the DSC requirement, the radio is not connected to an operating GPS, or the owner has not programmed the MMSI number into the VHF. An older noncompliant radio should be upgraded to one with DSC capability in order to take advantage

*You can request an MMSI number from the Federal Communications Commission (fcc.com), SeaTow (seatow.com/boating_safety/mmsi), or BoatU.S. (boatus.com/mmsi). For international use, the FCC is the best choice. However, for use within the United States, BoatU.S. has a free service and an easy-to-use online form. Remember to jot down your user name and password so that you can update your MMSI registration information if your situation changes or you sell your boat.

of one of the best and least expensive ways to get help in an emergency.

SSB-HF RADIOS

For communication offshore beyond VHF range, a more powerful and elaborate single-sideband (SSB) radio transmitter is required. The SSB's clear advantage over the satellite phone is that a transmitted distress message (voice and via DSC) will be heard by anyone who is listening or monitoring SSB frequencies, which are part of the internationally adopted Global Maritime Distress Safety System (GMDSS). All commercial ships operating under GMDSS are required to monitor marine SSB frequencies while at sea. Satellite phones cannot provide ship-to-ship safety communications or communications with rescue vessels or aircraft because the phone numbers of those craft are unknown. An SSB radio allows an unlimited number of people to listen in on a transmission. Other nearby commercial and recreational vessels that are monitoring the airwaves could lend a hand or communicate directly to offer advice, act as relay, or help in other ways. SSB is also the only way to participate in various regional safety and cruising nets, such as the Bahamas Air Sea Rescue Association (BASRA) Weather, Safety, and Traffic Net; Cruiseheimers Net; or Chris Parker's popular Marine Weather Center. Many cruising events, such as the Atlantic Rally for Cruisers' Caribbean 1500, require participants to have an SSB radio onboard, so that they can stay in contact and share important safety and weather bulletins. Until recently, all offshore sailboat races required SSB equipment, including daily check-ins to report positions.

Medium-frequency/high-frequency marine radio-telephone equipment operates between 2 and 23 MHz. This equipment can also be used to receive high seas weather broadcasts and, in combination with a laptop computer and special HF modem, can provide an easy and relatively inexpensive way to send and receive email. The five principal SSB email system providers for the recreational market are CruiseEmail, MarineNet, SailMail, WinLink, and SeaWave. Each provider uses a different software package with the Pactor-2 or Pactor-3 modem. Email is not just for social exchanges; it offers cruising boats a safety advantage for communicating safety-related data to boats around the world. Depending on the radiofrequency band and atmospheric conditions, the communication range may be several thousand miles.

As of August 1, 2013, the U.S. Coast Guard terminated its radio guard of the international distress and calling frequency 2182 kHz, and the international DSC distress and safety frequency 2187.5 kHz. On HF bands, the frequencies 4125 kHz (channel 450), 6215.5 kHz (channel 650), 8291.0 kHz (channel 850), 12,290.0 kHz (channel 1250), and 16,420 kHz (channel 1650) have all been designated for distress and safety calls. The HF transceivers can call and receive voice and digital communications to and from anywhere in the world on land and sea. As with a VHF radio, DSC (see below) can be used with the SSB radio when it is interfaced with GPS and has its own registered MMSI. All U.S.-flagged vessels require a Ship Radio Station License from the FCC to get an MMSI number. This number will be coded for international waters and registrations entered into the international SAR database. The Coast Guard transmits voice and weather information on various marine HF frequencies. The transmitters cover the Atlantic and Pacific Oceans, Caribbean, Gulf of Alaska, and Gulf of Mexico.*

The best way to select an optimum emergency frequency is to listen to the quality of a radio broadcast. Any station that is received loudly and clearly will also provide good reception for an emergency broadcast when needed. SSB is an excellent receiver for voice weather and weatherfax broadcasts. Optimal use of a marine SSB radio requires instruction and practice. Mobile Marine Radio (ShipCom) in Mobile, Alabama, is the sole provider of worldwide ship-to-shore HF SSB (and VHF in some locations) radiotelephone service in the United States.†

FIGURE 70-27 EPIRBs float upright and can transmit while bobbing in the water next to a life raft or damaged vessel. They include a tether that must be attached to the vessel or raft so that the EPIRB does not drift away.

EMERGENCY BEACONS

Emergency position-indicating radio beacons (EPIRBs) are handheld portable radio transmitters that can transmit signals interpreted as MAYDAY calls (Figure 70-27). These signals are the satellite-linked equivalent of a 9-1-1 call for mariners in distress. In the absence of a marine radio, or when out of range of coastal VHF stations, an EPIRB is the most important piece of signaling equipment. An EPIRB should be used when there is a life-threatening emergency; activation should be considered the equivalent of a MAYDAY call on VHF or SSB. The EPIRB should be located in a readily accessible location.

EPIRB signals are transmitted on established distress frequencies of 406/121.5 MHz. Signals are monitored by the global COPAS-SARSAT (search and rescue satellite-aided tracking) satellite system, coordinated by the United States, Canada, France, and Russia. This system (Figure 70-28) is a constellation of polar orbiting and geostationary satellites fitted with transponders to receive the distress signal and locate the beacon. Polar satellites orbit 966 km (600 mi) above the earth and have an orbit time of 105 minutes.

First-generation class A and class B beacons are no longer in use, having been replaced by the superior categories 1 and 2 EPIRBs and PLBs (personal locator beacons). The 406-MHz EPIRB provides the most reliable worldwide coverage. Satellites with 406-MHz transponders can store the signal in memory until a ground station is in view and then can retransmit the signal. The distress signal is quickly relayed to a ground station called

*Up-to-date schedules and frequencies used are online at nws.noaa.gov/om/marine/hfvoice.htm and weather.gov/om/marine/hfvprod.htm.

†Complete information regarding these radiotelephone channels can be obtained by calling 251-666-5110 or found online at shipcom.com/services.

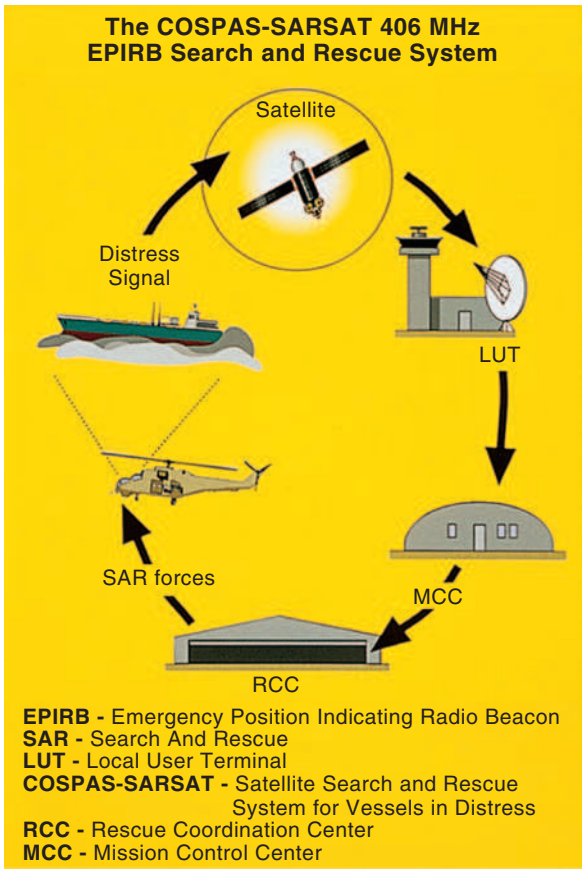

FIGURE 70-28 With an EPIRB or a PLB, one is able to transmit to a worldwide network that listens for distress signals, and then forwards them to the correct agency. Register the EPIRB!

a local user terminal, passed on to the mission control center, and relayed to the appropriate maritime rescue coordination center. Satellites are able to compute the beacon position to within 2 km (1.24 mi). The 406-MHz EPIRB transmits a digital signal with a unique identification code, which can be instantly identified through a NOAA-encoded transmission program. If the unit is properly registered with NOAA, vital information regarding the vessel can be passed on to SAR units. All registered EPIRBs are issued a dated decal that provides proof of registration and includes a unique 15-character hexadecimal code, registration expiration date, and the vessel's eight-digit registration code. Every owner who registers an EPIRB receives a sticker from NOAA printed with the beacon's registration number. This number should be verified to match the ID number on the beacon before it is attached to the unit. If the numbers do not match, the beacon is not properly registered. According to NOAA, 30% of beacons are not registered by their owners (registration can be done online at beaconregistration.noaa.gov/).

Another reason to register an EPIRB is to prevent unnecessary SAR operations. According to the Coast Guard, 96% of all EPIRB distress signals are false alarms resulting from faulty or accidental activation. If the rescue coordination center is able to phone the contacts listed on the beacon's registration form, they can in the majority of cases validate legitimacy of the distress signal.

The newest generation of EPIRB is the 406-MHz unit with a GPS interface or a built-in GPS receiver. The GPS-enabled beacon (also called a self-locating beacon) transmits its exact location within 100 m (328 feet), using GPS-derived latitude and longitude coordinates, along with the EPIRB signal. GEOSAR satellites receive the signal as soon as the beacon is activated. Position information is continually updated and stored every 20 minutes in the unit as long as it maintains a direct connection to the GPS receiver.

All EPIRBs have a homing signal at 121.5 MHz. This is the homing frequency used by SAR vessels and aircraft equipped with radio direction-finding equipment to locate the craft in distress. Once activated, the EPIRB should be left on until the emergency is over; to update position, it must broadcast continuously. EPIRB radio signals cannot penetrate water, wood, metal, or fiberglass (the unit must be outside the cabin), but signals will be received when transmitted from inside a life raft, on deck, or on the water's surface. All category 1 and 2 EPIRBs will transmit for at least 48 hours at −40°C (−40°F). PLBs will transmit for at least 24 hours, and some transmit for more than 40 hours.

Many cruising sailors believe a 406 EPIRB alert will bring rescuers in a matter of hours, or at most a couple of days. These expectations are unrealistic in many parts of the world. Although the SARSAT system and rescue coordination centers that support 406 EPIRBs save more than 1000 lives each year, the system is far from perfect. Some nations responsible for rescue coordination centers covering specific areas have failed to ratify the Search and Rescue Convention of 1979, and even after signing, lack resources to meet their obligations under the guidelines. These rescue coordination centers are not equipped to carry out effective SAR operations in their designated areas; moreover, SAR may not be a top priority for the planet's poorest countries. For example, when the 10-m (32-foot) Down East cutter *Leviathan* went down in a storm off the island nation of Madagascar, a 6-hour EPIRB signal was not picked up by the rescue coordination center in Madagascar, because the center is not available on a 24-hour basis. Rescue ships and planes were not immediately diverted to the beacon's location, and 10 days passed before an air search was launched in the Western Indian Ocean. By then, the task was futile; the cruising couple and lifeboat were never found.

Potential delays justify carrying a second 406-MHz EPIRB when cruising in remote areas and far offshore. While the EPIRB is activated, continue signaling by all other methods available. EPIRBs with lithium batteries have a shelf life of 10 years (although the recommended battery replacement interval is 5 years), making it feasible to store the second one in the life raft. If a single EPIRB is carried on a vessel, it should be packed into an abandon-ship bag or other storage area for safety gear, or it should be mounted in a special bracket for automatic activation and deployment (category I).

SEND DEVICES

Satellite emergency notification devices (SENDs) allow brief messages to be sent either one way (to shore) or two ways, either from continental and coastal waters or worldwide. A variety of devices are available, and this is an area of rapid technological development. A feature common to all devices is the ability to send an emergency message to a private firm that can then forward the message to the appropriate rescue agency. A second common feature is the ability to leave a "bread crumb trail" of locations that track one's vessel or personal movements, which can in turn be followed by selected individuals.

Although many consider a SEND to be a second-tier safety product compared with an EPIRB or a PLB, it has been instrumental in many rescues and extremely helpful in instances where the track function has helped document events aboard a vessel prior to an accident. Safety experts generally recommend that an EPIRB or a PLB be the first choice in alerting rescue agencies, but a SEND device can also provide useful information to friends and family, as well as to rescue agencies.

ABANDON SHIP AND LIFE RAFTS

DECISION TO ABANDON SHIP

There are many reasons to abandon ship.[6] Flooding, fire, or collision damage will generally sink a vessel if not controlled quickly. Crew should only abandon ship when the vessel is about to sink or is burning out of control. The adage "always step up to the life raft" means it is best to abandon the vessel no sooner than when the decks are awash. All options should

be considered. Abandoning ship prematurely puts crew at greater risk than remaining with a disabled craft. With rare exceptions, a floating disabled vessel is always the best lifeboat. The mother ship provides a stronger and more visible rescue platform than does a life raft. It is better stocked with provisions and equipment for communication and survival; conditions will always be more harsh in a life raft.

LIFE RAFT CLASSIFICATIONS

The three classes of life rafts (ocean, offshore, and coastal) refer to regions where the ship will be sailing and reflect differences in size, design, construction, quantity of survival stores, and packed equipment. For vessels cruising on a small boat near land, an inshore rescue platform, such as ResQpod, affords an adequate level of protection. Such platforms are not a substitute for a coastal life raft, and are suitable for use only on protected bays, sounds, and inland waters, especially in warm waters. A stable inflatable or rigid dinghy can also support the crew on inland waters and very near shore when abandoning ship. Coastal rafts are rated for open water, mostly protected or within 20 miles of shoreline; they have one or two buoyancy tubes and a manually or automatically erected canopy. This is ideal for offshore fishermen and coastal cruisers who need a light, compact raft, but its equipment must be augmented with a well-stocked abandon-ship bag (as with all rafts; see below). Offshore rafts are designed to ISO 9650 specifications, and are most appropriate for boats encountering offshore conditions for a relatively short duration but that are not taking a transoceanic voyage (Figure 70-29). SOLAS A Transoceanic Life Rafts are intended for the toughest conditions, with water temperatures below 5°C (41°F). These rafts include the greatest array of equipment and are the most sturdily constructed, to maximize self-sufficiency and permit survival for extended periods of time.

Regardless of the raft selected, the gear supplied will seem woefully inadequate, or at least that is the frequent lament from those who have abandoned ship. Due to the low likelihood that a life raft will ever be used, severely limited space available for survival gear, and desire to limit the amount of perishable items in the raft, it becomes incumbent on the raft owner to add incremental gear in the form of a grab bag, ditch bag, or abandon-ship bag. Regardless of its name, this bag contains incremental gear to improve the chances of rescue, and comfort and safety of the crew while awaiting rescue. See Box 70-10 for a comprehensive list of gear, but focus on having an EPIRB and a source of water before other items.

The life raft should be serviced according to the manufacturer's recommended interval by an authorized service facility. Owner and/or crew can request to be present when the raft is unpacked and repacked. The owner has a deep personal interest in seeing how well the job is done, and the raft's service appointment is an invaluable opportunity to become familiar with all components. Sailors should be intimately familiar with boarding methods, equipment onboard, expiration dates of supplies (e.g., flares, batteries), and inflation methods.*

FIGURE 70-29 Example of a modern offshore life raft. There are lots of options for adjusting visibility and ventilation, excellent ballasting, and redundant buoyancy.

BOX 70-10 Abandon-Ship Bag for Ocean Passage

Personal Gear
- Personal medication
- Crew passports (or photocopy), cash, credit cards, watch, prescription glasses, sunglasses
- Notebook and pencils

Signals and Communication
- Registered 406-MHz EPIRB, preferably with integrated GPS
- Waterproof and submersible portable VHF handheld radio transmitter with spare batteries. Optional handheld satellite phone in waterproof case with RCC and CG phone numbers
- Optional Search and Rescue Transponder (SART)
- SOLAS flare kit, smoke signals, canister and lung-powered horn, ship's bell, whistle, kite, dye marker, signal mirror, rescue streamer
- Radar reflector
- Waterproof binoculars
- Cyalume chemical light sticks, rescue laser light
- Waterproof LED flashlight with extra batteries
- Strobe light with spare batteries

Protective Gear
- Survival suits, survival blanket, gloves, spare sunglasses, hats (items vary with voyaging area)
- Expanded medical pack with extra seasickness medications, SAM Splints, narcotics, sunscreen, lip balm, emollient skin cream, tampons
- Optional backup sea anchor and line

Sustenance
- Portable reverse-osmosis desalinator (Katadyn Survivor 06 or equivalent)
- Fishing harpoon and spear gun with spare heads, extra fishing gear
- Knife, can opener, sharpening stone, small cutting board, scissors, storage bags, eating utensils, nylon mesh bag, waterproof bags, zip-lock freezer bags, sponge
- High-carbohydrate, low-protein energy bars, water packets
- Collapsible bucket, urine bottle, toilet tissue, feminine hygiene supplies
- Comprehensive survival manual, fish identification book, cards, reading material
- Collapsible water containers

Tools
- Tools, or multitool device
- Dive mask
- Duct tape for raft repair and crew repair
- Raft patching clamps

HOW TO ABANDON SHIP

All crew should don personal flotation equipment prior to entering the life raft. Excess layers of quick-drying clothing are recommended. One crewperson should broadcast a MAYDAY message on all available radios. A satellite telephone should be used to notify appropriate contacts of the crew's condition and coordinates of the ship's location. A 406-MHz EPIRB should be activated, and the life raft should be launched after locating the abandon-ship bag. Duplicates of essential communication equipment should be in this bag; if such equipment is not in the bag because it is in everyday use while sailing, a laminated list should be made of equipment to be loaded into the bag before

*An excellent way to become more familiar with life raft designs, features, and survival techniques is to take a U.S. Sailing Safety at Sea course, or a Sea Survival course outside of the United States. These courses combine lectures on a wide variety of safety topics, as well as demonstrations and hands-on interactions. See ussailing.org/seminars.

abandoning ship. It is recommended to augment the modest amount of survival gear found in most life rafts by having select items packed inside the raft container at its annual repack (include VHF radio, EPIRB, food, water maker, medical kit, signal pack, sharp knives). However, it is not recommended to put those items in the raft if they might be needed independently. These items may be packed into a waterproof bag securely attached by a short line to the raft.

For extended offshore and ocean trips, each crew member should have an easily accessible prepacked waterproof bag containing extra dry clothing, personal medications, passport, prescription glasses and sunglasses, personal strobe, safety harness and tether, wallet, and any other personal valuables and necessities. If the vessel's abandon-ship bag has been properly stocked, little else is needed from the sinking ship except synthetic blankets, jerry jugs of freshwater, extra food, and navigation tools; freshwater containers should be filled no more than two-thirds full so that they float. Additional communication and signaling equipment should be collected from the stricken vessel, even if emergency equipment has already been placed in the bag or raft, because it might become essential for backup if other equipment fails. This includes an EPIRB, SART, PLB(s), and a waterproof handheld VHF radio. In a life raft, one can never have too many EPIRBs, radios, flares, and fishhooks (see Box 70-10).

Life Raft Storage

Store the raft securely, either on deck with hardware capable of withstanding shearing forces due to capsize, or in the cabin. The life raft must be rapidly deployable in all conditions (offshore racing rules require 15 seconds or less) to maximize the chance of successful use.

If storing the raft on deck, a hard canister version is recommended, designed to withstand the rigors of a wet, salty environment (Figure 70-30). Rafts enclosed in a fabric valise that are stored on deck are much less waterproof than the canister models, and often end up with water vapor, rainwater, or seawater trapped inside, which can cause deterioration of seams and result in leaks. Some manufacturers now use a vacuum-bagging process to protect the raft from water intrusion while stored. Deck-mounted canister models have a hydrostatic release, which releases the raft from its cradle and allows it to float to the surface if the crew cannot manually launch it before the boat sinks. If the raft is stored below decks, make sure it too can be brought above decks and launched within 15 seconds. For offshore racers, rafts stowed below cannot be heavier than 40 kg (88 lb).

Launching the Raft

Before launching the raft, firmly secure the line coming out of the canister or valise to a strong point on the vessel. The canister should be thrown overboard on the downwind (leeward) side of the boat before inflation. Inflating the raft while it is still onboard is dangerous because it may become wedged in the rigging of the sinking ship or be punctured accidentally. A sharp jerk on the outstretched painter triggers the nitrogen/CO_2 cylinder and inflates the raft. Full inflation normally takes less than 30 seconds; care should be taken that the canopy arches inflate completely before boarding. The hissing sound heard after inflation does not signify a leaking or defective raft. The relief valve is simply releasing excess gas pressure. If the raft fails to inflate in the water, it should be brought onboard and manually inflated with the hand pump.

FIGURE 70-30 On the round-the-world racing Volvo 60s, 70s, and 65s, safety gear is stored where it can be reached quickly in an emergency. Redundancy and the highest-quality gear give sailors the best possible chances in inhospitable oceans.

FIGURE 70-31 Keeping dry is advised when boarding a life raft, but it is not easily done. Safety at Sea Seminars allow sailors to practice launching, boarding, and righting a capsized raft.

The first crew into the raft should be strong and free of seasickness; the abandon-ship bag should be handed directly to that person. If possible, the abandon-ship bag should be firmly attached to the painter so that it cannot be lost during transfer. The first aboard then helps remaining crew to board, while trying to keep water out of the raft (Figure 70-31). When possible, the crew should remain as dry as possible and enter the raft directly from the sinking boat, rather than from the water. If immersion is unavoidable, ease into the water slowly to avoid the cold shock response. Rafts are intentionally stable and drift slowly, and are therefore hard to maneuver to a person in the water. Therefore, the first person into the raft should throw the heaving line to those who might have drifted away. The line has a rubber doughnut-shaped quoit attached to the end, which allows a person to hold on firmly. Be sure to protect the airways of unconscious crew once they are in the raft, to avoid drowning. If rescuers must enter the water to rescue additional crew, they should be secured to the raft with a safety line.

LIFE IN THE RAFT: EXTENDING SURVIVAL TIME AND ANTICIPATING RESCUE

After successfully abandoning ship and securing both crew and equipment in the life raft, begin preparations for rescue. Panic, fear, and hopelessness can easily defeat the best-equipped and most experienced crew; an optimistic crew is more likely to survive. If the EPIRB is activated and the raft is near busy shipping lanes, the probability of rescue in a few days is high.

After everyone is aboard, the painter line should be payed out quickly and evenly to separate the raft from the sinking vessel. A safety knife is located in a pocket by the entrance of the raft to cut the painter should the ship begin to sink irreversibly. If the ship remains afloat, the raft should be attached to the wreckage with a quick release line; wreckage is easier to spot than a small life raft during rescue, and the main vessel remains a source of additional food and supplies. Because of the

CO_2/nitrogen mixture in the raft's interior space, the life raft should be ventilated periodically and thoroughly. The floor should be inflated separately with the manual pump to provide stability and insulation from the cold sea. With recovery of each crewperson from the sea, a significant volume of water enters the raft, adding to what may enter through wave action. Bail the raft as dry as possible, wring out all clothing, and bail again. All equipment should be inventoried and then secured. The EPIRB should be activated and left on until rescue is completed.

The first medical action should be distribution of seasickness medication. In a life raft (sometimes referred to as "an inflatable vomitorium"), everyone is susceptible to seasickness, especially in the first 24 hours. The raft should be made as dry as possible, and every effort should be made to maintain and conserve body heat, strength, and morale, in order to prepare for rescue.

No immediate danger exists if the raft capsizes with the crew inside, because there is sufficient air available in the space under the canopy. It is frequently necessary to enter the water in order to right the raft, unfortunately, because it is unlikely to right itself unless rolled again by successive waves. With the raft empty, pulling on the righting strap at the bottom can right it. One or more crew should kneel on the downwind side with feet braced on the CO_2 cylinder, and lean back (Figure 70-32). The raft will right easily when caught by the wind. It may also be possible to right the raft from inside.

To avoid recurrent capsizes in heavy wind and seas, take additional measures. Every raft is equipped with a cone-shaped sea anchor; this device is essential in rough weather. It provides stability in high winds, helps prevent capsizing, aids in directional control, and helps reduce drift. The benefit of reducing drift in storm conditions is controversial, because the raft may become sluggish with the sea anchor deployed. If sea room is sufficient, occupants may be more comfortable and the raft less likely to capsize if it is allowed to drift at the same rate as the waves. Weight distribution is also critical to avoid capsizing. Most of the crew should be positioned on the windward side, the side from which the sea anchor is deployed, to act as ballast. Weight on the windward side reduces the chance of the raft being lifted and flipped over by the wind. In seas with high wave crests, the crew must be prepared to maintain the raft's balance and quickly shift as needed to prevent capsize in the opposite direction.

Signals and Watch Schedules

Each type of distress signal should be reviewed, with inventory of signaling devices available, and an agreed-upon order of use. The most important duty aboard the raft is to maintain a continuous and effective lookout for land, ships, rescue boats, and airplanes. A watch schedule should be created to rotate duties every 2 hours. In calm seas, intermittent observations should be performed while standing up in the raft. The visible horizon is 2.6 nautical miles if the observer's eye is at 1.5 m (5 feet); raising the eye to 3 m (10 feet) (standing up in the raft as it rides on a swell) adds another mile.

FIGURE 70-32 Righting a life raft is not difficult, but remember to keep the inflation bottle (cylinder) close to the feet so that it does not strike one on the head.

Raft Maintenance

The raft is subject to tears, punctures, and chafe, which may cause air chambers to leak and lose air-holding integrity. As the temperature drops at night, the raft should be topped off with the manual pump to keep chambers taut with no apparent folds, which will provide the fabric floor with greater rigidity and support. The raft's pressure relief valves will prevent overinflation, but they must be operational (not plugged with provided emergency valve caps) during daytime to release heated expanding air. The raft should be protected against sharp, pointed, and abrasive objects, such as knives, tools, or angular debris.

Internal and/or external dome lights are attached to the canopy. These lights help survivors find the raft at night when abandoning ship and provide interior lighting; they are not intended to be rescue lights. Occupants should determine if the light(s) can be turned off for energy conservation if not needed.

Health Issues and Hypothermia

If sea conditions permit, the raft door should be left open to permit the crew to view the horizon and allow circulation of fresh air. After seasickness, no other condition is more debilitating than chronic sleep deprivation. Sleep deprivation causes decreased alertness with blackouts of attention, which become increasingly prolonged and frequent. A groggy, inattentive lookout can easily miss the opportunity to observe and signal a passing ship. Conversely, the person may begin to hallucinate and see ships and planes. Inability to make quick decisions may further jeopardize the rescue. Crew should rest, and be encouraged to stretch and relax muscles that are constantly working to keep the body stable in the raft. Each person's recumbent body should be insulated against heat loss, especially from the cold raft floor. Hypothermia, rather than exposure to severe weather, is the greatest threat to survival in most abandon-ship scenarios. A Mylar blanket reduces body heat loss by 80% to 90%; however, the major heat loss in raft survivors is by conduction through the raft floor, against which a foil blanket affords little protection. The raft floor should be lined with sails, tarps, and extra clothing. The double-layer floor also protects against the bumps of sharks, dorado, and other fish. In tropical climates, spraying salt water directly on skin or clothing to cool the body by evaporation is not recommended. Salt-encrusted skin is more likely to break down and become infected. Dry clothing protects skin from painful saltwater boils and other bacterial infections. Apply emollient creams to knees, hands, elbows, and buttocks to decrease skin abrasion and wear. Any area of skin breakdown should be treated with antiseptic or antibiotic cream and covered with a dry dressing.

Water

It is imperative to drink water at regular intervals to avoid dehydration and heat-related illness. In tropical climates, keep clothing on to help reduce fluid losses; long-sleeved shirts, trousers, and a hat are more effective than shorts and T-shirts in prolonging the cooling effects of evaporative fluid loss, as well as offering superior sun protection.

Body water losses in excess of 8% to 10% of body weight cause significant deterioration in mental and physical performance. Hallucinations and delirium from hypernatremia (high serum sodium levels) are common with progressive dehydration; death occurs with acute dehydration when water loss approaches 15% to 20% of body weight. By contrast, complete starvation leads to death in 40 to 60 days provided there is enough fresh water to drink. Decrements in physical and cognitive performance do not begin in well-hydrated individuals until they acutely lose 10% or more of body weight. Fit and healthy individuals are able to maintain a normal work capacity during short periods (< 10 days) on severely restricted diets. Reducing activity to a minimum decreases food and water requirements.

Water requirement changes dramatically with exercise, sweating, diet, and ambient conditions. Evaporative loss from sunlight in an open boat in the tropics is estimated at 2.4 L (5 pints) per day if the body is at rest.

Life rafts may be equipped with 500 mL (1 pint) to 1 L (2 pints) per person in 125-mL sachets; it is recommended that castaways should restrict water intake to about 600 mL (1.25 pints) daily, unless supplies are plentiful. Analysis of protracted voyages in life rafts during World War II found that the critical amount of potable water for survival was 120 to 240 mL (4 to 8 oz) per day.

Hand-operated, portable reverse-osmosis desalination units contain a semipermeable membrane that allows only fresh water to pass. The water makers weigh 2.5 pounds, and produce 250 mL (1 cup) of water in 15 minutes, or 1 L (2 pints) per hour by hand pumping, which is enough for the occupants in a 4-to 12-person offshore life raft. As an added benefit, the units remove bacteria, viruses, protozoa, and other contaminants.

Rainwater can be collected with the life raft canopy. An exterior gutter collects and routes the water to a large container for storage. Daily canopy washing with seawater, whenever practical, can help remove the buildup of salt deposits. In a heavy sustained downpour, rain should be allowed to rinse the canopy before water is collected.

Fish and other sea creatures contain water with extremely low salt content in their eyes, flesh, and cerebrospinal fluid. Fish blood has a high salt concentration and is not recommended. Juices can be pressed out of the flesh by twisting pieces of fish in a cloth. The blood from sea birds and turtles is a source of hydration for castaways.

Seawater is not potable. After continuous exposure to harsh elements, intolerable thirst may drive castaways to drink seawater. Succumbing to this powerful temptation is a major cause of death and hastens dying. Seawater is approximately four times saltier than blood. Drinking seawater usually causes immediate vomiting. If ingested and absorbed, seawater triggers a process of osmosis in which free water is shifted from the intracellular space into the blood and other extracellular fluids to restore equilibrium. The fluid shift dehydrates every cell in the body, including brain cells, which contributes to the reported madness in persons who have drunk large quantities of seawater.

At high latitudes, old sea ice is a good source of water. Sea ice loses its salt content after one year. The ice is brittle, bluish in color, and has round edges. New sea ice is gray, salty, opaque, and hard. Melting the ice allows tasting and judging of salinity. If the temperature drops to freezing, seawater can be collected in a can and allowed to freeze. Fresh water freezes first. Therefore, the salt concentrates in the center, forming slush surrounded by ice containing very little salt. Sea ice should not be confused with ice from an iceberg. Water from melted iceberg chunks is glacial fresh water. As a last resort, chewing a piece of gum or cloth will help moisten the mouth and reduce thirst.

Unless survivors are assured of an early rescue or have a reverse-osmosis water maker, they should consume no water in the first 24 hours, and use the body's reserves; thereafter, survivors should restrict intake to about 500 mL (1 pint) a day. If water is plentiful, drink up to 1 L (1 quart) daily.

Food

Nutrition is the last priority for survival. If rescue is expected to take many days or weeks, the crew should eat little or nothing for the first 24 hours; thereafter, lifeboat rations of carbohydrates should be eaten. Some rations should be saved until rescue is imminent, when extra energy will be needed. Rations should be regarded as more of a medicine to extend survival time, rather than as a meal. Dried food should not be consumed unless water is available, and protein intake should be limited to conserve the body's water. When 2 or more quarts of fresh water are available per day, normal eating patterns may be resumed.

Fish are usually the mainstay of a diet at sea once survival routine and water consumption have been established. The raft casts a dark shadow beneath the surface, which appears as a safe haven for a variety of fish, especially dorado (school dolphin). With practice and patience, fish can be taken near the sea surface with a harpoon, gaff, or spear gun. Care must be taken not to puncture the raft with these devices. At night, fish are attracted to bright lights. Instead of a flashlight, a signal mirror or any other shiny surface may be used to reflect moonlight onto the water.

When bringing a fish aboard the raft, the creature should be wrapped in a piece of cloth or canvas, because both dolphin and wahoo have serrated teeth and tend to thrash wildly when they come out of the water. Wounds from fish-induced bites, punctures, and scrapes heal poorly and easily become infected. A cutting board should be ready to kill the fish quickly by cutting the spine right behind the head. A large fish can be stunned with a blow to the top of the head at eye level. Simply covering its eyes will calm it down sufficiently to position for a quick kill.

Certain fish are inherently poisonous (see Chapter 77); these are usually located around shoals and reefs in shallow waters. These include pufferfish, porcupine fish, and ocean sunfish *(Mola)*. Any fish with spines or bristles instead of scales should not be eaten.

As a general rule, a fish that smells bad after storage should not be eaten. If there is uncertainty about the safety of a fish, it should be tested for edibility. If the flesh burns, stings, or tastes bad on the tip of the tongue, it should not be consumed. If it has an acceptable taste, a small piece should be eaten every hour for 3 to 4 hours initially, and if there are no ill effects after 12 hours, edibility and nontoxicity may be inferred. If there is excess fish after eating, the flesh may be cut into thin strips about an inch wide and one-half inch thick, and spread out to dry in the sun on a flat surface. If the flesh is cut with the muscular fibers running the long way, the strips can be hung on a piece of string and allowed to dry in the open air under the raft canopy. Fish spoils within hours in the heat, so the drying process should be started immediately after the fish is caught. Fresh-caught fish (except tuna), as well as dried turtle and bird meat, are good for days when the heat and humidity are not too high. Most ocean fish can safely be eaten raw. Freshwater fish should not be eaten raw because they harbor parasites.

Seaweed is valuable in two ways. As a floating nest, it can harbor a variety of small edible creatures, including small fish, barnacles, crabs, and other crustaceans. All kelp and almost all brown and green seaweed are edible, although of limited nutritive value. Red seaweed is highly toxic, and any seaweed that tastes bitter may be one of the rare poisonous varieties.

Many sea creatures follow a raft. Dolphins and whales may accompany the boat, but are not likely to harm the survivors in a raft. Sharks can be a menace as they swim about the raft for days or even weeks, and drive away other potentially edible fish. The shark's habit of frequently bumping a raft with its abrasive skin causes wear on the flotation chambers. Blood and offal should be disposed of at night to avoid attracting sharks and other carrion feeders, preferably when the raft is moving. Contamination of the waters around the raft, resulting in a waste trail for sharks to follow, should be avoided, and hooked or speared fish should be brought aboard as quickly as possible.

RESCUE AND EVACUATION OF THE SICK AND INJURED

Transferring personnel from a boat or a life raft to a rescue ship or helicopter entails risk for everyone; this may well be the most dangerous aspect of the survival ordeal.

AUTOMATED MERCHANT VESSEL REPORTING PROGRAM

AMVER, the **A**utomated **M**erchant **VE**ssel **R**eporting program[1] (formerly named the Automated Mutual-Assistance Vessel Rescue System) provides resources to help any vessel in distress on the high seas. AMVER is sponsored by the U.S. Coast Guard, and uses a unique, computer-based, global ship reporting system to assist worldwide maritime SAR authorities. Although reporting is voluntary for ships from many countries, AMVER participation is mandatory for U.S.-flag shipping. With the advent of the GMDSS,

the role of AMVER complements the emerging technology. Today, more than 22,000 ships from over a hundred nations participate in AMVER. An average of 4000 ships are on the AMVER plot each day, and this number continues to increase. These merchant ships are not specifically designed for SAR and their crews may not be trained for recovering survivors from small boats or life rafts under storm conditions. Offshore, however, they may be the only rescue option.

In ship-to-ship rescue, collision between vessels is the greatest risk. The typical scenario is of a large merchant ship with limited maneuverability approaching a smaller vessel in distress with almost no maneuverability. Bringing a small boat alongside a commercial ship in a gale is a skill that cannot effectively be practiced; "Even under ideal circumstances, it is a highly dangerous, heart in the throat, adrenaline-fueled action," a transpacific racing sailor once said who had abandoned ship and was aided by a huge container ship. Unless the rescue craft is designed for rescue work, the captain and crew experienced, and the seas relatively calm, it is much safer for boats and crew to use a smaller craft to transfer personnel between the boats. Options include a rigid-hull inflatable boat, lifeboat, or even a life raft. In rough seas, a boat or survival raft should never be secured to the rescue vessel. The constant battering of the two hulls is likely to damage the smaller craft, and may sink it. Whether to approach the rescue vessel upwind or downwind depends on wind, sea, and size and relative drift of the two vessels. Becoming pinned and capsizing are risks when sitting in the lee (downwind) side of a large, rolling ship.

The transfer of personnel between ships is also hazardous if sick, injured, or exhausted crewmen are required to climb a cargo net or pilot ladder; climbing out can be a difficult task even for a healthy crewman.

A large rescue ship most often approaches upwind of the survival craft unless the ship's rate of drift is much greater than that of the craft. This provides a calmer sea in the lee of the rescue vessel as it slowly drifts to the survivors' craft. If a sea anchor is deployed, it should be pulled in to prevent entanglement in the rescue vessel's propeller. Communication should be established with the rescue craft on channel 16 to coordinate the rescue. If the raft is to be lifted aboard with injured survivors, the floor must be fully inflated. Lifting lines should be attached firmly to the towing bridles on both sides of the raft, along with two steadying lines to each side.

The dangerous climb up a net, pilot ladder, or Jacob's ladder during a rescue should not be attempted without a safety line. When boarding a rescue ladder, the climb out of the life raft should be initiated when the raft is on the crest of a wave; at that moment, transfer to the ladder should be attempted. Subsequently the survival craft will drop down while the crewmember is ascending and in theory reduces the danger of the craft rising up on a wave and crushing the climber. The safest procedure is to be hoisted up to the deck in a harness by a deck cargo crane.

HELICOPTER EVACUATION

Helicopter emergency evacuation and rescue have become relatively common within 483 km (300 mi) of coastline. A detailed briefing is radioed to the crew when the helicopter is en route to the vessel in distress. A crewmember should be assigned to monitor the radio and listen for the pilot's radio briefing on channel 16 VHF radio, or 2182 or 4125 kHz on SSB radio; radio contact should be maintained with the pilot until the evacuation is completed (Figure 70-33).

All loose gear onboard must be well secured, including cockpit cushions, coils of line, winch handles, dive gear, hats, and clothing. Any gear not secured on deck will become a flying missile in the 161-km/hr (100-mph) downdraft generated by the helicopter. This debris may be sucked into the intake of the helicopter's engine or become tangled in the rotor blades.

All crew should wear life jackets, with extra layers of clothing to protect against the chilling downdraft. Avoid shining flashlights and laser lights on the helicopter. Flares should not be fired in the vicinity of a helicopter.

FIGURE 70-33 Coast Guard MH-65 helicopter off of Pearl Harbor, Hawaii. With a duration of about 3 hours, the MH-65 is used within 100 miles of the coast.

The transfer device is either a rescue basket or a Stokes litter (Figure 70-34). Selection depends on the victim's medical condition and whether it is necessary to remain horizontal during the hoist. The horizontal position is particularly important for persons with suspected spine injuries or severe hypothermia. A basket is the preferred device for lifting; baskets are easy to enter, especially in rough weather, and have positive flotation. The basket will settle on the sea surface, enabling someone in the water to float into it.

A horse collar sling (Figure 70-35) is a padded loop placed over the body, around the back, and underneath the armpits. The hoist is made with the line in front of the face. Always wear a PFD when entering the basket or hoist, and follow directions for securing safety straps.

The helicopter builds up static electricity traveling to the rescue scene, and the charge is transferred down the cable to the basket. The basket should touch the deck or the water first to discharge any static electricity. Failure to follow this procedure will deliver a strong, but nonlethal, electric shock to the victim. The orange steadying line, which is lowered first, is safe to handle and will not produce any shock.

The hoist cable or steadying line should never be attached to any part of the vessel or life raft, even temporarily. The winch operator will react immediately, and move to sever the cable from the hauling winch to prevent disaster.

If a Coast Guard helicopter picks up survivors from a life raft, survivors may be instructed to sit on the roof and on the inflated support arch to help stabilize the raft from capsize and decrease the amount of surface exposed to downdraft. A rescue swimmer

FIGURE 70-34 The interior of an MH-60 Jayhawk is crowded, even before the survivors are taken onboard. The basket, rafts, and pumps can be seen in the photo. This gear can be jettisoned if room is needed for rescued mariners.

FIGURE 70-35 Rescue swimmer being hoisted by an MH-60 Jayhawk with a lifting sling.

from the helicopter crew will assist in transfer, and when crew are required to jump into the water in order to be hoisted aloft. The raft may also be used as an intermediate rescue platform between the distressed vessel and the helicopter.

The helicopter is a versatile, effective and powerful means of transportation and rescue, with multiple applications for safe and effective SAR.*[3] The U.S. Coast Guard operates two types of helicopters (Table 70-3).

The C-130 Hercules is the largest of the U.S. Coast Guard SAR fleet, with a range in excess of 1609 km (1000 mi). It can air drop an enormous amount of lifesaving equipment, including dewatering pumps, life rafts, and survival and signaling equipment. A new twin-engine turboprop, the HC144 Ocean Sentry Medium Range Surveillance Aircraft, has joined the Coast Guard's air wing. Although it is smaller than the Hercules, the Ocean Sentry has similar systems, allowing it to carry out a wide range of Coast Guard surveillance, SAR, and transport missions. It can be outfitted with mission system pallets, a roll-on, roll-off suite of electronic equipment that enables the air crew to compile data from the aircraft's multiple integrated sensors to transmit and receive both classified and unclassified information from other assets, including other aircraft, surface vessels, and shore facilities (uscg.mil/acquisition/mrs). It can cruise at 215 knots and has a range of 2100 nautical miles.

TABLE 70-3 Coast Guard Search and Rescue Helicopters

Model	MH-60T Jayhawk	MH-65D Dolphin
Crew	Pilot, copilot, rescue swimmer, flight mechanic	
Maximum takeoff weight	9926 kg (21,884 lb)	4300 kg (9480 lb)
Maximum speed	180 knots	175 knots
Range	700 NM	375 NM
Duration	6 hours	3 hours
Capacity (additional people [passengers])	4+6	4+5

*To view an excellent instructional video for recreational boaters on helicopter rescue, see the Cruising Club of America website: cruisingclub.org/seamanship/seamanship_safety_heli.htm.

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CHAPTER 71
Diving Medicine

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There are five generally recognized global diving communities, each with its own set of medical, operational, training, and regulatory frameworks: recreational (including diving that is technical in nature), public safety, scientific, commercial, and military.

Recreational scuba diving is an adventurous and comparatively safe activity for a cross section of the general public. It is practiced by individuals of all ages who prior to training complete a medical history form (e.g., as provided by the Recreational Scuba Training Council). If there are any potential contraindications, the participant is referred to a physician (preferably one trained in diving medicine) for further evaluation. The diving depth window for recreational divers breathing from an open-circuit scuba system with compressed air or an enriched air mixture of nitrogen and oxygen (nitrox) is generally limited to 40 m (130 feet). Technical diving often far exceeds recreational diving in depths and times, may use mixed gases, and commonly incurs obligatory decompression stops. Using advanced equipment (such as multiple cylinders and gases or closed-circuit rebreathers [RBs]) mandates an advanced level of training and an in-depth knowledge of decompression techniques and equipment.

Public safety diving is an activity practiced by lifeguards, law enforcement officers, firefighters, U.S. Coast Guard personnel, Environmental Protection Agency workers, Department of Homeland Security personnel, and others. This type of diving is performed as a function of search and rescue attempts, crime scene investigations or body recoveries, or hazardous material recoveries. The public safety diving community consists of employees from various jurisdictions but also has a large contingent of volunteer divers who participate in the search and rescue teams of sheriff, police, or fire departments. The public safety diver's exposure to contaminants in the water column or sediments is of acute and chronic medical concern, yet can be largely mitigated by using encapsulated diving equipment and appropriate decontamination procedures following a dive. Radiologic, chemical, and microbiologic hazards can trigger a number of serious systemic or dermatologic signs and symptoms. Often, the connection is not made between the diving exposure and the clinical

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presentation, which emphasizes the need for further medical and operational education of public safety divers.

Scientific diving is performed by scientists whose sole purpose for diving is to use their expertise in making underwater observations and gathering data to advance knowledge. Medical, training, and operational standards are consensually promulgated by the American Academy of Underwater Sciences (AAUS; aaus.org), a professional society recognized by the U.S. Department of Labor Occupational Safety and Health Administration (OSHA). Due to the international and interdisciplinary nature of marine science, international organizations are increasingly adhering to AAUS standards. This facilitates the conduct of collaborative, multiinstitutional, international diving research projects. Many of these research projects are conducted at remote sites such as under polar ice, at South Pacific atolls and coral reefs, and from research vessels at otherwise inaccessible dive sites adjacent to third-world countries. As such, diving safety risk assessment and mitigation are carefully planned in consideration of emergency evacuation, hyperbaric chamber location, method of transport, and emergency oxygen supplies.

Commercial diving activities involve underwater construction, troubleshooting for and inspections of underwater structures, oil field work, and ship's husbandry, among other tasks. Like scientific diving, commercial diving is governed by OSHA (29CFR1910 Subpart T, Commercial Diving Operations). The Association of Diving Contractors International sets standards for commercial diving, collects exposure data, and creates educational and training tools for the commercial diving community.

Most military diving procedures are under the auspices of the U.S. Navy. Several Department of Defense sections manage diving operations, but by and large, the Navy tables and Navy-specified dive computers are the standard; the Navy's *Diving Manual* is the reference source, and its dive school curriculum is often the baseline for training. Of the five diving communities, the military and commercial diving sectors are best equipped from a resource and training perspective to manage diving emergencies, such as decompression sickness (DCS) and gas embolism.

The diving communities recognize that DCS is a risk of diving, but the incidence rates are acceptable; in fact, DCS occurs infrequently. Nonetheless, scuba diving can be a physically demanding activity because of intrinsic hazards of the aquatic environment and the physiologic effects of breathing compressed gas at depth.

The hazards of diving include the generic problems found in other aquatic activities, such as drowning, immersion pulmonary edema, hypothermia, skin and ear, nose, and throat disorders, water-borne infectious diseases, and interactions with hazardous marine life, as well as unique problems related to the increased pressure gradients underwater and effects of increased partial pressures of gases on human physiology.

There are many millions of recreational, public safety, scientific, commercial, and military divers worldwide; hundreds of thousands of new recreational divers are trained each year. The U.S. recreational diver population is estimated at 2.7 to 3.5 million divers based on the Diving Equipment and Marketing Association Certification Census and the Professional Association of Diving Instructors entry-level diver certification history.[145] Diving is conducted in every imaginable aquatic setting, including the open ocean and along the shores of oceans, fresh water, and confined environments such as aquariums. Despite the extent of activity, diving-related fatalities and serious injuries are rare. For recreational divers, the average death rate (based on data derived from case claims of insured divers) is 16.4 deaths (range 12.1 to 22.9) per 100,000 divers per year.[82] The fatality rate per dive is a better measure of exposure risk.[283] Training dive logs have reported a mean annual fatality rate of 0.48 deaths per 100,000 student dives per year.[243] The chance of suffering DCS during any single recreational dive is approximately 4 in 10,000 in warm water and 59 in 10,000 in cold water.[281]

The most up-to-date information about recreational diving accidents is compiled by the Divers Alert Network (DAN), a not-for-profit diving safety medical organization supported by clinical and academic affiliations, including the University of California at San Diego and a network of referral diving physicians. Established in 1980, DAN provides diving medical assistance to recreational, public safety, and scientific diving communities all over the world. Services include dissemination of medical information online (DAN.org) and through a medical hotline (919-684-9111), research results from studies and workshops, educational courses, and insurance products. DAN is a member-supported organization with more than 250,000 American members and an additional 175,000 international (IDAN) members.

The number of diving-related accidents in North America reported by DAN has been relatively stable over the past three decades. DAN's research department analyzes these observational data to eliminate reporting bias and for completeness of information about the frequency and type of diving performed. Diving medical safety continues to be DAN's primary mission-oriented focus. One of DAN's key roles in the diving industry is management of recreational diving fatality and accident data. Diving-associated fatalities peaked in the mid-1970s, with annual rates as high as 150, and have been stable since that time, with an average of 84 (range 77 to 91) fatalities per year.[82] In a recent effort to further define the scope of diving fatalities and overcome the perennial problem of uncertain total numbers (denominators), DAN researchers used claims data from 2000 to 2006 from insured members. Over that 7-year period, there were 187 death claims among 1,141,367 insured member-years, for which the mean annual fatality rate was 16.4 (range 12.1 to 22.9) deaths per 100,000 persons.[82] Fatality data from the British Sub-Aqua Club are similar for the same period, with 14.4 deaths per 100,000 divers.

DAN hosted a 2010 workshop on Recreational Diving Fatalities[283] to address industry concerns about root-cause analysis (trigger, disabling agent/action, disabling injury, and cause of death) of diving accidents and to solicit input from industry leaders about possible intervention strategies. Denoble and colleagues stated, "The most common disabling injuries associated with death were asphyxia, arterial gas embolism (AGE), and acute cardiac related events. The most common root causes were gas supply problems, emergency ascent, cardiac health issues, entrapment/entanglement, and buoyancy trouble. The risk for death while diving increased with age, starting in the early-thirties years of life. This is likely due to the naturally increased prevalence of cardiac disease with age, but an increased association of AGE and asphyxia were also associated with aging."[81]

Since scuba diving made its debut in the United States in 1951, the nature of the diving population has substantially changed. Scuba divers of the 1950s and 1960s were generally "water people" who were well-trained, strong swimmers; experienced in breath-hold diving; and mostly male. For these individuals, donning a scuba tank and regulator was a natural extension of a familiar activity. These early scuba divers seldom encountered problems that required attention from the general medical community. However, as scuba diving equipment became more available, adventure-minded persons from all walks of life became attracted to the sport. Popularization of recreational scuba diving in recent years has attracted participants who are poorly conditioned, have little or no experience in aquatic or other sports, are of advanced age, have significant underlying medical conditions, or are sometimes severely disabled. Because of the hostile and unforgiving nature of the aquatic environment and the time-sensitive nature of underwater emergencies, such persons may be at increased risk for a diving-related injury or illness. Certain medical conditions may constitute temporary or absolute contraindications to diving.

In the past two decades, recreational divers have increasingly sought out more technically complicated, and often more remote, diving activities in efforts to increase the amount of time or depth attained underwater. Although these advanced diving techniques (e.g., using an RB apparatus or mixed gas) have long been used in scientific, commercial, and military diving programs under controlled and supervised conditions, there are significant concerns about the safety of "technical" diving in the typically less-controlled recreational setting.

All primary care physicians should be prepared to answer basic questions about fitness for diving and to initially manage

diving-related medical emergencies. Every emergency medical treatment facility should be prepared to evaluate a dive accident victim, provide emergency care, and if needed, arrange appropriate transport to a hyperbaric treatment facility if a diver is suspected to be suffering from DCS or AGE. The DAN emergency hotline (919-684-9111) is available for consultation with diving medical experts.

This chapter focuses primarily on the pressure-related diving syndromes collectively known as dysbarism; additional conditions relevant to diving are discussed in other chapters (e.g., immersion hypothermia in Chapter 8, submersion incidents in Chapter 69, hyperbaric oxygen therapy in Chapter 72, and hazardous marine life in Chapters 73 to 75).

HISTORICAL PERSPECTIVE

Humans did not evolve for an aquatic existence and are not well adapted for functioning in the aquatic environment. Nonetheless, for thousands of years, humans have been breath-hold diving to gather food and other natural resources from the oceans. There is archaeologic evidence that Neanderthal man breath-hold dived for shellfish 40,000 years ago. The Ama of the Izu Peninsula of Japan and the Haenyeo of the South Korean Island of Jeju (in both cases women) have been breath-hold diving to collect shellfish, lobster, sea urchins, octopus, and seaweed for at least 6000 years. Women of the Yahgan, Alakaluf, and other nomadic sea-going female Fuegian peoples of southern Patagonia engaged in similar diving practices for probably 5000 years before these primitive Native Americans became extinct in the late 19th and early 20th centuries. The fires that these divers built to warm themselves along the shores of what is now known as the Straits of Magellan inspired Ferdinand Magellan to name the area Tierra del Fuego (land of fire).

Written records of diving for salvage and military purposes date back to around 500 BC, when the Greek historian Herodotus recorded the feats of Scyllis and his daughter Cyana as they dived in the Mediterranean Sea for the Persian king Xerxes during the 50-year war between Greece and Persia. Many early cultures around the Mediterranean Sea made use of divers in military operations, usually to cut the anchor cables of ships, bore holes in the hulls of enemy vessels, and build harbor defenses. In 360 BC, Aristotle described the use of diving bells (basically, upside-down buckets) to supply air to sponge fishermen, and in 332 BC, Alexander the Great is reported to have gone underwater in a specially constructed glass diving bell (Colimpha) to observe his divers removing defensive obstructions from the besieged harbor of Tyre.

Colorful accounts of military and salvage divers dot the history of Roman and other early cultures. By 100 BC, diving operations around the major shipping ports of the eastern Mediterranean were so well organized that there were legally binding payment schedules, which recognized that the risk to the diver increased with depth underwater.

Although written records of diving in the Americas were not discovered until after European explorers arrived in the New World in the 16th century, Peruvian artifacts dating to AD 200 show divers wearing goggles and holding fish, so it is reasonable to assume that breath-hold diving had been practiced long before the Europeans arrived. Spanish explorers are reported to have enslaved native divers and forced them to dive for pearls in the Caribbean. These explorers also made extensive use of divers to salvage galleons wrecked in the Caribbean and along the coast of Florida.

Human underwater exploits remained limited to breath-hold diving until approximately 300 years ago, when a series of technological developments began to expand human underwater activity. These developments principally involved the use of different types of external air supplies to prolong submergence.

In the 17th century, primitive bells containing air were carried from the surface, allowing Swedish divers to stay underwater longer than a single breath and to salvage cannons from Stockholm's harbor.[228] In 1690, Sir Edmund Halley devised a leather tube to carry surface air to barrels, which resupplied air to manned bells at a depth of 18 m seawater (msw) (60 feet seawater [fsw]). These barrels were submerged, and the air they contained was compressed.[73]

The first practical diving suit was fabricated by Augustus Siebe in 1837.[5,73,228] Atmospheric air was supplied to the diver as compressed air from a manually powered pump on the surface. By 1841, French engineers had developed the technique of using compressed air to keep water and mud out of caissons sunk to the bottom of riverbeds for bridge footings and tunnels. Soon thereafter, it was noted that people working in a compressed-air environment sometimes suffered joint pains, paralysis, and other medical problems soon after leaving the caisson. This poorly understood condition was called caisson's disease, and was the first recognition of what is now known as decompression sickness (DCS).[236]

Underwater diving remained an esoteric activity having limited commercial and military utility until the 1930s. By that time, and increasingly during World War II, the military importance of submarines and other undersea activities became evident to navies around the globe. With the development of submarine forces came the need to train men to escape from submarines that became disabled at depth (an all-too-frequent occurrence in the early days of submarines). Given the shallow operational depths of these early boats, it was usually possible to escape by simply exiting the vessel and ascending to the surface. It was noted early that failure to exhale while ascending through the water column led to pulmonary overpressurization accidents and a new and dramatic syndrome that we now know to be AGE.

In 1865, the French engineers Rouquayrol and Denayrouze developed a device that could supply air on demand at increased ambient pressures relative to the 1 atmosphere of pressure found at sea level. These inventors were able to supply air on demand at appropriate breathing pressure to persons underwater with a "demand regulator," (versus a free-flowing breathing apparatus). This device originally required a surface air supply connection.[5] The demand valve regulator was later modified to supply auxiliary oxygen for pilots operating at high altitude. In 1943, while working with the French resistance against Nazi Germany, Jacques-Yves Cousteau and Emile Gagnon combined a demand valve regulator with a compressed-air tank, giving rise to what they called a self-contained underwater breathing apparatus, or *scuba*.

The potential military usefulness of scuba was immediately recognized and led to a considerable amount of investigation during World War II. As initially configured, scuba was used in an open-circuit mode in which exhaled air was simply vented into the water. This was wasteful of the compressed-air supply and had other disadvantages for military uses. Further work led to refinement of RB devices (both closed-circuit and semiclosed-circuit systems), such as Christian Lambertsen's amphibious respiratory unit.[13] These RB systems conserved the breathing gas by using a carbon dioxide scrubber and recirculating all or part of each exhalation (see Rebreather Diving, later). These specialized scuba systems were useful for military purposes because they allowed longer submergence times and could be used in clandestine operations or when disarming pressure-sensitive explosive devices. However, they had a greater frequency of mishap, so RB systems were not widely used until the 1970s for science. In the 1990s, there began a resurgence of interest in RBs by technical recreational divers.

After World War II, development and marketing of open-circuit scuba equipment to the general public made the underwater world accessible to growing numbers of people. The first civilian scientific diving course was taught at the Scripps Institution of Oceanography in 1951 by Conrad Limbaugh and Andreas Rechnitzer. This was the precursor curriculum to the Los Angeles County Parks and Recreation Scuba Program pioneered in 1954 by Al Tillman and Bev Morgan, the first recreational diving training association in the United States. In the last six decades, scuba diving has opened the underwater world to millions of divers and hundreds of millions of film and photography observers. Scuba is now used as a basic tool with myriad commercial, military, scientific, public safety, and recreational applications (Box 71-1).[10]

BOX 71-1 Types of Diving

- **Commercial Diving**
  - ***Harvest of Natural Resources***
    - Oil and natural gas
    - Minerals (e.g., gold)
    - Fish and shellfish
    - Pearls, corals, and shells
    - Algae
    - Wood (e.g., underwater logging)
    - Aquaculture
  - **Salvage and Recovery Operations**
  - ***Maintenance and Construction***
    - Ship hulls
    - Nuclear power plants
    - Bridges and tunnels
    - Piers and jetties
    - Aquariums
    - Water treatment plants
    - Sewers
    - Dams
  - **Underwater Photography and Motion Picture Productions**
- **Scientific Diving**
  - ***Marine Studies***
    - Biology
    - Geology
    - Archeology
    - Other sciences
  - ***Polar Ice Diving***
- **Public Safety Diving**
  - ***Rescue Operations***
- **Recreational Diving**
  - ***Sport-Diving Instructors and Tour Guides***

TYPES OF DIVING AND DIVING EQUIPMENT

There are several general types of diving, each using different equipment and having different logistical support needs. From the least to the most sophisticated equipment used, the types of diving are breath-hold diving, open-circuit scuba diving, RB diving (closed-circuit and semiclosed-circuit diving), and surface-supplied (tethered) diving. Mixed gas and technical diving, saturation diving, and one-atmosphere diving are also discussed later.

BREATH-HOLD DIVING

Breath-hold diving is the simplest and oldest form of underwater activity, dating back thousands of years. In breath-hold diving, no supplemental air source or underwater breathing device is used, so submergence is limited to the length of time the diver can hold the breath. There are several types of breath-hold diving, each characterized by activity and equipment.

Snorkeling is the most common form of breath-hold diving. Snorkelers typically use a face mask to facilitate underwater vision, fins for propulsion, a snorkel to breathe air while swimming facedown on the water's surface, attire for environmental protection (e.g., a neoprene wetsuit or full-body spandex [Lycra] suit), and sometimes lead weights to counterbalance the positive buoyancy of a wetsuit or one's innate positive buoyancy. Snorkelers remain mostly on the surface, breathing through the snorkel with face submerged, with little actual diving under the surface. Snorkeling is widely practiced at tropical resorts to introduce people to the beauty of coral reefs.

Freediving generally refers to one of several types of competitive breath-hold diving. Freediving is classified as an "extreme sport," and many consider it to be the original extreme sport. Competitive freediving dates back to at least the early 1900s, with perhaps the best recorded account involving the Greek sponge diver Giorgios Haggi Statti. In 1913, he was offered a few dollars to dive more than 61 msw (200 fsw) underwater to retrieve the anchor of the Italian ship *Regina Margherita* that had become stuck in the Aegean Sea at a depth of 70 msw (230 fsw). He freed the anchor after three consecutive dives of between 1.5 and 3.5 minutes' duration, diving as deep as 80 msw (263 fsw). He did not consider this an especially taxing feat, saying that he had dived as deep as 110 msw (361 fsw) and stayed underwater for as long as 7 minutes on other occasions. Some consider Haggi Statti to be the "father of freediving." However, it was not until Jacques Mayol of France dived to 101 msw (331 fsw) in 1976, considered a stunning feat at the time, that freediving really began to grow in popularity as an extreme sport. In 1988, the film *The Big Blue* portrayed the lives of Jacques Mayol and Italian Enzo Maiorca as competitive freedivers and further popularized the sport. Extreme no limits freediver record holders, known as the world's deepest man and woman, are Herbert Nitsch (253.2 msw; 831.8 fsw) and Tanya Streeter (160 msw; 525 fsw).

Competitive freediving attracts athletes from around the world and is regularly featured on sports television channels. The sport is governed by the Association Internationale pour le Développement de l'Apnée (also known as the International Association for the Development of Apnea [AIDA; aida-international.org/]) or the International Association for the Development of Freediving. Since 1992, AIDA has been the officiating body for freediving, setting standards and recognizing records. AIDA recognizes eight types of freediving and breath holding; descriptions of these disciplines and the world records for each can be found in Table 71-1.

Medical Problems of Breath-Hold Diving

The major medical concern of breath-hold diving is development of hypoxia leading to loss of consciousness and drowning, especially if submergence is preceded by hyperventilation (see Hyperventilation and Shallow Water Blackout, later). Breath-hold divers also may become hypothermic, become entangled in underwater debris (e.g., fishing line, ropes, and cables) or vegetation, be harmed by marine animals, or be injured by boats or other watercraft. Divers are also subject to barotrauma of the ears, sinuses, and lungs, as described later. Although it is a very rare occurrence, breath-hold divers also can suffer from DCS (see Decompression Sickness, later).

SCUBA DIVING

Scuba diving uses a cylinder fitted with a single-hose, two-stage regulator that supplies compressed air to the diver at a pressure equal to ambient water pressure. Dive cylinders are available in a range of sizes and are predominantly made of aluminum or steel. Common volumes are 80 cubic feet of filtered, oil-free compressed air pressurized to approximately 3000 pounds per square inch gauge (psig; 250 bar). Compressed air in a full cylinder weighs approximately 6 pounds, which affects the buoyancy of the cylinder toward the end of the dive. High-pressure steel cylinders are pumped to 3500 psi and are heavier than their aluminum counterparts of similar volume.

The regulator reduces pressure in two stages. The first stage is attached to the cylinder and makes an initial reduction in pressure from high (3000 psi) to intermediate pressure (145 psi) delivered to the lower-pressure second stage attached to the diver's mouthpiece.

Like the snorkeler, the scuba diver wears a face mask covering the eyes and nose. Full-face masks cover the entire face to allow underwater vision and communication. Fins are donned for propulsion; a thermal protection suit, weight belt, and buoyancy compensator combine to adjust buoyancy underwater and for surface flotation in case of an emergency. Dive computers that track time and depth underwater have largely replaced wristwatches and depth gauges. A compass and dive light may be worn as auxiliary equipment, but a submersible pressure gauge is required to monitor air consumption. Because of the higher thermal conductivity of water, divers typically wear neoprene wetsuits to stay warm (as well as to provide protection from

TABLE 71-1 AIDA Competitive Freediving World Records (as of July 2016)

Discipline	Description	World Record: Male	World Record: Female
Pool			
Static apnea	Resting, immersed breath holding in controlled water (pool)	Stephane Mifsud 11 min 35 sec 06/2009	Natalia Molchanova 9 min 02 sec 06/2013
Dynamic apnea, without fins	Horizontal swim in controlled water	Mateusz Malina 226 m 11/2014	Natalia Molchanova 182 m 06/2013
Dynamic apnea, with fins	Horizontal swim in controlled water	Mateusz Malina 285 m 05/2016	Natalia Molchanova 237 m 09/2014
Ocean			
Constant weight, without fins	Vertical self-propelled swimming to a maximum depth and back to the surface without a line	William Trubridge 101 m 12/2010	Sayuri Kinoshita 72 m 04/2016
Constant weight, with fins	Vertical self-propelled swimming to a maximum depth and back to the surface without a line	Alexey Molchanov 128 m 09/2013	Natalia Molchanova 101 m 09/2011
Free immersion	Vertical excursion propelled by pulling on the line during descent and ascent; no fins	William Trubridge 121 m 04/2011	Natalia Molchanova 91 m 09/2013
Variable weight	Vertical descent to a maximum depth on weighted sled; ascent by pulling up the line with kicking	Stavros Kastrinakis 146 m 11/2015	Nanja Van Den Broek 130 m 10/2015
No limits	Vertical descent to a maximum depth on a weighted sled; ascent with a lift bag deployed by the diver	Herbert Nitsch 214 m 06/2007	Tanya Streeter 160 m 08/2002

AIDA, Association Internationale pour le Développement de l'Apnée.

sunburn, scrapes, and stinging marine life), even in relatively warm tropical oceans. These suits maintain a layer of water warmed by body heat between the skin and suit. The suits are typically 7 mm in thickness when diving in temperate water, and 2 to 3 mm (or Lycra) when diving in warm tropical water. An impermeable drysuit and undergarments are usually worn when diving in water colder than 10° C (50° F). Drygloves attached to the drysuit provide increased thermal protection for the hands, which is an important safety consideration in polar diving. Increasingly, electrically heated undergarments (e.g., BlueHeat by Diving Unlimited International) worn underneath drysuits are available to use as adjuncts to passive thermal protection. In addition to the preceding basic equipment, additional equipment (e.g., dive knife, camera, spear gun, and/or game bags) may be needed for safety, navigation, communication, or other purposes.

REBREATHER DIVING

Although the first self-contained underwater dive is thought to have been as a scuba dive, it was actually first done with the assistance of a RB in the 1880s. RB devices for diving were perfected over the years, but with the advent of scuba in the 1940s, RBs were relegated primarily to military operations. In the 1970s, scientific diving research using RBs was successfully performed from saturation habitats, such as by the nationally sponsored Scientist-in-the-Sea programs TEKTITE I and II.[65] Beginning in the 1990s, use of RBs started to increase in technical recreational diving (especially for underwater photography and cave diving).

RBs are used in recreational, technical, and military diving activities and have a place in the scientific diving community's underwater research toolbox. Current RBs mandate continuous attention and monitoring of equipment life-support functions that may detract from the purpose of diving missions. The amount of time invested in training, maintaining equipment before and after a dive, and keeping up the skill level requirements of advanced RBs is generally not realistic for broad application within the recreational and scientific diving communities. Furthermore, the commercial industry rarely uses this technology. More recently, the diving industry has recognized that although there will always be a limited universe of technical RB divers, nontechnical RB divers are needed to meet the needs of the scientific diving community. RBs have in some cases been demonstrated to be a powerful tool for extended range and technical diving. There also exists an extraordinary potential to extend bottom times and diving activities in depths less than 30 msw (99 fsw), coupled with a reduced logistical footprint for remote site diving.

Rebreather Devices

A brown paper bag can be considered the simplest form of RB. Inhaling from, and exhaling into, a bag allows two things to occur, neither of which is physiologically advantageous: (1) Carbon dioxide accumulates, and (2) the air becomes hypoxic. A RB is an underwater life-support system that consists of one-way valves to ensure unidirectional gas flow, a counterlung, and the ability to remove carbon dioxide through a scrubber (soda lime) and replace it with oxygen. There is a finite absorbing capacity of scrubbers, requiring periodic replacement. Scrubbers are prone to installation and packing errors, although new models exist as prepacked canisters.

Some publications comprehensively discuss the types of RBs currently available for recreational, technical, commercial, scientific, and military diving; the history of RBs; the applicable physics, physiology, and theory of RB diving; operational predive, dive, and postdive procedures; and RB maintenance.[243,278]

Oxygen Rebreather. The oxygen RB supplies pure oxygen via a demand valve that feeds into the whole breathing loop. There is depth- and time-dependent potential for oxygen toxicity. Generally, a maximum partial pressure of oxygen (PO_2) of 1.6 ATA is not to be exceeded due to increased potential for oxygen toxicity to result in a seizure. The maximum operating depth for breathing 100% oxygen would be 6 msw (19 fsw) for a single exposure of 45 minutes (per NOAA limits).

Semiclosed-Circuit Rebreather. A semiclosed-circuit RB uses a nitrox mix supplied via a constant mass flow regulator and demand valve. A quantity of gas is periodically bled into the water column. The PO2 can be variable and uncertain, and can change with the workload and depth.

Manual Closed-Circuit Rebreather. The manual closed-circuit RB consists of a source of diluent gas and oxygen supplied via a constant mass flow regulator; it can be adjusted manually as well. Triple-redundant oxygen sensors with a PO_2 display warrant careful attention because the cells are prone to failure. No gas escapes into the water column. Normal operation of this relatively simple life-support system relies on diligence of the diver.

Electronic Closed-Circuit Rebreather. The electronic closed-circuit RB system consists of a source of diluent gas, oxygen source, and battery-powered microprocessor. Triple-redundant oxygen sensors monitor PO_2 in the loop. From these data the microprocessor sends information to a solenoid valve, triggering when to open to add more oxygen into the system. Normal operation requires little diver input, but this is a complicated, electronically managed system that can foster complacency and has many failure points.

Open-Circuit Scuba System versus Rebreather Approach

Several helpful presentations comparing open-circuit scuba systems with RBs were made at the Rebreather Forum 3 in Orlando, Florida, on May 18 to 20, 2012; Lang and Steller have summarized the presentations discussed here in their report on the AAUS Rebreather Colloquium in Monterey, California, in September of that year.[175a] Simon Mitchell of the University of Auckland discussed a 90-msw/20-minute dive.[198a]

Open-Circuit Air Approach. The narcotic effect of nitrogen would incapacitate the diver at this depth of 10 ATA, so that work could not be accomplished with compressed air. The inspired PO_2 at depth would be 2.1 ATA (1.3 ATA is the generally accepted advisable maximum for technical diving) with the concomitant risk of oxygen toxicity. The gas density would be quite high at 13 g/L (8 g/L is often considered the advisable maximum), rendering the work of breathing very high. Further, decompression on air is known to be very inefficient. For the bottom depth of this profile, one would ideally use a helium-based mix (trimix 13:47, where 13% O_2 gives a PO_2 of 1.3 ATA, 40% N_2 gives an equivalent narcosis level as an air dive to 40 m, and the 47% balance will be He). Decompression would ideally consist of EAN_{36} (36% O_2 and the balance N_2) from 27 m, and 100% O_2 from 6 m. The total run time for this open-circuit dive would be 131 minutes and 18 seconds. Gas volumes required for such a dive are calculated by multiplying the ambient pressure by the time by the surface air consumption rate for each of the depth levels, and then adding the totals and including a 1.3 safety factor. For a 90-m/20-minute profile with the decompression as described, a dive would require 7852 L of trimix (expensive He), 1365 L of EAN_{36}, and 991 L of O_2. The big problem with the open-circuit scuba system for this dive profile is that it takes longer to decompress because the optimal PO_2 (1.3 ATA) is not breathed at each stage of decompression on ascent.[175a]

Rebreather Approach. Using a RB eliminates the need to carry multiple bottles for various stages of the dive profile. No bubbles are emitted from the unit, warm humidified gas is breathed, there is minimal gas consumption (which is important on deeper dives), the optimal mix is always available during descent and ascent of a dive, and the RB allows for constantly optimal PO_2 diving. The logistical aspects of a 90-msw/20-minute RB dive are more complex than those for an open-circuit dive. Besides the dive platform and topside support, bailout gas requirements must be planned as they would be for open-circuit diving. Checklists are imperative; they outline a step-by-step procedure for preparing a RB before a dive to ensure the unit will perform for the planned dive. Going through the checklists demands no rushing, no shortcuts, and no distractions, and there must be methodical, meticulous attention to detail. The carbon dioxide scrubber needs to be packed and installed, and the following items checked: one-way valves, general assembly of the unit, positive and negative pressure, diluent and oxygen pressures, and sensor calibrations.

When discussing the procedures for the four phases of an RB dive, Simon Mitchell also observed that the diver must perform the final RB check and ensure that the cylinders are open; the set point for oxygen is appropriate for the surface and the descent; the mouthpiece is closed when it is out of the mouth; a leak check is done; and a minimal loop volume is maintained (poor buoyancy is exhibited with a high loop volume); if the loop is open, it can be flooded and buoyancy lost. On descent, one must ensure that the ears are cleared, buoyancy adjustments are made, situational awareness is maintained, PO_2 and diluent are checked to maintain the loop volume, and the set point is changed at some time during the dive. Once on the bottom, one must ensure that the bailout is checked and working, set point is changed to a bottom mix, and PO_2 is checked. On ascent, one must ensure that PO_2 is frequently checked, given that the diver's life depends on it; minimal loop volume is maintained; buoyancy is monitored; situational awareness is maintained; oxygen is manually added as needed; and the mouthpiece is shut off before removing the unit at the surface.[175a]

Andrew Fock of the Alfred Hospital Hyperbaric Service in Melbourne presented the results of his survey on RB fatalities at the forum.[113a] Data were taken from the Internet, and in some cases, the information was incomplete and/or unconfirmed. Actual numbers of fatalities and accidents are unknown because of lack of reporting, and there is also uncertainty about numbers of RB divers worldwide, RB dives logged per year, and numbers of RB units currently being used. Data therefore are estimates taken from the Rebreather World website, a Dutch RB survey, a Diver Mole survey, and a British Slovakia Club survey. For the time frame of 1998 to 2010, the following observations were provided:

1. The increased potential for RB diving accidents includes high-risk behavior and RB unit cleaning and assembling procedures.
2. Approximately 20 RB deaths per year were reported worldwide from 2005 to 2010, with the top three causes identified as hypoxia, carbon dioxide intoxication, and oxygen-induced seizure.
3. Consideration of the relative safety of manual and electronic RB units acknowledges that manual units have no electronic failure potential because they are human operated. However, divers are required to know the PO_2 at all times. Electronic units monitor PO_2 without distraction, which allows divers to become complacent and not worry about PO_2. It is an accepted fact that both systems have the potential to fail.
4. The number of deaths involving manual and electronic units appear to be proportional to the current market share of each type of unit. No one brand of RB is more dangerous than another.
5. Proper training and understanding of physics and physiology will make RB diving safer. It is clear that progress is being made toward a simpler, more robust RB.[175a]

William Stone of Stone Aerospace in Austin, Texas, shared his views of three truths about RBs[266a]:

1. Sensors are the "eyeballs" of any autonomous system; if you cannot see what is going on, you are headed for trouble. This translates to RB reliability and user safety. Being aware of the PO2 and autocalibration are essential.
2. Redundancy paths must exist for all critical systems. There must be a clear and simple abort mechanism to a safe haven with no accumulation of a need for decompression.
3. The industry should mine data and learn more about the RB to better assess the nature of the triggers.[175a]

Finally, Thalmann remarked, "A scuba regulator is the steam engine of diving gear. It has been around for a long time. It has been honed to a fine art and is incredibly reliable. By comparison, a rebreather is like a space shuttle."[269a] RBs are a form of reemerging technology for the scientific, recreational, and technical diving communities, first used extensively for underwater research in TEKTITE II.[65] Notwithstanding this early RB use, the vast majority of the experience of the scientific and recreational diving communities is with no-decompression, open-circuit scuba systems.[193a]

Disappointment about the failure to replace standard open-circuit scuba systems with RBs since 1970 has been expressed by many. RBs appear to be more expensive, but one must ask whether they truly are, because with them, one can accomplish twice the work in a given unit of time and carry out investigations or missions that take more bottom time than one has with open-circuit scuba systems.

Mechanical failure is more likely with RBs than with open-circuit scuba systems, but the risk is mitigated because RBs have an extra underwater breathing apparatus. RB diving is more dangerous by an order of magnitude than is open-circuit diving. RBs have facilitated many fabulous dives for research and discovery; however, RBs are very complex and being used by fallible humans in a hostile nonrespiratory environment that has the potential to create many problems. Most mishaps are preventable; this fact emphasizes the importance of following proper procedures, including those for assembly and maintenance of equipment, and using checklists.[175a]

SURFACE-SUPPLIED (TETHERED) DIVING

Surface-supplied (tethered) diving uses several different technologic systems; in all of them, the diver breathes gas (compressed air or mixed gas) supplied by a hose from a surface source or a diving bell at a pressure equal to the ambient water pressure.

The best-known form of surface-supplied diving is classic hardhat diving, which is sometimes called mud diving or blackwater diving because it is often done in harbors with a muddy bottom and in dirty water with almost no visibility. In this type of diving, which was portrayed in the 2000 movie *Men of Honor,* the diver wears a large bronze helmet with glass faceplates, canvas suit, weight belt and weighted shoes, and other gear. Altogether, the traditional hardhat diver's gear weighs 87 kg (192 lb). Although traditional hardhat gear is still used in a variety of settings, most surface-supplied divers use modern gear that is not as heavy or cumbersome as traditional gear has communication capabilities, and can keep the diver warm.

Surface-supplied diving is most often used in commercial or military settings and can be combined with hot-water suits. It is frequently performed in arduous circumstances. The diver often operates in total darkness in cold water or in clear offshore waters where oil platforms are located. Working against a current or surge and performing tasks primarily by feel are commonplace.

The diving techniques of surface-supplied diving are quite different from those of scuba diving, and are not further discussed here; however, most of the physiologic and medical problems of surface-supplied divers are identical to those encountered in scuba diving.

MIXED-GAS DIVING

Diving can be done using either compressed air or mixed gas. Compressed air is most commonly used, especially with scuba systems, but there are a number of settings where mixed gas is needed or preferred.

In mixed-gas diving, a breathing mixture other than compressed air (e.g., a mixture in which the concentrations of nitrogen and oxygen have been changed or in which a different inert gas such as helium is substituted for nitrogen) is used. Mixed-gas diving can be used in surface-supplied, saturation, RB, or scuba diving modes, although historically it has been used most often in surface-supplied or saturation diving operations.

Mixed-gas diving has been used for many decades, but because of the greater logistical support required and associated greater expense and hazards, it has been used primarily in commercial, scientific, and military diving operations. This has changed in the past 15 years as technical divers have sought to go deeper and stay down longer. Increasing numbers of recreational divers now use mixed gas, especially nitrox.

Enriched Air Nitrox

Nitrox is a breathing gas mixture containing oxygen and nitrogen in concentrations different from those found in air. Nitrox was popularized when Dick Rutkowski began in 1985 to transfer this methodology from the scientific diving community to the recreational diving world after his retirement from the NOAA Diving Program. Nitrox use was the initial core training curriculum of the International Association of Nitrox and Technical Divers. More than a dozen different such mixtures have been used by recreational and scientific divers, all of which are lumped under the term *nitrox,* but the two most commonly used are the ones labeled by NOAA as nitrox I (containing 32% oxygen and 68% nitrogen, denoted as EAN_{32}) and nitrox II (containing 36% oxygen and 64% nitrogen, denoted as EAN_{36}). Each nitrox mixture, or blend, requires its own decompression parameters, bottom time limits, and maximum operating depth. Most modern-day dive computers are easily programmable in their settings for the oxygen content of a nitrox mix. Nitrox tables are used as infrequently as are air tables; most scuba training agencies have abandoned their teaching and use in favor of dive computers.

Of note, the term *nitrox* historically was used to refer to gas mixtures having less than 21% oxygen. These mixtures were used in diving habitats or other saturation diving situations in which the diver wanted to avoid, or at least lessen, the risk for oxygen toxicity. Technically, if the oxygen percentage is adjusted to greater than 21%, the mixture is called *enriched air nitrox* (EAN) or *oxygen-enriched air* (OEA), although *EAN* and *nitrox* are used interchangeably in the common parlance of divers.[133]

Beginning in the 1980s, an increasing number of recreational divers began using EAN to extend bottom time (compared with what was possible using compressed air) and reduce the risk for DCS. Many thousands of recreational EAN divers have been certified in recent years, and EAN has become the norm on many live-aboard charter dive boats.

EAN diving enthusiasts typically claim that nitrox is safer than compressed air because it carries less risk for DCS for equivalent bottom times. At relatively shallow depths, EAN diving allows considerably increased bottom times before decompression is required, when compared with compressed air diving. For instance, at 18 msw (60 fsw), EAN diving allows an extra 45 minutes (60 minutes with air, 105 minutes with EAN). However, this advantage diminishes greatly at depths beyond 30 msw (100 fsw). At 30 msw (100 fsw), EAN diving provides for only 8 extra minutes.[171]

Although diving with nitrox may lessen the risk for DCS compared with diving with compressed air, it definitely does not eliminate all risk, and EAN has risks of its own. The main concern is with central nervous system (CNS) oxygen toxicity, which usually manifests suddenly (with few, if any, prodromal symptoms) by loss of consciousness and seizures. Because of the risk for CNS oxygen toxicity at a maximum PO_2 of 1.6 ATA, EAN_{32} must not be used below 40 msw (130 fsw) and EAN_{36} is limited to a maximum operating depth of 34 msw (110 fsw).

A diver can still suffer DCS diving with EAN if he or she stays down too long, surfaces too fast, bypasses a required decompression stop, or uses the wrong nitrox decompression table or dive computer oxygen setting for the particular breathing medium. Furthermore, although nitrox may increase a diver's allowable no-decompression bottom time, this is often irrelevant because a bottom time is as much a function of gas supply as it is of the decompression limit (i.e., most scuba divers will exhaust their gas supply before reaching the no-decompression limit).

Nitrox use for recreational diving purposes is now a mainstream practice and readily available at the majority of live-aboard and dive store operations.[171] EAN has advantages and disadvantages (Box 71-2), with the advantages most likely realized in a setting that ensures adherence to safety.

Heliox

Other than nitrox, the most commonly used mixed gas is heliox, or oxy-helium, a mixture of helium and oxygen. Helium is used as the inert gas, replacing nitrogen (and thus eliminating the risk for nitrogen narcosis). With heliox, the oxygen level is reduced to prevent oxygen toxicity. Like nitrox, heliox is a generic term that applies to a number of different blends or mixtures of helium and oxygen.

BOX 71-2 Advantages and Disadvantages of Enriched Oxygen Nitrox Diving Compared with Compressed Air at Depths Between 15 and 40 msw (50 and 130 fsw)

Advantages

Decreased risk for decompression sickness
Decreased occurrence of nitrogen narcosis
Reduced residual nitrogen time
Shorter surface interval times
Reduced decompression times if maximum bottom time limits are exceeded
Reduced surface intervals between diving and flying

Disadvantages

Requires special training
Requires equipment dedicated for use with nitrox only
Increased oxidation of scuba cylinders
Possible increased rate of deterioration of equipment
Increased fire hazards
Potential for nitrox mixing and filling problems
Risk for central nervous system oxygen toxicity

Because it causes negligible, if any, narcosis and is easier to breathe at greater depths (because of reduced density), heliox is the preferred gas for commercial diving at depths beyond 40 msw (130 fsw). The major problems with helium are its expense (which precludes its widespread use in recreational or scientific diving), greater thermal conductivity, and hindrance of speech. In commercial and military settings, helium speech unscramblers are typically used. However, at depths beyond 183 msw (600 fsw), and especially with rapid descent, helium causes a poorly understood condition known as *high-pressure nervous syndrome* (HPNS). HPNS is characterized by dizziness, nausea, vomiting, postural and intention tremors, fatigue, somnolence, myoclonic jerking, stomach cramps, numbness, and sleep disturbances.[17] HPNS is a major barrier to prolonged manned undersea activity at depths beyond 183 msw (600 fsw).

Trimix

Trimix is a generic term referring to mixtures of helium, nitrogen, and oxygen. This breathing medium was pioneered by military and commercial diving interests for operations at depths greater than those possible by diving with compressed air. Helium replaces some of both the nitrogen and oxygen in an effort to eliminate or minimize nitrogen narcosis and to prevent CNS oxygen toxicity. The precise concentrations of helium, nitrogen, and oxygen used in trimix vary according to the specific depth profile of the dive. Obviously, in deep-diving operations, the percentages of both nitrogen and oxygen will be much less than those present in air, which means that a "travel" gas mixture is needed for breathing in shallower depths that must be traversed to get to the depth at which the trimix will be used. Currently, the U.S. Navy specifies the use of trimix for diving at depths greater than 58 msw (190 fsw), and trimix is typically used in extreme-depth (> 183 msw [600 fsw]) commercial diving because the addition of small amounts of nitrogen partially mitigates the occurrence of HPNS.

A spin-off of trimix that has begun to be used in recreational diving is an oxygen-enriched trimix, or *helitrox*. A trimix blend commonly used in this setting is 26% oxygen, 17% helium, and 57% nitrogen (trimix 26/17). Helitrox advocates promote its use for diving to depths of up to 46 msw (150 fsw), either using helitrox to decompress or switching to pure oxygen at the 6-msw (20-fsw) decompression stop.

A new world record for open-circuit scuba diving using trimix was set by the South African Nuno Gomes in June 2005 when he dived to 318.25 msw (1044 fsw) in the Red Sea. It took 20 minutes to descend to depth, incurring more than 12 hours of decompression on ascent. A total of nine different gas mixes were used for the dive (4 trimix, 3 nitrox, 1 air, and 1 oxygen). Also in June 2005, Pascal Bernabé of France dived to 330 msw (1100 fsw) off Propriano, Corsica, using an open-circuit scuba system (an event unwitnessed by Guinness). His descent time was only 10 minutes, requiring decompression of 9 hours. On 19 September 2014, Ahmed Gabr, a 41-year-old Egyptian special forces officer and technical diving instructor, recorded a Guinness World Record open-circuit scuba dive of 332.35 msw (1,090 fsw) off Dahab, Egypt. His descent time took 12 minutes to maximum depth, incurring nearly 13 hours and 35 minutes of decompression on ascent.

A high incidence of fatalities among divers using trimix has limited its use in recreational diving, but interest in its use remains high, and the recent emergence of trimix dive computers for RBs portends a much greater future use of trimix.

TECHNICAL DIVING

Beginning in the late 1980s, a small but rapidly growing number of recreational divers began to use mixed-gas systems, RBs, and other technical systems previously used only by military and commercial divers to dive deeper and stay down longer than was possible with conventional scuba systems. Technical diving (a term coined in 1990) is employed by wreck, cave, and deep decompression divers.

Technical diving represents the leading edge of underwater discovery and challenge. Its technology and techniques are at the forefront of diving developments. This type of diving is inherently more hazardous than typical recreational diving because diving deep is more hazardous, has a smaller margin for error, and relies on technology more extensively. Mitigation of these hazards has improved through training of divers and advances in equipment design.

Despite its hazards, technical diving has markedly increased in global popularity, as witnessed by establishment of the International Association of Nitrox and Technical Divers, Technical Diving International, and other technical diving associations. Technical diving conferences drawing thousands of divers have proliferated in the United States, Europe, and Asia, as have manufacturers of equipment that cater specifically to needs of the technical diving community.

SATURATION DIVING

The physiology and pathophysiology of saturation diving have been thoroughly reviewed..[38] Under pressure, the diver begins to absorb increased amounts of nitrogen or other inert gas, depending on the breathing medium, until a new equilibrium is established according to the pressure of the depth of submergence. In most deep-diving scenarios, the time needed to "off-gas," or *decompress,* inert gas upon returning to normal atmospheric pressure may be much greater than the time spent at depth. The need to minimize prolonged decompression after deep diving led to development of saturation diving. In the late 1950s, experiments by Navy diving medical officers George Bond and Robert Workman coincided with those of Jacques-Yves Cousteau and Edwin Link in the commercial sector, all of whom were working on ways to stay underwater at great depths long enough to perform useful work.[5]

The basic concept of saturation diving is that after approximately 24 hours at any given depth, the diver's tissues establish equilibrium with the gases in the breathing mixture. From that point forward, if the diver can be kept at pressure for a prolonged period, the decompression obligation remains essentially the same no matter how long the diver remains at that depth. Modern saturation complexes allow divers to live for days in large chambers at the pressure of a given work site and to be transported to the underwater site by locking into a personnel transfer capsule or sealed diving bell. When the desired depth is reached, the water pressure equals the gas pressure within the capsule and divers may exit the personnel transfer capsule into the water while breathing gas is supplied by umbilical hoses. To maintain the diver's thermal balance in the cold water found at great depths, heated water through an umbilical is circulated in a hot-water suit.

Medical considerations include the prolonged time the diver spends in isolation in a saturation chamber, exposure to potentially toxic gases and bacteria, and bubble formation during

decompression. Hyperoxia may lead to production of reactive oxygen species that interact with cell structures, causing damage to proteins, lipids, and nucleic acid. Hyperoxia and vascular gas bubble formation may lead to endothelial dysfunction.[38] Saturation diving in commercial and military applications has proven to be a safe and controlled method for working underwater, obviating the need for multiple decompressions and ascents as utilized with surface diving. To date, no long-term impact on health attributable to saturation diving has been documented.

Another application of saturation diving, used primarily for scientific purposes, uses underwater habitats. These are steel chambers situated at a given depth of water and pressurized with a compressed-gas atmosphere at the same pressure as the surrounding water. Divers may live for days or weeks in the habitat, leaving the chamber with scuba equipment (excursions) to perform studies or observe marine life in its natural state. In this specialized type of diving, rigorous precautions are taken to avoid inadvertent surfacing, thermal stress, and skin and ear infections during prolonged stays in the continuously moist environment of the habitat. Prolonged saturation decompression schedules, often taking several days, are required to return divers safely to sea level pressure upon completion of the underwater mission.

POLAR DIVING

Approximately four decades ago, scientists were first able to enter the undersea polar environment to make biologic observations for a nominal period of time. The conduct of underwater research in extreme environments, such as under ice, requires special consideration of diving physiology, equipment design, diver training, and operational procedures. Since the days when the first ice dives were made in wetsuits and double-hose regulators without buoyancy compensators or submersible pressure gauges, novel ice-diving techniques have expanded the working envelope based on the scientific need to include the use of dive computers, oxygen-enriched air, RB units, blue-water diving, and drysuit systems. The 2007 International Polar Diving Workshop in Svalbard, Norway, drew together polar diving scientists, equipment manufacturers, physiologists, decompression experts, and diving safety officers, who coordinated their expertise to form recommendations for diving in extreme conditions.[175] The polar diving environment has unique hazards associated with marine life, types of emergencies that may develop, and physiologic considerations, as discussed below.

Marine Life Hazards

Few polar animal species are considered dangerous to the diver. Southern elephant seals *(Mirounga leonina)* and Antarctic fur seals *(Arctocephalus gazella)* may become aggressive during the late spring/early summer breeding season. Crabeater seals *(Lobodon carcinophagus)* have demonstrated curiosity about divers and aggression toward humans on the surface. Leopard seals *(Hydrurga leptonyx)* have been known to attack humans on the surface and have threatened divers in the water. There is a single known in-water fatality caused by a leopard seal.[211] Should an aggressive seal approach a diver in the water, the diver's response is similar to that for a shark. Polar bears *(Ursus maritimus)* and walruses *(Odobenus rosmarus)* in the Arctic are considered predatory mammals against which diving personnel must be safeguarded. Encounters with all of the aforementioned mammals are usually restricted to areas of open water, ice edges, or pack ice. Divers in the fast ice around McMurdo Station, Antarctica, may encounter Weddell seals *(Leptonychotes weddellii)* in the water. Occasionally, a Weddell seal returning from a dive may surface to breathe in a dive hole to replenish its oxygen stores after a hypoxic diving exposure.[167] Usually, the seal will vacate the hole after it has taken a few breaths, particularly if divers are approaching from below and preparing to surface. Divers must approach such a seal with caution because an oxygen-starved seal may aggressively protect its air supply. Weddell seals protecting their surface access will often assume a head-down, tail-up posture to watch for rivals. Divers entering or exiting the water are particularly vulnerable to aggressive male Weddell seals, which tend to bite each other in the flipper and genital regions. There are no recorded incidents of killer whale *(Orcinus orca)* attacks on divers.

Polar Diving Emergencies

The best method for mitigating scuba diving emergencies is prevention. Divers must halt operations any time they become unduly stressed because of cold, fatigue, nervousness, or other physiologic reasons. Diving is also terminated if there are equipment difficulties, such as free-flowing regulators, tether-system entanglements, leaking drysuits, or buoyancy problems. Emergency situations and accidents rarely have a single major cause; more often, they result from accumulation of several minor problems. Maintaining the ability to not panic and to think clearly is the best preparation for the unexpected.

Most emergencies can be mitigated by assistance from a dive buddy; contact between two comparably equipped divers in the water is, therefore, important. Loss of contact with the dive hole may require a diver to retrace his or her path. Scanning the water column for the downline is done slowly and deliberately because the strobe light flash rate may be reduced in cold water. If the hole cannot be found, an alternate access to the surface may need to be located. There will often be open cracks at the point where fast ice touches a shoreline. Lost divers need to constantly balance a desirable lower air consumption rate in shallow water with the need for the wider field of vision available from deeper water. Maintaining safe proximity to the surface access point has made losing the dive hole an unlikely occurrence. Loss of the tether on a fast-ice dive that requires its use is one of the most serious polar diving emergencies. Lost diver search procedures are initiated immediately (i.e., assumption of a vertical position under the ice where the tethered buddy will swim a circular search pattern just under the ceiling to catch the untethered diver). The danger associated with loss of a tether in low visibility is mitigated if the divers have previously deployed a series of benthic lines. If a diver becomes disconnected from the tether downcurrent under fast ice, it may be necessary to crawl along the bottom to the downline. To clearly mark the access hole, divers deploy a well-marked downline; establish recognizable "landmarks" (such as specific ice formations) under the hole at the outset of the dive; leave a strobe light, flag, or other highly visible object on the substrate just below the hole; or shovel surface snow off the ice in a radiating spoke pattern that points the way to the dive hole.

The under-ice platelet layer can be several meters thick and become a safety concern if positively buoyant divers are trapped within this layer, become disoriented, and experience difficulty extricating themselves. The most obvious solution is to exhaust air from the drysuit to achieve negative buoyancy. If this is not possible and the platelet layer is not too thick, the diver may stand upside-down on the hard undersurface of the ice so that the head is out of the platelet ice to orient to the position of the dive hole and buddy. Another concern is that abundant platelet ice dislodged by divers will float up and plug a dive hole.

Fire is one of the greatest hazards for any operation in polar environments. Low humidity ultimately renders any wooden structure susceptible to combustion, and once a fire has started, it spreads quickly. Dive teams must exercise utmost care when using heat or open flame in a dive hut. If divers recognize during the dive that the dive hut is burning, they must terminate the dive and ascend to a safety hole or to the undersurface of the ice next to the hole (but not below it) in order to conserve air.

Physiologic Considerations in Polar Diving

Cold Water. A cold ambient temperature is the overriding limiting factor on dive operations, especially for the thermal protection and dexterity of hands. Dives are terminated before the hands become too cold to effectively operate gear or grasp a downline. Loss of dexterity can occur quickly (in 5 to 10 minutes if hands are inadequately protected). Grasping a camera, net, or other apparatus increases the rate at which a hand becomes cold. Switching the object from hand to hand or attaching it to the downline may allow the hands to rewarm. Dryglove systems and, more recently, electrically heated gloves and socks

have greatly improved thermal protection of the hands and feet. The cold environment can also cause chilling of the diver, resulting in reduced cognitive ability with progressive cooling. To avoid life-threatening hypothermia, it is important to monitor progression of the following symptoms: cold hands or feet, shivering, increased air consumption, fatigue, confusion, inability to think clearly or perform simple tasks, memory loss, reduced strength, shivering cessation while still cold, and finally, hypothermia. Heat loss occurs because of inadequate insulation, exposure of areas such as the head when a hood is inadequate, and breathing cold air. Scuba cylinder air is initially at ambient temperature and chills from expansion as it passes through the regulator. Air consumption increases as the diver cools, resulting in additional cooling with increased ventilation. Significant chilling also occurs during safety stops while the diver is not moving.

Polar diving requires greater thermal insulation, which results in decreased general mobility and increased potential for buoyancy problems. Fatigue results from increased drag, increased swimming effort, and the need to don and doff equipment.

Surface Cold Exposure. Dive teams are aware that the weather can change quickly in polar environments. While they are in the field, all divers and tenders have in their possession sufficient cold-weather clothing for protection under any circumstance. Possible circumstances include loss of vehicle power or loss of a fish hut caused by fire. Boat motor failure may strand dive teams away from the base station. Supervisors and tenders on dives conducted outdoors must also be prepared for the cooling effects of inactivity while waiting for divers to surface. In addition, food and water are part of every dive team's basic equipment. Besides serving as emergency rations, water is important for diver rehydration after the dive.

Hydration. In addition to the dehydrating effect of breathing filtered, dry, compressed air on a dive, the low humidity of the Antarctic and Arctic regions can lead rapidly and insidiously to dehydration. Continuous effort is advised to stay hydrated and maintain proper fluid balance. Urine should be copious and light colored, and diuretics (e.g., coffee, tea, and alcohol) should be avoided before a dive.

Decompression. Mueller[210] reviewed the effect of cold on decompression stress. The relative contributions of tissue nitrogen solubility and tissue perfusion to the cause of DCS are not completely resolved. Overwarming of divers, especially active warming of cold divers following a dive, may induce DCS. Therefore, divers in polar environments should avoid becoming cold during decompression and/or after the dive, and if they feel hypothermic, should wait before taking a hot shower until they have rewarmed themselves, for example, by walking. The effect of cold on bubble grades (as measured by Doppler scores) may be the same for a diver who is only slightly cold as for one who is severely hypothermic. Long-term health effects for divers with a high proportion of cold water dives is amenable to investigation.

A 2007 research report from the Navy Experimental Diving Unit entitled "The influence of thermal exposure on diver susceptibility to decompression sickness"[125] is misinterpreted by some divers to think they should be cold during the dive if they want to reduce decompression risk. This misinterpretation of the results of an exemplary study may be causing divers to unnecessarily endure uncomfortable diving conditions. There is no substitute for comfort and safety on a dive. Gerth and associates[125] questioned the conventional wisdom that cold at depth increases the risk of DCS. After conducting a carefully designed experiment, they were surprised to find that exactly the opposite was true. Some degree of cooling was beneficial, as long as the diver was warm during ascent. The temperature regimes used for their experiment were hardly reflective of operational diving conditions, and therefore extrapolation of the results is questionable in practice.

Dive computers were examined for use by scientific and recreational divers[174] and have now been effectively used in diving programs for more than three decades in lieu of U.S. Navy or other dive tables. Battery changes may be needed more frequently because of higher discharge rates in extreme cold. Advantages of dive computers over tables include their display of ascent rates, no-decompression time remaining at depth, and dive profile downloading function. Future dive computer functions should provide for additional safety and functional features.[172] Safety stops of 3 to 5 minutes between the depths of 3.3 to 10 msw (10 to 30 fsw) are required for all dives.[173]

ONE-ATMOSPHERE DIVING

Clearly, humans cannot reliably or safely function at the greatly elevated pressure of deep water, and the human factor aspects of diving have become the limiting factors in manned exploration of the ocean depths. This has led to numerous developments in one-atmosphere diving systems. In the 15th century, Leonardo da Vinci drafted schematic drawings of systems that look similar to modern one-atmosphere diving systems, but these systems had to await late 20th-century advances in metallurgy, engineering, and communications before they could become functional.

One-atmosphere absolute (ATA) diving systems are, in essence, small submarines with various types of propulsion systems and manipulators that allow the operator to work at great depth. The interior of the unit is maintained with environmental control systems to retain safe physiologic parameters. These systems range from one-person ATA suits (e.g., Exosuit by Nuytco research; nuytco.com), in which a diver can walk or "fly" through the water (Figure 71-1), to submersibles that accommodate two or more occupants (e.g., Curasub by Nuytco Research).

DIVING PHYSICS

Divers encounter many challenging environmental conditions underwater. These include cold, changes in light transmission and sound conduction, lack of air to breathe, increased density of the surrounding environment, and increased atmospheric pressure. Not surprisingly, diverse medical problems are related to diving (Box 71-3).

Of the various environmental factors affecting divers, pressure is by far the most important because it contributes either directly or indirectly to the majority of serious diving-related medical problems. Therefore, knowing the basic physics and physiologic

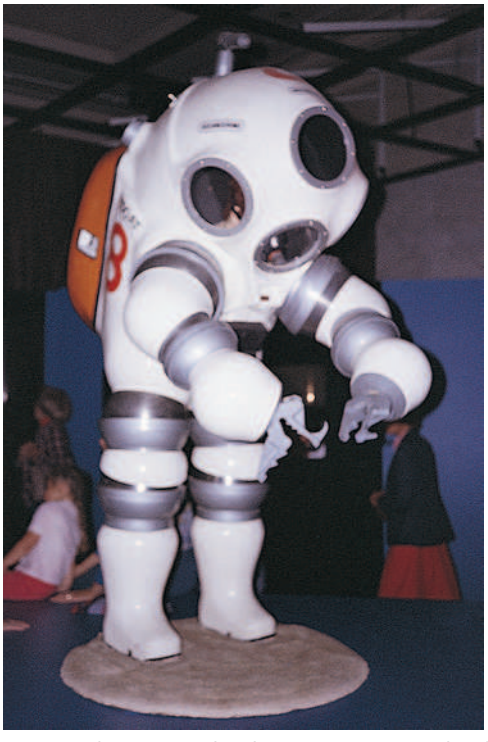

FIGURE 71-1 JIM diving suit. The diver remains at sea level pressure inside the suit and can work for prolonged periods of time at extreme depths underwater. *(Courtesy Kenneth W. Kizer, MD.)*

BOX 71-3 Medical Problems of Divers

Problems Related to Environmental Exposure
Motion sickness
Near drowning
Hypothermia
Heat illness
Sunburn
Phototoxic and photoallergic reactions
Irritant and other dermatitis
Infectious diseases
Mechanical trauma

Disorders Related to Diving
Barotrauma
Arterial gas embolism
Decompression sickness
Dysbaric osteonecrosis
Dysbaric retinopathy
Immersion pulmonary edema
Shallow water blackout

Problems Related to Breathing Gas
Inert gas narcosis
Hypoxia
Oxygen toxicity
Hypercapnia
Carbon monoxide poisoning
Lipoid pneumonitis

Problems Related to Hazardous Marine Life

Miscellaneous Problems
Hyperventilation
Hearing loss
Carotid-related blackout
Panic and other psychological problems

effects of pressure is essential to understanding and treating pressure-related disorders.

Diving-related disorders most often develop acutely because of problems caused by the mechanical effects of pressure on closed air spaces (barotrauma) or problems, such as nitrogen narcosis or DCS, caused by breathing gases at elevated partial pressure. Less often, clinical effects are delayed for months or years, as in the case of dysbaric osteonecrosis, the pathophysiology of which is not well understood.[254]

Pressure is defined as the force per unit of area. *Atmospheric pressure* is the pressure exerted by the air above the earth's surface and varies with altitude. At sea level, atmospheric pressure is 760 mm of mercury (mm Hg), or 14.7 lb per square inch (psi). *Barometric pressure* at sea level is generally referred to as 1 atmosphere (atm). *Absolute pressure* is the total barometric pressure at any point. With pressure gauges calibrated to read zero at sea level, gauge pressure is the amount of pressure greater than atmospheric pressure. In general, gauge pressure is 1 atm less than absolute atmospheric pressure. It is necessary to specify whether pressure is expressed in terms of *gauge* (psig) or *absolute pressure* (psia). Except in situations requiring laboratory precision, the following units are commonly used to express water pressure:

Feet of seawater (fsw)
Feet of fresh water (ffw)
Meters of seawater (msw)
Meters of fresh water (mfw)
Atmospheres absolute (ATA)
Pounds per square inch gauge (psig)
Pounds per square inch absolute (psia)

As a diver descends underwater, absolute pressure increases much faster than in air. Each foot (0.3048 m) of seawater exerts a force of 0.445 psig. Therefore, if the 14.7 psi pressure of 1 atm is divided by 0.445 psi per foot of seawater, the absolute pressure will have doubled at 33 fsw. In the ocean, each 33 feet of depth adds one additional atmosphere of pressure. The gauge pressure at 33 fsw is 14.7 psig (in excess of atmospheric pressure), and the absolute pressure is 29.4 psia. Because of the weight of solutes in seawater, seawater is slightly heavier (64 lb/ft^3) than is fresh water (62.4 lb/ft^3). In fresh water, 10.4 msw (34 fsw) equals one additional atmosphere of pressure.

The pressure change with increasing depth is linear, although the greatest relative change in pressure per unit of depth change occurs nearest the surface, where it doubles in the first 33 fsw. Table 71-2 lists commonly used units of pressure measurement in seawater.

When a diver submerges, the force of the tremendous weight of the water above is exerted over the entire body. Except for spaces that contain air, such as the lungs, sinuses, intestines, and middle ears, the body behaves as a liquid. The law that describes the behavior of pressure in liquids is named for the 17th-century scientist Blaise Pascal. Pascal's law states that pressure applied to any part of a fluid is transmitted equally throughout the fluid. Thus, when a diver reaches 10 msw (33 fsw), pressure on the surface of the skin and throughout the body tissues is 29.4 psia or 1520 mm Hg (Figure 71-2). The diver's body is generally unaware of this pressure, except in spaces of the body that contain air. The gases in these spaces obey Boyle's law (Figure 71-3), which states that the pressure of a given quantity of gas at constant temperature varies inversely with its volume. Thus, air in the middle ear, sinuses, lungs, and gastrointestinal tract is reduced in volume during compression or descent underwater. Inability to maintain gas pressure in these body spaces equal to the surrounding water pressure leads to various untoward mechanical effects, which are discussed below.

Because of the weight of the water exerting pressure over the chest wall, humans can breathe surface air through a snorkel or tube connected to the surface typically only to a depth of 1 to 2 feet. Attempts to breathe at greater depths through the tube are not only impossible but are dangerous, because the respiratory effort greatly augments the already physiologic negative-pressure breathing. In other words, when the respiratory muscles are relaxed at sea level, alveolar pressure is equal to surrounding air pressure. At a depth of 1 foot, the total water pressure on the chest wall is nearly 91 kg (200 lb). Because of loss of normal chest expansion and pressurization of intraalveolar air, the diver has to use forceful negative-pressure breathing to draw surface air into the lungs through the tube. Even at a depth of 1 foot, the great respiratory effort required is rapidly fatiguing, and respiration becomes impossible at further depths of only a few inches. Forced negative-pressure breathing can ultimately result in pulmonary capillary damage, with intraalveolar edema or hemorrhage. Symptoms include dyspnea and hemoptysis. Should this occur, there is no specific treatment; therapy is purely supportive.

BAROTRAUMA

Gas pressure in the various air-filled spaces of the body is normally in equilibrium with the surrounding environment. However,

TABLE 71-2 Commonly Used Units of Pressure in the Underwater Environment

Depth (fsw)	Depth (msw)	psig	psia	ATA	mm Hg (absolute)
Sea level		0.0	14.7	1	760
33	10	14.7	29.4	2	1520
66	20	29.4	44.1	3	2280
99	30	44.1	58.8	4	3040
132	40	58.8	73.5	5	3800
165	50	73.5	88.2	6	4560
198	60	88.2	102.9	7	5320
231	70	102.9	117.6	8	6080
264	81	117.6	132.3	9	6840
297	91	132.2	147.0	10	7600

ATA, atmospheres absolute; fsw, feet of seawater; msw, meters of seawater; psia, pounds per square inch absolute; psig, pounds per square inch gauge.

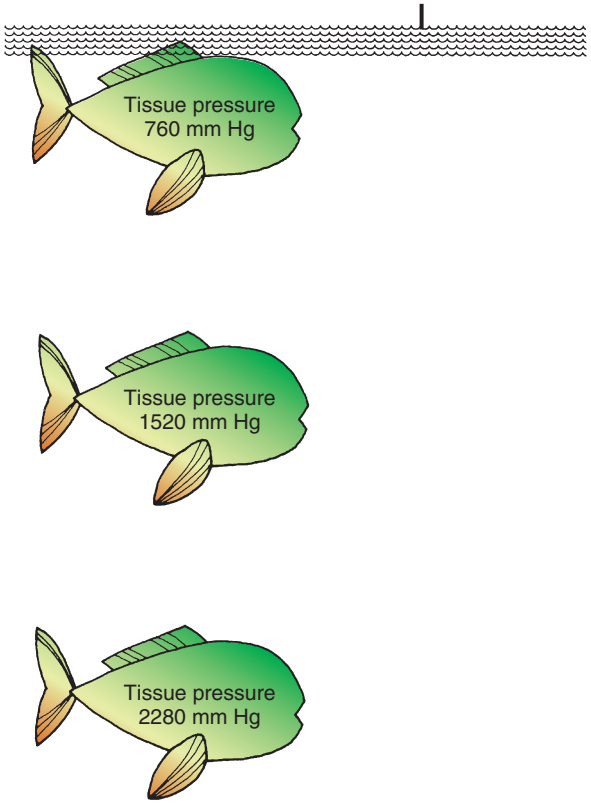

FIGURE 71-2 Pascal's law. Pressure applied to any part of a fluid is transmitted equally throughout the fluid.

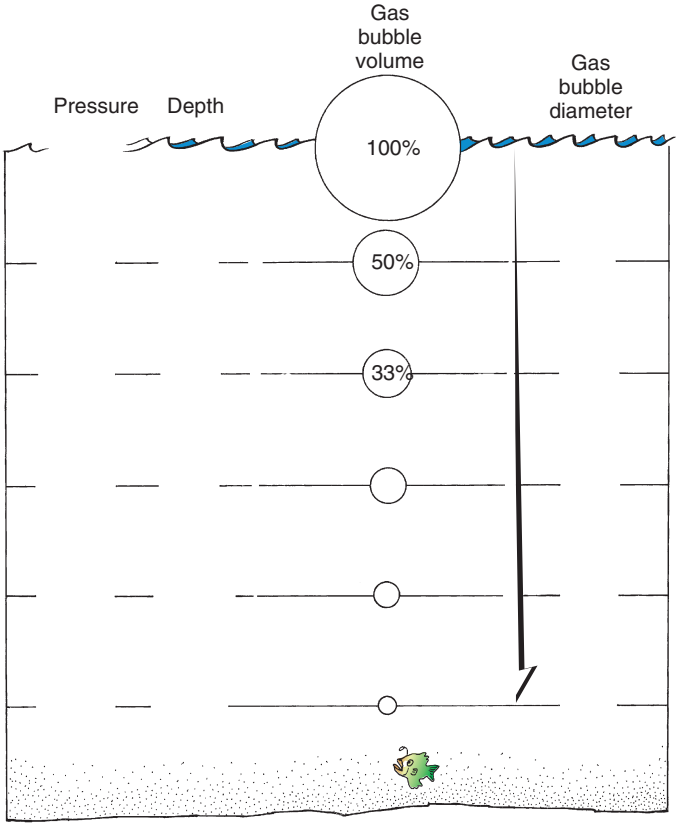

FIGURE 71-3 Boyle's law. The volume of a given quantity of gas at constant temperature varies inversely with pressure: $P_1V_1 = P_2V_2$.

if anything obstructs the passageways of gas exchange for these spaces and a change in ambient pressure occurs, pressure dysequilibrium develops. The tissue damage resulting from such pressure imbalance is known as barotrauma and commonly referred to as a *squeeze*.

Overall, barotrauma is the most common medical problem in scuba diving, potentially involving any structure or combination of structures that leads to entrapment of gas in a closed space. This includes the ears, sinuses, lungs, gastrointestinal tract, teeth, portion of the face under a face mask, and skin trapped under a fold in a drysuit.

BAROTRAUMA OF DESCENT

Mask Barotrauma

For humans to see underwater, an air space must be present between the eyes and water. In scuba diving, this is created using a face mask consisting of tempered safety glass in a soft malleable mask that seals across the forehead, on the sides of the face, and under the nose to allow nasal exhalations into the mask space to maintain air pressure inside the mask. As a diver descends in the water, the ambient pressure increases; thus, the volume within the mask decreases. The diver must add air to the gas inside the face mask to equalize the water pressure. If inexperience or inattention causes the diver to forget to maintain this balance, negative pressure in the mask can be sufficient to rupture capillaries, causing petechiae, skin ecchymosis, subconjunctival hemorrhage, lid edema, and, rarely, hyphema. This unusual condition is known as *mask barotrauma* or *squeeze* (Figure 71-4; see also Figure 48-20). Most divers with mask barotrauma are asymptomatic. The condition usually resolves over a few days to a week without any intervention, but can be treated with cold compresses and analgesics if needed. Face mask barotrauma is easily prevented simply by exhaling through the nose during descent.

In recent years, full-face masks, which are standard issue for commercial, military, and public safety divers, have become more popular with recreational divers as more models have become available (e.g., Ocean Reef, Ocean Technology Systems, Scubapro), but they are still not commonly used. A full discussion of the advantages and disadvantages of full-face masks is beyond the scope of this chapter, but these masks should be remembered as a potentially good alternative for persons who have trouble with face mask fit, face mask flooding, or feelings of claustrophobia.

Orbital hemorrhage and subperiosteal orbital hematoma from face mask barotrauma are unusual complications and can be

FIGURE 71-4 Mask barotrauma. A novice diver with mask barotrauma, showing subconjunctival hemorrhages, petechiae, and edema.

associated with diplopia, proptosis, and visual loss.[3,44,176,245,302] Although such neurologic findings after scuba diving may suggest AGE or neurologic DCS, the presence of the unmistakable stigmata of mask squeeze and a consistent history should prompt consideration of orbital hemorrhage. Under these circumstances, instead of immediate referral to a hyperbaric facility, the diver should be referred for immediate orbital CT scanning or magnetic resonance imaging (MRI) and ophthalmology consultation, because of the possibility of permanent vision loss caused by compression of the optic nerve or elevated intraocular pressure. In rare cases, surgical intervention may be necessary.[245] Recompression is contraindicated for orbital hemorrhage unless the diver also suffers from AGE or DCS.

Sinus Barotrauma

The four paired paranasal sinuses (frontal, maxillary, ethmoid, and sphenoid) have narrow connections to the nasal cavity via the sinus ostia. If there is inability to maintain the air pressure in any sinus during descent, a relative vacuum develops in the sinus cavity. This negative pressure causes congestion of the mucosal lining with subsequent edema and intramucosal bleeding and possible hematoma, hemorrhagic bullae, and bleeding into the sinus (Figure 71-5). In cases of sinus barotrauma, the diver usually experiences increasingly severe pain over the affected sinus during descent, which often causes the diver to abort the dive and return to the surface. On ascent, the remaining gas in the sinus expands and may force mucus and blood into the nose and mask.

The frontal sinus, followed by the maxillary sinus, is most commonly affected by barotrauma,. With maxillary sinus involvement, the diver often experiences pain in the maxillary teeth caused by compression of the posterior superior branch of the fifth cranial nerve, which runs along the base of the maxillary sinus. Additionally, maxillary sinus barotrauma can cause compression or ischemic neurapraxia of the infraorbital branch of the fifth cranial nerve, causing tingling and numbness of the cheek and upper lip.[43,220] Other complications include sinusitis from infection of the intrasinus fluid, or periorbital emphysema from air dissecting through the lamina papyracea from the ethmoid sinus into the orbits.[234] Unilateral optic neuropathy and a case of transient vision loss at depth from possible sphenoidal sinus barotrauma have been reported.[130,139]

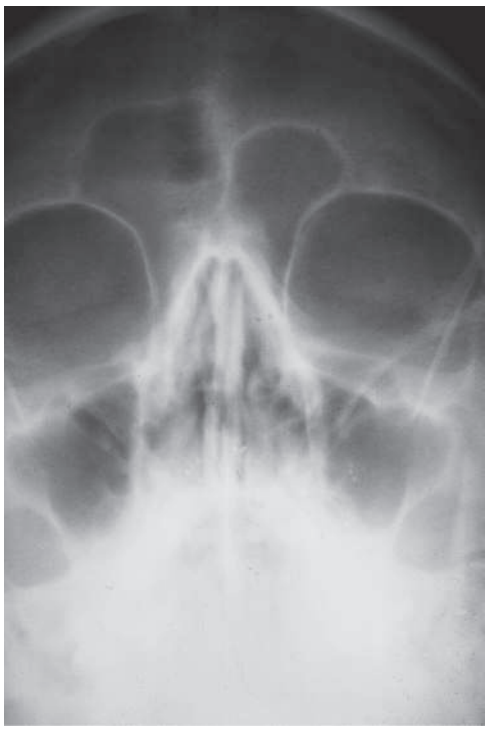

FIGURE 71-5 Frontal sinus barotrauma that first occurred 3 days earlier. Note the persistent air-fluid level. *(Courtesy Kenneth W. Kizer, MD.)*

Treatment of sinus barotrauma involves use of systemic (e.g., pseudoephedrine) and topical (e.g., phenylephrine or oxymetazoline) vasoconstrictors, analgesics, abstinence from diving until resolved, and antihistamines if needed. Antibiotics are indicated only if signs of sinusitis, including fever or purulent nasal drainage, are present. In the field, a 3- to 5-day course of corticosteroids may hasten recovery and allow an otherwise healthy diver to return to diving. On rare occasions, drainage of the affected sinus by an otolaryngologist is required for persistent pain.

Sinus barotrauma usually occurs in the setting of a diver who has an upper respiratory infection or severe allergies, or who has an anatomic deformity such as nasal polyps or a deviated septum. Divers with a history of sinusitis or middle-ear barotrauma may be more prone to sinus barotrauma.[279] Consequently, prevention of sinus barotrauma includes avoidance of diving when suffering from an upper respiratory infection, while symptomatic from allergic rhinitis, and when sinusitis, nasal polyps, or any other condition is present that impairs free flow of air from the sinus cavity to the nose. Significant nasal deformity may predispose to sinus barotrauma and warrant surgical correction to allow one to continue diving.

External Auditory Canal Barotrauma. A tight-fitting wetsuit hood or drysuit hood can trap air in the external auditory canal and potentially lead to painful external ear squeeze during descent as the volume of air is reduced according to Boyle's law. External ear canal barotrauma can occur if cerumen, exostoses, or foreign objects, such as earplugs, block the canal.

Symptoms and signs of external ear canal barotrauma include pain, swelling, erythema, petechiae, and/or hemorrhagic blebs of the ear canal wall, and possible bleeding when the diver's hood is removed. Bullae may be present in the canal and on the tympanic membrane. In very severe (and very rare) cases, the tympanic membrane can rupture from negative pressure in the ear canal. The diver, feeling pain in the canal, may believe there is inadequate equalization of the middle ear and attempt a forceful Valsalva maneuver, which increases pressure on the tympanic membrane, leading to rupture. If this occurs, further diving is contraindicated until the tympanic membrane has healed.

Treatment of ear canal barotrauma includes washing the canal with lukewarm water. Bullae should not be incised. Antibiotic drops, such as a fluoroquinolone preparation combined with hydrocortisone, should be used to prevent infection due to contamination with seawater and should always be prescribed for tympanic membrane perforation.

Ear canal barotrauma can be prevented by remembering to break the seal of the wetsuit hood to allow water to fill the external ear canal before descent. Earplugs should never be worn when scuba diving, with perhaps the exception of vented, flexible, fitted earplugs often used by freedivers to prevent ear infections (e.g., Doc's Proplugs).

Middle Ear Barotrauma. Middle ear barotrauma is the most common medical problem in scuba diving, probably affecting more than 40% of divers at one time or another.[128] The problem can be explained by direct application of Boyle's law (Figure 71-6), potentially compounded by the structure of the eustachian tube.

Boyle's law describes the inverse relationship of pressure and volume in an enclosed air space and explains why the greatest relative volume change for a given depth change occurs near the surface, where the greatest risk for middle ear squeeze occurs. As the diver descends, hydrostatic water pressure forces the tympanic membrane inward, and the volume within the middle ear cavity is reduced. The diver can add air into the middle ear through the eustachian tube, equalizing the pressure in the middle ear cavity with the external ambient pressure.

Because each foot of seawater exerts a pressure of approximately 23 mm Hg, a diver who descends 76 cm (2.5 feet) and does not equalize pressure in the middle ear will develop a relative vacuum in the middle ear because of contraction of air volume. If middle ear pressure cannot be equalized on descent, the diver should ascend several feet to minimize the pressure

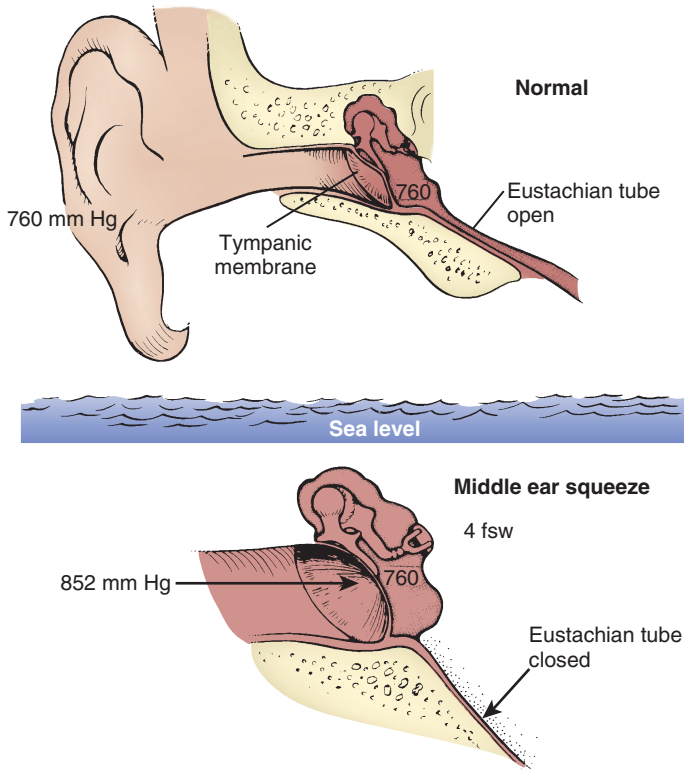

FIGURE 71-6 Middle ear barotrauma. Symptoms include fullness and pain caused by stretching of the tympanic membrane.

imbalance while attempting to equalize. Typically, the diver notices slight pain at a pressure differential of 60 mm Hg between air in the middle ear and ambient water pressure. This pressure differential causes the tympanic membrane to stretch and bulge inward, causing increasing discomfort and, eventually, severe pain. Additionally, when the tissues lining the middle ear cavity are exposed to this vacuum, vasodilation, edema, transudation, and vascular rupture occur, causing bleeding into the mucosa and middle ear cavity.

At a depth of 1.2 msw (4 fsw), a pressure differential of 90 mm Hg is generated, and the unsupported, flutter-valve, medial one-third of the eustachian tube collapses and becomes obstructed. At this point, attempts to autoinflate the middle ear by the Valsalva or Frenzel maneuver may be unsuccessful. The diver must ascend to equalize middle ear pressure with ambient environmental pressure.

If a diver does not heed the initial symptoms of middle ear barotrauma and allows the pressure differential to reach 100 to 400 mm Hg (i.e., at depths of 1.3 to 5.3 msw [4.3 to 17.4 fsw]), the pressure imbalance may lead to rupture of the tympanic membrane.[111] Symptoms of middle ear barotrauma include ear pain during descent, sensation of fullness, and reduced hearing in the affected ear. There may be mild tinnitus or vertigo. With tympanic membrane rupture, pain is relieved, but cold caloric stimulation created by seawater entering the middle ear cavity causes severe vertigo with nausea, vomiting, and disorientation underwater. The vertigo may resolve after a few minutes or continue for hours after surfacing.

The otoscopic appearance of the tympanic membrane in cases of middle ear barotrauma varies with the severity of injury. A commonly used grading scheme is the TEED classification, which grades severity according to the amount of hemorrhage in the tympanic membrane (Table 71-3).[102] Each higher grade tends to be more painful than the preceding one, except for grade 5, which may be relatively painless. With grade 5, cessation of pain corresponds with the membrane tearing, which immediately equalizes pressure in the middle ear with the external environment. Use of this grading scheme facilitates communication when describing these injuries.

In addition to having an abnormal-appearing tympanic membrane, persons with middle ear barotrauma occasionally have a small amount of bloody drainage around the nose or mouth. Audiometry usually reveals conductive hearing loss.

Middle ear barotrauma should be treated with decongestants and analgesics, although most cases clear spontaneously in 3 to 7 days without complication. Antihistamines may be used if the eustachian tube dysfunction has an allergic component. Divers should abstain from diving until the condition has resolved. Combining an oral decongestant with a long-acting topical nasal spray (such as 0.5% oxymetazoline or phenylephrine) for the first few days is usually most effective. Repeated gentle autoinflation of the middle ear by using the Frenzel maneuver also can help to displace any collection of middle ear fluid through the eustachian tube. If the tympanic membrane has ruptured, antibiotic drops, such as a fluoroquinolone preparation combined with hydrocortisone, should be used to prevent infection due to contamination with seawater. The diver should refrain from diving until the tympanic membrane is fully healed. The majority of tympanic membrane perforations from diving heal spontaneously without complications in 1 to 3 months. Surgical repair can be considered if healing has not occurred by 1 month.

Prevention is key for middle ear barotrauma. Training must emphasize the importance of early and frequent pressure equalization techniques. The diver should start equalizing immediately upon descending below the surface. Without equalization, the eustachian tube may become "locked" by 1.2 msw (4 fsw), as described earlier.

There are several maneuvers for equalizing pressure in the middle ear. The *Valsalva maneuver* involves blowing with an open glottis against closed lips and nostrils to increase pressure in the nasopharynx to inflate the middle ear through the eustachian tube. This may force open a collapsed eustachian tube. The *Toynbee maneuver* is performed by swallowing with a closed glottis while the lips are closed and nostrils pinched. The *Frenzel maneuver* is performed by pinching the nose, closing the glottis, and keeping the mouth closed while moving the jaw forward and down. This moves the pharyngeal muscles, which open the eustachian tube. This does not increase intracranial pressure. Descending feet first underwater also makes it easier to equalize pressure in the middle ear.

Divers who understand the pathophysiology of middle ear barotrauma generally take steps to inflate the middle ear immediately while submerging and thereby prevent the problem as they descend in the water. If middle ear pressure is kept equal to or greater than water pressure, no problem should occur. However, if the diver forgets to inflate the middle ear or suffers from eustachian tube dysfunction (caused by mucosal congestion due to upper respiratory infection, allergies, smoking, mucosal polyps, excessively vigorous autoinflation maneuvers, or previous maxillofacial trauma), middle ear barotrauma may occur. This most often happens just after the diver leaves the surface, with the diver complaining of ear fullness or pain. Generally, the pain rapidly becomes so severe that the diver either corrects the problem or aborts the dive.

Topical and oral decongestants are often used before diving to facilitate clearing the ears.[227] Pseudoephedrine has been

TABLE 71-3 TEED Grading System for Middle Ear Barotrauma

Grade	Description
0	Symptoms without otologic findings
1	Erythema and mild retraction of the tympanic membrane
2	Erythema of the tympanic membrane with mild or spotty hemorrhage within the membrane
3	Gross hemorrhage throughout the tympanic membrane
4	Grade 3 changes plus gross hemorrhage within the middle ear (hemotympanum)
5	Free blood in the middle ear plus perforation of the tympanic membrane

reported to reduce the incidence and severity of middle ear barotrauma in novice divers.[36] Using intranasal surfactant has been suggested to improve eustachian tube function to prevent middle ear barotrauma during repetitive diving.[97] Divers are sometimes taught not to use decongestants before diving because of theoretical concern about the medication wearing off while diving and causing problems during ascent; however, no data support this concern, and judicious use of oral or nasal decongestants can facilitate pressure equalization.

Inner Ear Barotrauma. A serious but relatively unusual form of aural barotrauma is inner ear barotrauma in the form of labyrinthine window rupture. This is the most serious form of aural barotrauma because of possible injury to the cochleovestibular system, which may lead to permanent deafness or vestibular dysfunction.[100,110]

Inner ear barotrauma results from rapid development of markedly different pressures between the middle and inner ear, as may occur from an overly forceful Valsalva maneuver or an exceptionally rapid descent, during which middle ear pressure is not adequately equalized. During descent, the tympanic membrane is pressed inward, pushing the stapes against the oval window. Perilymph and endolymph are not compressible; the resulting increased pressure causes the round window to bulge outward. The diver may attempt a forceful Valsalva maneuver to equalize the middle ear. This raises intracranial pressure, which is propagated through the perilymphatic duct to the inner ear, causing the round or oval window to rupture. This pressure dysequilibrium may cause several types of injury to the cochleovestibular apparatus, including hemorrhage within the inner ear; rupture of Reissner's membrane, leading to mixing of endolymph and perilymph; fistulation of the oval or round window, with development of a perilymph leak; or a mixed injury involving any or all of these conditions.[226] During ascent, the expanding middle ear gas may be forced through the perilymph fistula and enter either into the scala tympani or scala vestibule, which may damage cochlear or vestibular structures, leading to permanent hearing loss.

The classic triad of symptoms indicating inner ear barotrauma is roaring tinnitus, vertigo, and hearing loss. In addition, a feeling of fullness or "blockage" of the affected ear, nausea, vomiting, nystagmus, pallor, diaphoresis, disorientation, and/or ataxia may be present in varying degrees. Symptoms of inner ear barotrauma may develop immediately after the injury or be delayed for hours, depending on the specific damage and the diver's activities during and after the dive. Vigorous isometric exercise after a dive may complete an incipient or partial membrane rupture. Findings on physical examination may be normal or reveal signs of middle ear barotrauma or vestibular dysfunction. Audiometry may demonstrate mild to severe high-frequency sensorineural hearing loss or severe loss of all frequencies. Symptoms usually improve with time. Tinnitus tends to decline over time, and vestibular injury is centrally compensated. The diver may be left with high-pitch tone and high-frequency hearing loss.

Persons with inner ear barotrauma should be treated with bed rest (head elevated to 30 degrees), avoidance of strenuous activity or straining that can lead to increased intracranial pressure, and symptomatic measures as needed. There is a good prognosis for full recovery of hearing in 3 to 12 weeks. Labyrinthine window fistulas usually heal spontaneously, and data support initial conservative treatment.[226] Deterioration of hearing, worsening vestibular symptoms, or persistent significant vestibular symptoms after a few days heralds the need for detailed otolaryngologic evaluation and possible surgical exploration and fistula closure. No consensus exists as to how long to wait before surgical intervention. If a perilymph fistula is suspected, one recommendation is to explore the ear surgically as soon as possible or if symptomatic after 24 hours.[29] Patients with a tear in Reissner's membrane have manifestations similar to those with inner ear hemorrhage, although there will be persistent localized sensorineural hearing loss commensurate with the area of membrane tear. Management is the same.

Prevention of this condition is aimed at avoiding sudden, dramatic increases in middle ear pressure. Special emphasis during diver training and education should be placed on gentle pressure equalization. Upper respiratory tract infections and allergies reduce eustachian tube function and may precede inner ear barotrauma.

A diagnostic dilemma exists whenever a diver complains of vertigo, tinnitus, and hearing loss after diving. These symptoms are classic for labyrinthine rupture, in which case recompression is contraindicated because of the potential for further barotrauma to worsen the injury. Conversely, these symptoms may indicate a diagnosis of inner ear DCS, which requires expeditious treatment in a hyperbaric chamber. In such cases, the most important differential feature for diagnostic use on a dive boat or other diving site is a careful history as to time of onset and dive activities preceding the onset of symptoms. If symptom onset was during descent and ear clearing was difficult or impossible, requiring forcible Valsalva maneuvers, perilymph fistula is more likely. If the onset was during or after a decompression dive, DCS must be assumed and hyperbaric chamber treatment sought. In some cases, however, it simply is not possible to rule out DCS or rarely, AGE, and a "trial of pressure" in the hyperbaric chamber may be necessary.

Suit Barotrauma

If an area of the diver's skin becomes trapped under a fold or wrinkle of a drysuit, causing a closed air space, the pressure-induced contraction of air under the fold and resulting partial vacuum can cause transudation of blood through the skin. This is unusual and generally benign, although the resultant skin ecchymosis may have a dramatic appearance. Thick thermal undergarments (e.g., 400-g Thinsulate jumpsuit) worn under the drysuit in extremely cold water allow for significant squeeze prior to symptoms. Suit squeeze requires no treatment and resolves in a few days to a few weeks.

Dental Barotrauma

Dental barotrauma, or barodontalgia (tooth squeeze), is an infrequent yet dramatic type of barotrauma. This painful condition, sometimes called aerodontalgia, is caused by entrapped gas in the interior of a tooth or in the structures surrounding a tooth. The confined gas develops either positive or negative pressure relative to ambient pressure, which exerts force on surrounding sensitive dental structures and causes pain.

Barodontalgia may be caused by an array of dental conditions, including caries, defective restorations, oral tissue lacerations, recent extractions, periodontal abscesses, pulpal or apical lesions or cysts, and endodontal (root canal) therapy.[233] If a pocket of trapped air remains at sea level pressure while ambient pressure increases during descent, the tooth can implode or empty cavity fill with blood. Conversely, air that is forced into a tooth during descent can expand during ascent, causing the tooth to explode. To prevent barodontalgia, a diver should wait at least 24 hours after dental treatment (including fillings) before diving. Approximately 5% of French military divers reported dental barotrauma (fracture or loss of dental restoration) at the time of medical examination.[129]

Other causes of tooth pain associated with pressure changes are less well understood. Pulpitis or other dental infections can produce pain during a dive. Upper tooth pain associated with pressure changes should raise suspicion for a pathologic condition in the maxillary sinus.

Lung Barotrauma

Lung barotrauma, or lung squeeze, is a very unusual form of barotrauma that has been observed with breath-hold diving. Persons having this syndrome complain of shortness of breath and dyspnea after surfacing from a deep (> 30.5 msw [100 fsw]) breath-hold dive. The diver may cough up frothy blood, and a chest radiograph may show pulmonary edema. The condition is treated with supplemental oxygen and respiratory support as needed. Symptoms typically resolve within a few days; however, breath-hold fatalities have been linked to lung squeeze.

The classic understanding of lung squeeze is that it occurs when a diver descends to a depth at which the total lung volume is reduced to less than the residual volume. At this point, transpulmonic pressure exceeds intraalveolar pressure, causing

transudation of fluid or frank blood (from rupture of pulmonary capillaries) and overt manifestations of pulmonary edema and hypoxemia. According to this scenario, a breath-hold diver with a total lung volume of 6000 mL and a residual volume of 1200 mL could dive to only 6000/1200 or 5 ATA (equal to 40.2 msw [132 fsw]) before lung squeeze would occur. However, breath-hold divers have dived much deeper without apparent problems.

In 1968, Schaefer and associates reported that breath-hold divers pool their blood centrally, accumulating a central volume increase of as much as 1047 mL at 27.4 msw (90 fsw).[252] If it is assumed that this adjustment in pulmonary blood volume reduces the residual volume, then theoretically, it should be possible for the diver with a total lung volume of 6000 mL to breath-hold to 6000/(1200 − 1047 = 153 mL), or almost 40 ATA. Although deep breath-hold dives seem to support the beneficial effect of central pooling of blood, cases of lung squeeze continue to occur at much shallower depths. The exact pathophysiology of this condition remains unclear.[182]

Underwater Blast Injury

Barotrauma can be caused by underwater explosions. Shock waves from a blast are propagated farther in the dense medium of water than in air.[62] Underwater explosions may result from ordnance or ignition of explosive gases during cutting or welding operations.

Underwater blasts can cause serious injuries to divers. Body cavities that contain air, such as the lungs, intestines, ears, and sinuses, are most vulnerable. Pneumothorax, pneumomediastinum, and air embolism may result from laceration of the lungs and pleura.[146] There may be intestinal perforation, subserosal hemorrhage, and subsequent peritonitis. Subarachnoid hemorrhage and rupture of the aorta and left ventricle have been reported in diving-related fatalities caused by underwater explosions.[235] The occurrence of blast-related air embolism at depth, which worsens with ascent to the surface, requires hyperbaric treatment. Otherwise, management of underwater blast injuries is the same as for terrestrial blast injury.

BAROTRAUMA OF ASCENT

Reverse Sinus or Ear Barotrauma (Reverse Squeeze)

The sinuses and ears are subject to barotrauma during ascent as well as descent. As the ambient pressure drops, the volume of the gas within the sinus or ear cavities expands, causing pain and tissue damage if not released. Gas pressure can exceed intravascular pressure in adjacent tissue, causing local ischemia. In the sinuses, a cyst or polyp can act as a one-way valve; air enters the sinus as the diver descends but cannot escape as the diver ascends. Fainting underwater as a result of frontal sinus pain has been reported.[112]

Pain can develop in the ear during ascent because of expanding gas pressure in the middle ear that is not released through the eustachian tube. If a diver makes frequent descents and ascents during a dive or multiple short dives back-to-back (bounce diving), the mucosal lining of the eustachian tube can become inflamed, making equalization of pressure in the ears difficult. The eustachian tube may not allow air to escape fast enough, and the pressure rises in the middle ear during ascent. This results in pain and often tinnitus and vertigo, and may lead to outward rupture of the tympanic membrane.

Alternobaric Vertigo

An unusual type of aural barotrauma is alternobaric vertigo. This usually occurs with ascent and is caused by sudden development of unequal (between the two ears) middle ear pressure, which causes asymmetric vestibular stimulation and resultant pronounced vertigo.[186] Vertigo, nausea, and vomiting may occur as the diver ascends. Although usually only transient and requiring no treatment, alternobaric vertigo may precipitate a panic response, leading to drowning or pulmonary barotrauma with resultant air embolism. The incidence of diving injuries due to alternobaric vertigo is unknown. The condition can be mitigated underwater by holding onto the ascent line and/or assistance from the dive buddy. Rarely, alternobaric vertigo lasts for several hours or days, in which case it should be treated symptomatically after excluding inner ear barotrauma. Diving should be avoided when middle ear equalization is compromised.

Facial Baroparesis (Alternobaric Facial Palsy)

The seventh cranial nerve courses through the middle ear and mastoid process via a bony channel. Parts of the nerve may be directly exposed to middle ear pressures through a defect in the canal wall. During ascent, if the eustachian tube mucosa is swollen due to irritation, infection, or allergy, middle ear pressure may exceed the capillary pressure of the facial nerve and cause ischemic neuropraxia.[99,148,149,152,207] The diver may complain of ear fullness and pain after surfacing, along with facial palsy symptoms. The diver is unable to close the eye on the affected side, and the mouth may be affected. Otoscopy reveals a bulging tympanic membrane.

The diver can try the Toynbee maneuver, as described above, to release middle ear overpressure. Oral and topical decongestants should be used. The middle ear gas is eventually absorbed; this reduces the pressure. However, permanent damage of the nerve can occur if the pressure is elevated for too long. Myringotomy is the preferred treatment to remove air from the middle ear cavity.[152] Recompressing the diver to 1 msw (3 fsw) can also restore capillary circulation to the nerve. The diver's diving profile and lack of other neurologic symptoms and signs help exclude the diagnosis of AGE or DCS.

Gastrointestinal Barotrauma

Because the intestines are pliable, contraction of intraluminal bowel gas during descent does not cause barotrauma. In unusual situations, however, expanding gas can become trapped in the gastrointestinal tract during ascent and cause gastrointestinal barotrauma, which is also known as aerogastralgia.[68,185] This infrequent condition has been noted most often in novice divers, who are more prone to aerophagia; in divers who repeatedly perform the Valsalva maneuver in the head-down position, which may force air into the stomach; in those who chew gum while diving; and in divers who consume large quantities of carbonated beverages or legumes shortly before diving.

Divers with gastrointestinal barotrauma typically complain of abdominal fullness, colicky abdominal pain, belching, and flatulence. Rarely, syncope has been reported and is presumed to result from a combination of decreased venous return and vagal reflex. Most often, gas accumulates in the gastric antrum.

The physical examination of a diver having symptoms of gastrointestinal barotrauma is usually normal because the condition typically resolves by the time medical care is obtained. However, abdominal distention, tympany, and abdominal tenderness may be found. In an extreme case, there may be signs of cardiovascular compromise as a result of obstruction of venous return.[101]

Gastric rupture is uncommon and normally occurs with blunt abdominal trauma. Gastric rupture secondary to barotrauma is very rare, with only 15 cases reported in the literature.[39,146a,206,274] Air that is swallowed during the dive expands rapidly during an uncontrolled ascent, causing gastric rupture and pneumoperitoneum.[168,206] Abdominal compartment syndrome from tension pneumoperitoneum has been reported in a diver.[39] Pneumoperitoneum from gastric rupture should be treated with needle decompression prior to surgery. Pneumoperitoneum has also been reported after pulmonary barotrauma; air escaping from alveolar rupture enters the mediastinum and can progress to the abdomen through either the esophageal or aortic hiatus.[168] This can lead to a diagnostic dilemma (see section below). Gastrointestinal barotrauma is most often self-remedied by elimination of the excess gas. Hyperbaric treatment may be necessary in very rare, severe cases.

Pulmonary Barotrauma

The most serious type of barotrauma is pulmonary barotrauma of ascent, which results from expansion of gas trapped in the lungs. If a diver does not allow the expanding gas to escape, a pressure differential develops between the intrapulmonary air space and ambient pressure. The combination of overdistention of alveoli

and overpressurization causes the alveoli to rupture, producing a spectrum of injuries collectively referred to as pulmonary barotrauma.

Divers suffering pulmonary overpressurization usually report a history of rapid and uncontrolled ascent to the surface before the onset of symptoms (typically as a result of running out of air, panic, or sudden development of uncontrolled positive buoyancy, as may occur when a diver drops his or her weight belt or inadvertently inflates his or her buoyancy compensator). However, pulmonary barotrauma may occur in divers who ascend slowly with no discernible cause. In these cases, there is presumed to be localized overinflation of the lung in these cases. Underlying lung pathology could theoretically lead to air trapping. One published case reports a 25-year-old male with coccidioidomycosis who suffered AGE after a normal ascent and was found to have a right upper lobe cavitary lesion on chest CT.[106] Pulmonary barotrauma has also been reported during normal ascent in divers with bullous disease.[196]

Localized overinflation of the lungs from focally increased elastic recoil may occur in divers who ascend at a proper rate.[63,64] Theoretically, if there are focal areas of decreased compliance in the lungs, adjacent areas of normal compliance would be subjected to greater forces, leading to barotrauma.[114] With immersion in diving, central pooling of blood causes an increase in intrapulmonary blood volume, and the lungs become stiffer. This decreased compliance may increase the risk for pulmonary barotrauma.

If a given intrapulmonary gas volume is trapped by forcible breath-holding or a closed glottis, or, even in a small portion of the lung, by bronchospasm during ascent, intrapulmonary volume increases (according to Boyle's law) until the elastic limit of the chest wall is reached. After that, intrapulmonary pressure rises until, at a positive differential pressure of about 80 mm Hg, air is forced across the pulmonary capillary membrane. This air usually enters either the pulmonary interstitial spaces or pulmonary capillaries.

It is important to remember that there is significant change in barometric pressure in shallow water. Boyle's law dictates greater volume changes for a given change in depth near the surface than at greater depths. Thus, shallow depths are the most dangerous for breath-holding ascents. A pressure differential of only 80 mm Hg (alveolar air) above ambient water pressure on the chest wall, or about 1 to 1.3 msw (3 to 4 fsw) of depth underwater, is adequate to force air bubbles across the alveolar-capillary membrane. Fatal pulmonary barotrauma has occurred from breath-holding during an ascent from a depth as shallow as 1.3 msw (4 fsw).[20]

The diagnosis of pulmonary barotrauma is based on development of characteristic symptoms after diving. Actual clinical manifestations may take several forms, depending on the course traveled by the extraalveolar air. Once alveoli rupture, air can remain in the interstitium, causing localized pulmonary injury and alveolar hemorrhage. Air can travel along perivascular sheaths and dissect into the mediastinum. This air can track superiorly to the neck, resulting in subcutaneous emphysema, and can dissect inferiorly and posteriorly, causing pneumoperitoneum. Air may dissect to the visceral pleura, causing a pneumothorax. If air enters the pulmonary vasculature, it can travel to the heart and embolize systemically, causing AGE (Figure 71-7).

Clinical Manifestations of Pulmonary Barotrauma. The specific clinical manifestations of pulmonary barotrauma depend on the location and amount of air that escapes into an extraalveolar location.

Local Pulmonary Injury. Air can rupture alveoli, causing localized pulmonary injury and capillary bleeding without other signs of pulmonary barotrauma, such as pneumomediastinum or AGE. Diffuse alveolar hemorrhage has been described as a rare manifestation of pulmonary barotrauma.[8] The diver may complain of chest pain, cough, and hemoptysis without any neurologic findings. Intraparenchymal lung injury and bleeding may be seen on chest x-ray. A diver with local pulmonary injury without any evidence of AGE does not require recompression and should be treated with supportive care. However, a complete history and neurologic evaluation must be performed to be certain the diver did not have a transient episode of neurologic dysfunction immediately after the event that could herald an AGE. Subtle parietal lobe dysfunction may be the only abnormality detected by the time the diver reaches the emergency department or hyperbaric chamber.[218]

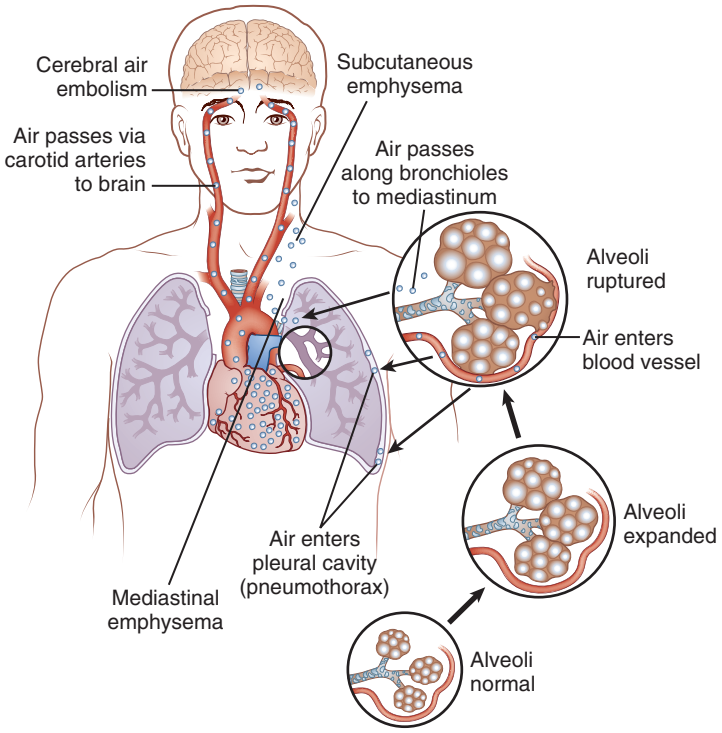

FIGURE 71-7 Pulmonary barotrauma. Diagram of different manifestations of pulmonary barotrauma, including mediastinal emphysema, subcutaneous emphysema, pneumothorax, and arterial gas embolism.

Pneumomediastinum, or mediastinal emphysema, is the most common form of pulmonary barotrauma, resulting from pulmonary interstitial air dissecting along bronchi to the mediastinum. The diver may be asymptomatic or complain of substernal chest pain. Respiratory distress is typically not present. If air dissects from the mediastinum up to the neck, the diver may experience hoarseness and neck fullness. Subcutaneous emphysema may be present and palpated as crepitance under the skin of the neck and anterior chest. In severe cases, the diver may complain of marked chest pain, dyspnea, and dysphagia. *Hamman's sign* (also known as Hamman's murmur or Hamman's crunch) is a crunching, rasping sound, synchronous with each heartbeat and heard over the precordium during auscultation of the chest. It may rarely be heard when air is present in the mediastinum around the heart. Radiographs may show extraalveolar air in the neck or mediastinum, or both, although radiographs are rarely necessary to make the diagnosis. The presence of air on a radiograph may be very subtle and is often best visualized along the pulmonary artery and aorta, and along the edge of the heart.

Treatment of pneumomediastinum is conservative, consisting of rest, avoidance of further pressure exposure (including flying in commercial aircraft), and observation. Supplemental oxygen administration may be useful in severe cases. Hyperbaric treatment is indicated only in cases associated with AGE. As mentioned above, any transient neurologic symptom in the presence of pneumomediastinum suggests AGE.

Pneumothorax is an infrequent manifestation of diving-related pulmonary barotrauma, because it requires that air be vented through the visceral pleura, a path generally having greater resistance than does air tracking through the interstitium. Although pneumothorax has been reported to occur in 5% to 10% of cases of AGE,[231] in the authors' experience its occurrence is much less frequent. Despite being infrequent, pneumothorax must be considered and excluded whenever pulmonary barotrauma or AGE is suspected, because a simple pneumothorax can become a tension pneumothorax during ascent in a hyperbaric chamber.

In cases of diving-related pneumothorax, the diver usually complains of pleuritic chest pain, breathlessness, and dyspnea, just as with pneumothorax from any other cause. Radiographs may confirm the diagnosis. Because the majority of diving-related pneumothoraces are small, treatment may consist simply of supplemental oxygen and close observation, repeating the chest x-ray at intervals to ensure resolution. Tube thoracostomy is usually reserved for a larger pneumothorax or if the diver is to undergo hyperbaric treatment. A chest tube is necessary if the diver is recompressed, because the expanding intrapleural gas of the pneumothorax cannot otherwise be vented to the environment during depressurization from hyperbaric treatment, and it may convert to a lethal tension pneumothorax.

Tension pneumothorax is one of a very few diving-specific disorders that can quickly kill a diver at the dive site (or in a hyperbaric chamber), but is curable if recognized. Although pneumothorax may result from pulmonary barotrauma, it is also associated with severe blunt chest trauma (e.g., from an underwater blast, landslide, or cave-in or from collision with a boat) or may develop spontaneously, as has been observed with other outdoor sports.[163]

Divers developing a pneumothorax underwater will almost always develop shortness of breath, dyspnea, and pleuritic chest pain, although these symptoms may not be recognized or their significance may not be recognized at the time. Shortness of breath always worsens during ascent, although the chest pain often improves during ascent as the pneumothorax enlarges and the lung no longer touches the chest wall. Breath sounds will be noted to be diminished when the chest is auscultated.

Treatment of tension pneumothorax in divers is with closed chest thoracostomy.

ARTERIAL GAS EMBOLISM

Arterial gas embolism (AGE) is the most feared complication of pulmonary barotrauma. It is one of the most dramatic and serious injuries associated with compressed-air diving and is a major cause of death and disability among recreational divers.[160] Many diver deaths officially listed as drowning probably have been cases of AGE. Unfortunately, the accident investigation and postmortem evaluation of many diving accident victims are insufficient to establish the precise cause of death. Autopsies of diving accident victims should be performed according to special procedures.[50,51,75]

PATHOPHYSIOLOGY

AGE results from air bubbles entering the pulmonary venous circulation from ruptured alveoli. When air is introduced into the pulmonary capillary blood, gas bubbles are showered into the left atrium; from the atrium, the bubbles move to the left ventricle and subsequently the aorta. From the aorta, they are distributed throughout the arterial vasculature. Bubbles may enter the coronary arteries and produce electrocardiogram (ECG) changes and elevation of cardiac enzymes, but rarely myocardial injury.[262] Myocardial dysfunction resulting from ischemia and stunning caused by gas bubbles in the coronary arteries after diving has been reported in a patient who developed acute pulmonary edema while diving.[251] Gas embolization to the coronary arteries also may induce arrhythmias.[156]

Most of the bubbles entering the aorta pass into the systemic circulation, lodging in small- and medium-sized arteries and occluding the more distal circulation. Bubbles travel up the carotid or vertebral arteries to embolize the brain, causing a combination of mechanical and reactive sequelae. Most bubbles pass through the cerebral vasculature after varying amounts of delay.[127] Occlusive bubbles lodge most frequently in small arterioles with a diameter of 30 to 60 μm, which are found at the junction between the white and gray matter.[83] Vascular occlusion causes distal ischemia, which is compounded by damage to the vascular endothelium and disruption of the blood-brain barrier, with resultant cerebral edema. Systemic hypertension occurs, cerebrospinal fluid pressure rises, and there is reactive hyperemia causing loss of autoregulation of cerebral perfusion. Cerebral blood flow now reflects changes in systemic blood flow.[109]

Bubbles damage the vascular endothelium, causing release of vasoactive substances both in the brain and lungs,[141] and adversely affect nitric oxide–mediated endothelial cell function.[37] These mechanisms explain the delayed effects of air embolization on circulatory dynamics.

Depending on the site or sites of circulatory occlusion, AGE produces myriad and often disastrous consequences. The neurologic pattern may be confusing as showers of bubbles randomly embolize the brain's circulation, producing ischemia and infarction of diverse brain regions. Combined carotid and vertebral artery embolization may produce severe, diffuse brain injury (Figure 71-8).[160]

AGE typically develops during ascent or immediately after the diver surfaces, at which time the high intrapulmonary pressure resulting from lung overpressurization is relieved, allowing bubble-laden pulmonary venous blood to return to the heart and pass into the systemic circulation. It is axiomatic that symptoms of AGE develop within 10 minutes of surfacing from a dive, although most often they are clearly evident within the first 2 minutes. Sudden loss of consciousness during ascent or upon surfacing from a dive should be considered to represent air embolism until proven otherwise.

In a pulmonary overpressure accident, as soon as normal breathing resumes at the surface of the water, the pressure differential that drives air bubbles into the pulmonary capillaries is equalized. From this point on, usually no further intraarterial air is introduced.

ARTERIAL GAS EMBOLISM AND SUDDEN DEATH

Approximately 4% of divers who suffer an AGE die immediately, presenting with sudden loss of consciousness, pulselessness, and apnea. These victims are not responsive to immediate cardiopulmonary resuscitation or recompression. It was previously thought that sudden death from AGE was caused either by reflex arrhythmias from brainstem embolization or by myocardial ischemia and

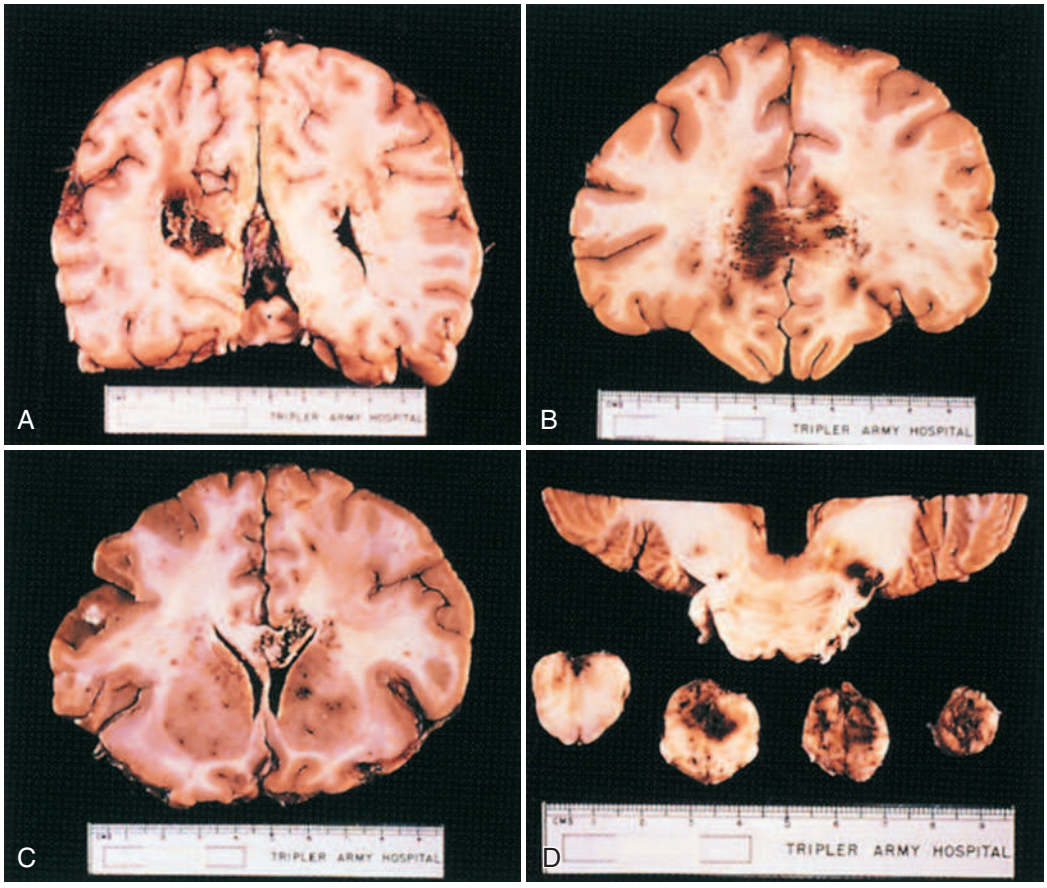

FIGURE 71-8 **A** to **D,** Cross sections of the brain of a 31-year-old male sport diver who suffered an arterial gas embolism, dying 4 days after the accident and after receiving extended hyperbaric oxygen therapy. Bubble-induced infarcts are found throughout the brain in the distribution of both the carotid and vertebral arteries. *(Courtesy Kenneth W. Kizer, MD.)*

death due to coronary artery embolization and occlusion.[48,109] However, these mechanisms do not explain what is seen in animal models or clinically in humans. Accidental injection of air into the coronary arteries of humans during cardiac catheterization does not result in sudden death. There is only one case report of myocardial infarction associated with an AGE,[66] and biochemical evidence for myocardial ischemia in cases of AGE has not been found.[262]

In sudden death from AGE, there is complete filling of the central vascular bed with air.[215] Radiographs of fatal cases of AGE demonstrate massive amounts of air in the central vasculature (Figure 71-9).[219] Autopsies typically reveal large amounts of air in the central vascular bed, particularly in the pulmonary arteries and right ventricle. Currently, it is believed that the primary mechanism of cardiac arrest in most cases of AGE is vascular obstruction caused by air, leading to pulseless electrical activity.[215,219]

CLINICAL MANIFESTATIONS

Clinical manifestations of cerebral air embolism are sudden, dramatic, and often life-threatening. Approximately 4% of victims of AGE suffer immediate cardiac respiratory arrest and die. Another 5% die in the hospital from consequences of the AGE or drowning that can accompany AGE. More than one-half of the remaining victims of AGE have complete functional recovery.

Victims of AGE present with varied neurologic and systemic signs and symptoms depending on the amount and distribution of air. Neurologic manifestations of AGE are typical of an acute stroke, although hemiplegia and other purely unilateral brain syndromes are infrequent. Loss of consciousness, monoplegia or asymmetric multiplegia, focal paralysis, paresthesias or other sensory disturbances, convulsions, aphasia, confusion, blindness or visual field defects, vertigo, dizziness, or headache is most often observed (Table 71-4).[84,109,160,197,218]

The physical findings of AGE are extremely variable and depend on the specific site or sites of vascular occlusion. Neurologic findings generally dominate the clinical picture because of the frequency of cerebral involvement. All patients with

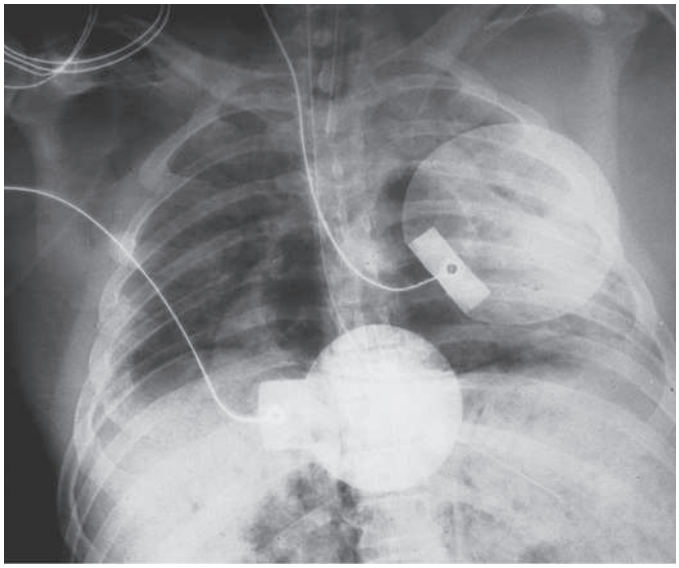

FIGURE 71-9 Chest radiograph of a diver who died suddenly from an arterial gas embolism, showing complete filling of the central vascular bed with air. Note the air in the heart and carotid arteries.

TABLE 71-4 Typical Symptoms and Signs of Pulmonary Overpressure Accident

Condition	Symptoms	Signs
Arterial gas embolism	Seizure (focal or general), unconsciousness, confusion, headache, visual disturbances, bloody sputum (rare)	Hemiplegia, monoplegia, altered level of consciousness, blindness, visual motor deficit, focal motor or sensory loss
Mediastinal-subcutaneous emphysema	Substernal pain, hoarse voice, neck swelling, dyspnea	Subcutaneous crepitus, gas patterns on radiographs of the mediastinum and neck
Pneumothorax	Chest pain, dyspnea	Loss of breath sounds, hyperresonant chest percussion, tracheal shift

suspected AGE should be carefully examined for neurologic deficits. Often, the neurologic findings are subtle and require detailed examinations, including cognitive function testing.[218] Such testing is rarely possible at the dive site, so divers who have a history suggestive of AGE (e.g., loss of consciousness after ascending from a dive) but who do not manifest any gross symptoms or signs of neurologic injury in the field should be given the benefit of the doubt and transported to the nearest emergency department for initial stabilization and referral to a hyperbaric treatment facility. Specific manifestations of pulmonary barotrauma (such as subcutaneous or mediastinal emphysema) should be carefully sought.

Persons suffering an AGE may present with various hematologic and biochemical abnormalities. Gas bubbles distribute systemically and are thought to cause direct organ injury or injury to the vascular endothelium, or both. AGE patients usually present with hemoconcentration due to plasma extravasation from endothelial injury. The degree of hemoconcentration correlates with the neurologic outcome of the diver.[264] Creatinine kinase (CK) is elevated in nearly all cases of AGE and correlates with eventual neurologic outcome of the diver.[262] The more elevated the CK, the less likely is full functional recovery. The majority of elevated CK is from skeletal muscle (the MM component). CK-MB (derived from cardiac muscle) is elevated in some cases, and nonspecific ECG changes can occur; however, true myocardial infarction due to AGE is extremely rare.[34] Even in cases of AGE with elevated CK-MB, functional studies of the heart show no evidence of wall motion abnormalities following recovery.[251,262] Bubbles cause injury to other organs. Elevated serum glutamic oxaloacetic transaminase, serum glutamic pyruvic transaminase, and lactate dehydrogenase can be found in victims of AGE.[263] Despite elevations of these enzymes, organ function usually does not decline.

AGE can lead to loss of consciousness upon surfacing; hence, it is not unexpected to find evidence of aspiration and drowning clinically and on chest x-ray. Radiographic findings consistent with aspiration can be found in more than 50% of chest x-rays of victims with AGE.[135] However, many of the same radiographic findings may be seen with venous gas embolism.[162]

Rarely, air bubbles may be visualized in the retinal arteries, or sharply circumscribed areas of glossal pallor *(Liebermeister's sign)* may be noted, but these findings cannot be relied upon to make the diagnosis. The diagnosis of AGE is clinical and based on the diving history and symptoms. Any diver who loses consciousness or presents with symptoms or signs of serious neurologic injury within 10 minutes of surfacing from a dive must be considered to have suffered an AGE.

TREATMENT

All cases of suspected AGE must be referred for hyperbaric oxygen treatment as rapidly as possible. This is the primary and essential treatment for the condition.[98]

As noted earlier, initial neurologic manifestations of AGE may resolve by the time the diver reaches a medical facility. Regardless, all patients should be referred for hyperbaric consultation if the history is suggestive of AGE, because neurologic impairment is difficult to exclude in the acute setting and subtle neurologic injuries may progress and become irreversible. Although early treatment with hyperbaric oxygen therapy (HBOT) is more likely to be efficacious than is delayed treatment, there are many reports of divers improving with HBOT after delays longer than 6 hours.[189]

DAN has a diving medicine physician available 24 hours a day, 7 days a week, who can help with diagnosis and immediate care of an injured diver and provide the location of the nearest hyperbaric treatment facility. DAN can assist with arrangement of transport and treatment for all diving injuries. In the United States, the DAN hotline number is 919-684-9111.

Prehospital Care

The affected diver should be given supplemental oxygen at a high flow rate (15 L/min by nonrebreather facemask) as soon as possible starting on the dive boat or in the field and continued during transport, emergency department evaluation, and transfer to the hyperbaric treatment facility. Supplemental oxygen enhances the rate of resolution of inert gas bubbles and treats arterial hypoxemia.

AGE patients should be maintained in the supine position, both in the field and during transport to an emergency medical treatment facility or hyperbaric chamber. Historically, much attention was directed toward keeping the AGE patient in the Trendelenburg position in the field. This was based on anecdotal reports and limited experimental data.[127,169,280] The rationale for keeping the patient with AGE in the head-down position was the belief that the weight of the column of blood would force bubbles through the cerebral capillary bed, that the buoyancy of the bubbles would keep them in the aorta or heart, and that the weight of the spinal fluid might compress bubbles in the spinal cord. These benefits were never well demonstrated or experimentally confirmed. More recent studies showed that the Trendelenburg position did not keep bubbles from being distributed to the systemic circulation.[47] Additionally, it is more difficult to oxygenate patients in a head-down position, and if someone is maintained in this position for longer than 30 to 60 minutes, it may cause or worsen cerebral edema.

Because the AGE event so often occurs while the victim is in the water, he or she frequently suffers concomitant drowning. The rescuer must be prepared to provide cardiopulmonary resuscitation and to protect the airway from aspiration of gastric contents secondary to vomiting.

While life support measures are being instituted, a member of the diving or rescue team should contact the nearest civilian or military hyperbaric treatment facility and contact an air ambulance if air evacuation is required. Aircraft selection is crucial because the stricken diver should not be exposed to a significantly lower atmospheric pressure in the aircraft. Ideally, the diver should be transported by aircraft pressurized to sea level so that any intraarterial bubbles do not further expand. In the case of helicopter evacuation or in the event that an unpressurized aircraft is required, the flight altitude must be maintained as low as possible, not to exceed 305 m (1000 feet) above sea level, if possible.

Intravenous fluid resuscitation should be started for divers with AGE. As mentioned earlier, victims of AGE present with hemoconcentration from plasma extravasation due to endothelial injury from gas bubbles. It is important to maintain adequate intravascular volume because inert gas cannot be effectively

eliminated from tissues or from intravascular bubbles at the arteriolar-capillary level without sufficient capillary perfusion. Additionally, autoregulation of blood flow in the brain is lost following AGE, and cerebral perfusion passively follows the systemic blood pressure. Hypotension should be avoided in cases of AGE. Intravenous infusion of isotonic solution should be started, and urine output maintained at 1 to 2 mL/kg/hr.[74]

The AGE-affected diver should be transported to a hyperbaric treatment facility as quickly as possible. Delay prolongs cerebral ischemia and cellular hypoxia, resulting in significant cerebral edema, which typically leads to a more difficult course of therapy. Transport should still be undertaken even if delay is unavoidable. Remarkable improvement has been seen in cases in which treatment was delayed for more than 24 hours after the onset of neurologic manifestations.[157]

If the AGE-stricken diver is first seen at a hospital emergency department or clinic, baseline laboratory tests, including hemoglobin and creatine phosphokinase, chest radiograph, and other diagnostic tests (such as an ECG) should be obtained while transport to a hyperbaric treatment facility is being arranged. CT or MRI of the brain should be deferred until after initial hyperbaric treatment unless intracranial hemorrhage, carotid artery dissection,[212] or other nondiving injury is strongly suspected. Diagnostic imaging has low sensitivity and should not delay initiation of hyperbaric oxygen therapy.

Hyperbaric Oxygen Therapy

Hyperbaric oxygen therapy (HBOT) consists of rapidly increasing ambient pressure to reduce intravascular bubble volume and restore tissue perfusion. Oxygen-enriched breathing mixtures enhance bubble resolution and deliver oxygen to hypoxic nervous tissue, followed by slow decompression to avoid bubble re-formation.[33,74,76] Additional details regarding HBOT tables used for treating AGE are provided in Chapter 72. Although patients with AGE have successfully been treated in a monoplace hyperbaric chamber,[290] it is better for these patients to be treated in multiplace hyperbaric chambers (Figure 71-10) capable of being pressurized to 6 ATA and in which both air and oxygen can be administered to the patient. There is growing evidence that HBOT to 2.8 ATA with 100% oxygen may be more effective treatment than initial pressurization to 6 ATA for divers with AGE because of the typical several-hour delay in bringing the patient to a chamber and, thus, less need for the higher pressure to resolubilize bubbles (and perhaps a greater immediate need for tissue oxygenation).[85,86,159,160,179,180]

FIGURE 71-10 Example of a large modern hyperbaric chamber. A hyperbaric chamber for treating decompression sickness or arterial gas emboli should preferably have a pressure capability of at least 6 ATA (165 fsw) and should have space for an attendant to provide ongoing hands-on care and repeated neurologic examinations. It must have provisions for supplying 100% oxygen and other gases for treating the stricken diver.

Although the majority of victims with AGE have neurologic deficits when examined, initial manifestations may spontaneously resolve by the time the victim is seen by medical personnel. Occasionally, a person has symptoms but no reproducible neurologic deficits on physical examination.[160,178] Nonetheless, all patients must be referred for diving medicine consultation and hyperbaric treatment if the history is suggestive of AGE, because neurologic impairment is impossible to exclude in the acute care setting, and waiting to complete definitive diagnostic studies may allow subtle neurologic injuries to become irreversible.[218] Additionally, patients with AGE who show clinical improvement or recovery may deteriorate a few hours later.[232] Therefore, early HBOT is recommended even for patients who have spontaneous recovery.

Adjunctive Treatment

Over the years, numerous medications (e.g., heparin, low-molecular-weight dextran, aspirin, corticosteroids) have been proposed as adjuncts to recompression and hyperbaric oxygen for treatment of AGE and DCS; however, experimental and clinical data do not support the use of any of these for AGE.

Lidocaine had been proposed as an adjuvant to HBOT for treatment of AGE. Limited experimental data support its use. Lidocaine is a class IB antiarrhythmic agent and a local anesthetic that crosses the blood-brain barrier and may have cerebroprotective effects by modulation of inflammatory mediators, preservation of cerebral blood flow, reduction in cerebral metabolism, and deceleration of ischemic ion fluxes. In animal models of AGE and brain ischemia, lidocaine acts to preserve blood flow in the brain, reduce brain edema and intracranial pressure, and preserve neuroelectrical function.[199,205] When given prophylactically, lidocaine reduces brain dysfunction after AGE in cats[108] and improves recovery of brain function in cats and dogs with AGE when given therapeutically.[98,107] Human data are less impressive because many of the case studies contain patients with both AGE and DCS.[61,90,291] Although earlier studies reported significantly less neurocognitive deficit in patients when lidocaine was used prophylactically during left heart valve surgery,[205] follow-up studies have not shown any neuroprotective effect of lidocaine during adult cardiac surgery.[192,204] Overall, the evidence does not support the use of lidocaine as an adjunct to recompression for treatment of AGE.

PREVENTION OF PULMONARY BAROTRAUMA AND ARTERIAL GAS EMBOLISM

In view of the potentially catastrophic consequences of AGE, one of the key goals of scuba diving training is to prevent pulmonary overpressure accidents. Divers must be warned of the potentially great intrathoracic volume changes that can occur at shallow depths and be trained to keep an open airway during ascent, particularly through the last 3 msw (10 fsw) to the surface. If equipment malfunction or depletion of air supply at depth makes this impossible, the diver must make every attempt to exhale continuously during an "emergency swimming ascent" in order to vent the increasing volume of air from within the lungs. With a satisfactory air supply, the diver should simply breathe normally on ascent to the surface, taking care to ascend slowly near the surface.

INDIRECT EFFECTS OF PRESSURE

Several diving-related problems may develop as a result of breathing gases at higher-than-normal atmospheric pressures. Chief among these are nitrogen narcosis, oxygen toxicity, and DCS.

DALTON'S LAW OF PARTIAL PRESSURES

Dalton's law of partial pressures states that the total pressure exerted by a mixture of gases is the sum of the pressures that would be exerted by each of the gases if it alone occupied the total volume. The partial pressure of a gas in a mixture is the pressure exerted by that gas alone. The symbols for partial

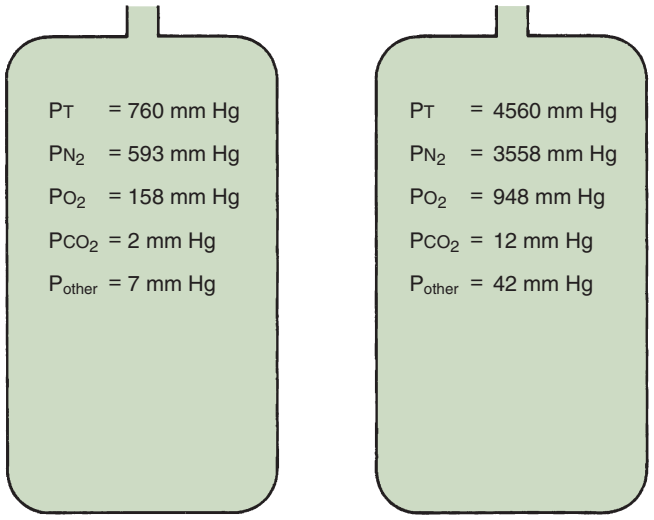

FIGURE 71-11 Dalton's law of partial pressures. The total pressure exerted by a mixture of gases is the sum of the pressures that would be expected of each of the gases if it alone were present and occupied the total volume.

pressure of oxygen, nitrogen, carbon dioxide, and water vapor are P_{O_2}, P_{N_2}, P_{CO_2}, and P_{H_2O}, respectively. Dalton's law states that in an air mixture, the total pressure (PT) = P_{N_2} + P_{O_2} + P_{H_2O} + P_{other}. The partial pressure of each gas in the mixture is found by multiplying the percentage of that gas present by the total pressure. In Figure 71-11, in a mixture of air, nitrogen is assumed to be present in a proportion of 78%, oxygen at 21%, and carbon dioxide at 0.03%, and the balance composed of water vapor and other trace gases.

The partial pressures of inspired gases in a gas mixture, not their percentages, are of prime importance in diving. For example, it has been shown that in hyperbaric chamber treatment of DCS, 100% oxygen can be safely used at depths to 2.8 ATA (60 fsw) for 20-minute periods with the subject at rest in the dry chamber. On the other hand, with 21% oxygen in a helium-oxygen mixture at 20 ATA (600 fsw), the diver would breathe 0.21 × 20 ATA, or 4.2 ATA of oxygen, which would rapidly produce CNS oxygen poisoning. This type of problem is avoided in deep diving by reducing the oxygen percentage in the gas mixture to between 0.35 and 0.50 ATA, so that the P_{O_2} will be between 266 and 380 mm Hg.

The scuba diver who uses open-circuit compressed air is subject to the effects of the component gases in the air according to their partial pressures. Thus, even though the gas mixture is simply air with normal percentages of oxygen and nitrogen, the increases in partial pressures of these gases and those of the trace contaminants at sea level create numerous potential problems related to the breathing medium. Most notable among these for scuba divers is the problem of nitrogen narcosis.

NITROGEN NARCOSIS

Nitrogen narcosis, also known as rapture of the deep or inert gas narcosis, is development of intoxication due to increased partial pressure of nitrogen in compressed air at increased depth. Nitrogen narcosis is important to divers because it causes anesthetic-like euphoria, overconfidence, and deterioration in judgment and cognition, all of which can lead to serious errors in diving techniques, accidents, and drowning. Many divers have died as a consequence of nitrogen narcosis.

During the 1930s, Behnke and associates[13,14] first suggested that mood changes described by divers breathing compressed air at 200 fsw were caused by a high inspired P_{N_2}. Much has been learned since that time, and in-depth analysis of inert gas narcosis has been provided by Bennett[16-18] and recently reviewed by Clark.[58]

Although the exact mechanism of inert gas narcosis is not known, the intoxicating effect of nitrogen is believed to be similar to the effects of gaseous anesthetics. According to currently accepted theory, alteration in electrical properties of cellular membranes is affected by absorption of gas molecules into their lipid component. The theory is supported by the observation that the greater the lipid solubility of a given gas, the greater is its narcotic potency. Thus, higher partial pressures of pure nitrogen are required to produce an anesthetic effect than is the case with nitrous oxide, the effects of which can be achieved by much lower partial pressures; nitrous oxide is much more soluble in lipid than is nitrogen. The lack of narcotic effect of helium is in accord with its low lipid solubility. Indeed, substitution of helium for nitrogen as the inert gas in the diver's breathing gas prevents nitrogen narcosis and is the main reason helium-oxygen mixtures are used for deep diving. In experimental rats, the activities of interneuronal $GABA_A$ receptors are desensitized during exposure to high partial pressures of nitrogen.[177] The actual mechanism of nitrogen narcosis is probably multifactorial.

Typically, a scuba diver breathing compressed air develops symptoms of nitrogen narcosis at depths between 21 and 31 msw (70 and 100 fsw). These symptoms include light-headedness, loss of fine sensory discrimination, giddiness, and euphoria. Symptoms progressively worsen at deeper depths. At depths over 46 msw (150 fsw), a diver becomes severely intoxicated, presenting with increasingly poor judgment and impaired reasoning, overconfidence, and slowed reflexes. At depths of 76 to 91 msw (250 to 300 fsw), auditory and visual hallucinations may occur, along with feelings of impending blackout. Most divers lose consciousness when they reach a depth of 122 msw (400 fsw).

Of note, individual and diurnal variability occur in the depth of onset and severity of symptoms in nitrogen narcosis. Oceanographic parameters, such as visibility and light penetration, can affect the onset or severity of narcosis. Also, some degree of acclimatization allows experienced divers to work more safely at greater depths than can inexperienced divers. Nonetheless, nitrogen narcosis is a major problem for all compressed-air divers at depths greater than 31 msw (100 fsw). This is one of the reasons why it is recommended that recreational divers not dive deeper than 39 msw (130 fsw).

Treatment of nitrogen narcosis simply requires ascent to a shallower depth (usually < 21 to 31 msw [70 to 100 fsw]), where symptoms promptly clear. Of course, the condition is prevented by avoiding deep dives. In commercial diving, where there may be good reasons to dive deeper than 31 msw (100 fsw), the problem is prevented by using heliox. In general, the effects of inert gas narcosis resolve by decreasing the partial pressure of the inert gas. However, there is evidence to indicate that some of the symptoms of nitrogen narcosis can be persistent. Measures of critical flicker fusion frequency (a measure of visual acuity) remained significantly altered 30 minutes after a single dive to 30 msw (97 fsw).[7] Symptoms were reversed by treatment with 100% oxygen, suggesting that some of the neurologic alterations caused by nitrogen narcosis may persist in a manner similar to delayed recovery from anesthetics.

OXYGEN TOXICITY

Although oxygen is essential for most life on Earth, it becomes a poison at elevated partial pressures. Oxygen toxicity in divers can affect either the CNS or pulmonary system. Understanding oxygen effects is necessary for safe diving operations. A complete discussion of the complex pathophysiology of oxygen toxicity is beyond the scope of this chapter. Thorough reviews of oxygen under pressure for diving and hyperbaric exposures are available.[38,57]

Inspired high P_{O_2} occurs in diving in two ways. Breathing 100% oxygen underwater or in a hyperbaric chamber is one. The second results from Dalton's law of partial pressures. If a normal 21% oxygen gas mixture is breathed at 10 ATA (300 fsw), an inspired P_{O_2} of 2.1 ATA is generated, which is equivalent to

breathing 100% oxygen at 36 fsw. Again, it is the partial pressure of a gas that determines its biologic effects.

Pulmonary Oxygen Toxicity

Retrolental fibroplasia in premature infants and pulmonary oxygen toxicity in adults are well-known problems associated with use of therapeutic oxygen. Pulmonary oxygen toxicity is induced by breathing above-normal, but relatively low, P_{O_2} for prolonged periods. The limit for indefinite exposure without demonstrable lung damage is generally considered to be a P_{O_2} of approximately 0.5 ATA. On a time-dose curve, it is generally considered safe to breathe 100% oxygen at 1 ATA for up to about 20 hours, or at 2 ATA for up to 6 hours. This time can be lengthened significantly by using intermittent exposures, such as interspersing a 5-minute air break between every 20 minutes of oxygen breathing.[138] At 2.8 to 3 ATA (60 to 66 fsw), at which depth 100% oxygen is used to treat DCS and gas gangrene, pulmonary oxygen toxicity is rarely a problem, because CNS manifestations usually intervene before sufficient time elapses to induce pulmonary damage. Pulmonary oxygen toxicity and its relationship to professional diving have been reviewed.[284] Using hyperbaric oxygen according to U.S. Navy Treatment Table 6 (used to treat DCS; see Chapter 72, Figure 72-11) does not produce clinical manifestations of pulmonary oxygen toxicity. However, animal studies involving more extreme oxygen exposures have shown a pathologic sequence of alveolar capillary endothelial damage, with increased permeability leading to pulmonary edema and hemorrhage. Variations in susceptibility are seen among species, but interruption of exposure usually results in reversal of pathologic changes.[59]

The most common clinical manifestation of pulmonary oxygen toxicity is substernal discomfort on inhalation. If exposure continues, this can progress to severe burning substernal pain and persistent cough. Reduction of inspired PO_2 to 0.21 to 0.5 ATA usually results in prompt relief. Severe cases of pulmonary oxygen toxicity may require endotracheal intubation and positive end-expiratory pressure ventilation to achieve adequate arterial oxygenation at the required lower partial pressures of inspired oxygen.

Central Nervous System Oxygen Toxicity

During the 1880s, the French physiologist Paul Bert described convulsions in animals breathing 100% oxygen at elevated chamber pressures. Behnke and others observed the same phenomenon in humans in the 1930s. Donald's classic observations on divers were published in 1947 and provide much of the current knowledge on predisposing factors and clinical manifestations of CNS oxygen toxicity.[87,88]

In brief, Donald found that at a given duration and pressure of oxygen, a diver in the water was more susceptible to oxygen-induced seizures than was the same diver in a warm, dry hyperbaric chamber. Based on this observation, a limit of 7.6 msw (25 fsw) was imposed for military special operations dives using 100% oxygen. Donald found a wide variation in oxygen tolerance among subjects exposed to the same conditions, as well as variability in oxygen tolerance in the same diver from day to day. Most people can tolerate breathing 100% oxygen for 30 minutes at rest at 2.8 ATA in a dry chamber, although some present with toxicity at this exposure.

Common symptoms and signs of CNS poisoning are shown in Box 71-4. Unfortunately, some people may have no warning symptoms, and the first manifestation of CNS oxygen toxicity may be a generalized seizure. If an oxygen-induced seizure occurs underwater, there is a high risk for drowning and death. With increased use of RBs and mixed-gas technical diving, oxygen toxicity is a serious consideration. Apart from physical injury or drowning, an oxygen seizure does not produce harmful or residual effects. CNS oxygen toxicity due to HBOT is discussed in Chapter 72.

BOX 71-4 Typical Manifestations of Central Nervous System Oxygen Poisoning

Apprehension
Feeling of air hunger
Sweating
Nausea
Focal muscle twitching
Isolated jerking of a limb
Auditory changes (such as "hearing bells ringing")
Tunnel vision
Diaphragmatic flutter
Convulsion

CONTAMINATED BREATHING GAS (CARBON MONOXIDE POISONING, HYPERCARBIA)

As breathing-gas cylinders are pressurized or filled, the sea level partial pressure of each gaseous component is multiplied. Therefore, any contaminant in the air source can potentially become dangerous to the diver at the elevated pressure found underwater. Compressor motors must be free of oil that could be pumped into tanks; otherwise, oil mist in the air may cause the diver to suffer lipoid pneumonitis.[161]

Compressed-air inlets should always be situated so that they avoid engine exhaust from the compressor, parking lots, or other combustion sources that produce carbon monoxide. Because carbon monoxide is colorless, odorless, and tasteless, the diver cannot detect it unless it is accompanied by other contaminants. The first warning of carbon monoxide poisoning may be headache, nausea, or dizziness during the dive. Examination at the surface may show lethargy, mental dullness, and nonspecific neurologic deficits, which may be confused with those accompanying DCS or air embolism. The cherry red skin color often mentioned in standard medical texts is rarely observed in carbon monoxide poisoning. Holt and Weaver[143a] reported a case of carbon monoxide poisoning in a commercial diver who was initially diagnosed and treated for presumed AGE. Fortunately, the treatment of choice for serious acute carbon monoxide poisoning, DCS, and air embolism is HBOT.

Another potential breathing gas problem involves carbon dioxide. Alveolar partial pressure of carbon dioxide (P_{aCO_2}) reflects the arterial P_{CO_2}; therefore, even as ambient pressure increases at depth, the P_{aCO_2} remains constant at approximately 40 mm Hg unless environmental or physiologic changes occur. Hypercapnia can occur because of increased P_{CO_2} in the breathing gas or decreased pulmonary ventilation. Unless there is regulator malfunction or contaminated breathing gas, hypercapnia is exceptionally rare in open-circuit scuba diving. Hypercapnia can occur in helmet or chamber diving if these closed spaces are inadequately ventilated or the breathing gas becomes contaminated by carbon dioxide, and in closed-circuit scuba diving (i.e., using an RB) if there is failure of the carbon dioxide–absorbent (scrubbing) material. It is believed that hypercarbia was the primary cause of David Shaw's death at Boesmansgat cave in South Africa in January 2005.[201] Shaw was attempting to recover the remains of 20-year-old Deon Dreyer, who had drowned in the cave on Mount Carmel farm more than 10 years earlier, from a depth of 276 m (905 feet).

At sea level, a concentration of 5% to 6% inspired carbon dioxide leads to dyspnea, increased respiratory rate, and mental confusion. At 10% inspired carbon dioxide, pulse rate and blood pressure may fall to the point that unconsciousness occurs. With prolonged exposure to 12% to 14% inspired carbon dioxide, such that the P_{aCO_2} exceeds 150 mm Hg, central respiratory and cardiac depression can be fatal.

HYPERVENTILATION AND SHALLOW WATER BLACKOUT

Hypocapnia can result from hyperventilation during or before diving. The well-known symptoms of hyperventilation, dizziness, and paresthesias around the mouth and in the distal extremities have been postulated to cause unconsciousness among divers, but whether this actually occurs is unclear. In contrast, unconsciousness associated with hyperventilation before a breath-hold dive is caused by hypoxia, rather than hypocapnia.

BOX 71-5 Causes of Unconsciousness in Divers

Breath-Hold Divers
Underwater hypoxemia after hyperventilation before the dive (shallow water blackout)
Near drowning

Divers Using Compressed-Gas Equipment
Hypoxic breathing gas
Contaminated breathing gas (such as carbon monoxide)
Equipment failure or exhaustion of breathing gas
Near drowning
Inert gas narcosis
Oxygen toxicity
Pulmonary barotrauma with arterial gas embolism

Divers Using Rebreathing Equipment
Carbon dioxide toxicity
Oxygen toxicity
Hypoxia

In what is commonly described as *shallow water blackout,* a diver hyperventilates before a dive, lowering alveolar P_{CO_2} to 20 to 30 mm Hg. However, because hemoglobin is nearly saturated with oxygen during normal respiration, there is little gain in arterial P_{O_2} by hyperventilating. In an underwater swim, even in a shallow swimming pool, exercise-induced hypoxia sufficient to cause unconsciousness may occur before arterial P_{CO_2} reaccumulates to provide sufficient stimulus to breathe.[12]

In a deep breath-hold dive, the problem is compounded by the effect of increased pressure. In addition to the initial depression of arterial P_{CO_2} secondary to hyperventilation, elevations occur in alveolar and arterial P_{O_2}. During the dive, these serve to suppress the respiratory response to hypercarbia. During descent in the water, $PaCO_2$ increases from the increased pressure, but after oxygen consumption at depth, depressurization on return to the surface causes a dramatic drop in alveolar and arterial P_{CO_2}. Even if $PaCO_2$ rises to the stimulatory breakpoint during ascent, hypoxemia may cause unconsciousness and near drowning. An expansion of Edmonds' most common causes of unconsciousness in divers is presented in Box 71-5.[101] Breath-hold diving fatalities average 3 per 10,000 divers annually.[191]

DECOMPRESSION SICKNESS

In the mid19th century, tunnel and bridge workers who labored in caissons pressurized with compressed air were sometimes observed to suffer joint pains, paralysis, and other medical problems after leaving the high-pressure caissons. The condition was not understood and became dubbed caisson disease or compressed-air illness.[236] Of course, these early high-pressure workers were experiencing the same symptoms that were later observed in divers and aviators, and that we now know to be decompression sickness (DCS).

For many decades, caisson disease remained a medical curiosity, but because of its occurrence in increasingly important areas of activity in the 20th century, considerable research has been directed to better understanding its causes and finding effective treatment.

DCS is caused by formation of bubbles of inert gas (e.g., nitrogen) within both intravascular and extravascular spaces after reductions in ambient pressure. This may occur in divers during or after decompression from being underwater; in persons in a caisson or hyperbaric chamber in which pressures are greater than at sea level; or in aviators, astronauts, or hypobaric (high-altitude) chamber workers who travel rapidly from sea level to pressures less than 0.5 ATA. DCS is also a major concern in commercial divers who breathe heliox and then switch breathing gases to nitrox; bubbles can form in the absence of a decrease in ambient pressure because of local tissue supersaturation due to a change in breathing gas.

CAUSE

Although DCS is a topic of considerable research and discussion among divers and diving medicine practitioners, it is fortunately rare. Based on DAN's Project Dive Exploration data, a DCS incidence summary based on 137,451 dive profiles collected from 1995 to 2008 resulted in a diagnosis of 41 cases.[95] Profiles were collected from various diving environments. Deep-wreck divers in the North Atlantic had a DCS incidence of 0.181%, whereas the incidence among warm-water live-aboard divers was 0.006%.[96]

To understand the cause of DCS, one must appreciate the temporal relationship between inert gas uptake and elimination. Earth's atmosphere is composed of 78% nitrogen and 21% oxygen; it is the presence of this inert nitrogen that forms the crux of the DCS problem. If it were safe for a diver to breathe 100% oxygen while underwater, one could prevent DCS because oxygen is rapidly metabolized by the body, and for all practical purposes does not contribute to bubble formation on ascent. Unfortunately, breathing pure oxygen at increased atmospheric pressure causes CNS toxicity. To prevent this, divers breathe an inert gas diluent (e.g., nitrogen) and thus must be concerned with inert gas (nitrogen) absorption and potential bubble formation during decompression.

The partial pressures of inspired gases increase as a diver descends. At 4 ATA (99 fsw), the absolute pressure is 3040 mm Hg (see Table 71-2), 79% of which is nitrogen (2400 mm Hg, as compared with 600 mm Hg P_{N_2} at sea level). Accounting for water vapor and carbon dioxide, the resulting alveolar partial pressure of nitrogen (P_{AN_2}) at this depth is approximately 2360 mm Hg. This P_{AN_2} is rapidly reflected across the alveolar-capillary membrane to the arterial blood, where (according to Henry's law) nitrogen becomes physically dissolved in the blood. Henry's law states that the amount of gas dissolved in a liquid at any given temperature is a function of the partial pressure of the gas in contact with the liquid. Thus, the amount of nitrogen absorbed by tissues during a dive is a function of depth (pressure) and time.

PATHOPHYSIOLOGY

DCS is a multisystem disorder caused by the separation of gas bubbles in the body's tissues as a result of inadequate decompression time, leading to an excessive degree of gas formation. Rapid decompression, due to either a rapid decrease in ambient atmospheric pressure from rapid ascent or omission of decompression stops, causes inert gas (nitrogen) to come out of solution and form bubbles in tissue and venous blood. Conceptually, DCS is the same illness whether it occurs in high-altitude aviators or deep-sea divers, although there are some differences in the symptoms of the disease, depending on whether it is caused by hyperbaric or hypobaric exposure.

Paul Bert first described bubbles in the bloodstream in experimental animals after decompression in 1878.[21] Bubbles form within tissues in which the inert gas partial pressure exceeds the pressure within the tissue. It is believed that bubble formation is initiated at sites of stable gas micronuclei.[282] The physiologic sequelae of bubble formation in tissue and venous blood are myriad. These effects include cellular distention and rupture; mechanical stretching of tendons or ligaments, producing pain; and intravascular or intralymphatic occlusion, resulting in congestive ischemia and infarction, or lymphedema. Boycott and colleagues[35] described a method for prevention of compressed-air illness in divers when a pressure reduction ratio not exceeding 2:1 is observed.

Venous Gas Emboli

The presence of bubbles in tissues or venous circulation does not imply DCS. Venous gas emboli (VGEs) probably originate in extravascular tissue and enter the bloodstream, where they enlarge. These bubbles travel to the right heart and are trapped by the pulmonary capillaries when the gas diffuses into alveoli. VGEs can be observed in the veins or right heart after diving using ultrasound or echocardiography.[94,266] VGEs can be found in varying quantities in some divers with no symptoms of DCS. There is no direct correlation of bubbles with DCS; however, their presence is thought by some researchers to be a marker of decompression stress.[118,183] Recent data suggest that there are some individuals who are more prone to developing VGEs after diving, leading to the concept of "bubble-prone" and

TABLE 71-5 Common Symptoms and Signs of Decompression Sickness

Condition	Symptoms	Signs
Musculoskeletal decompression sickness, limb bends	Severe joint pain, single joint or multiple joints involved, paresthesia or dysesthesia around the joint, lymphedema (uncommon)	Tenderness, which may be temporarily relieved by local pressure with a blood pressure cuff; pain worsened by movement of the joint
Neurologic decompression sickness		
Spinal cord	Back pain, girdling abdominal pain, extremity heaviness or weakness, paralysis, paresthesia of extremities, fecal incontinence, urine retention	Hyperesthesia or hypoesthesia, paresis, anal sphincter weakness, loss of bulbocavernosus reflex, urinary bladder distention
Brain	Visual loss, scotomata, headache, dysphasia, confusion	Visual field deficit, spotty motor or sensory deficits, disorientation or mental dullness
Fatigue	Profound generalized heaviness or fatigue	May precede signs of other forms
Cutaneous manifestations	Intense pruritus	No visible signs, mottling, local or generalized hyperemia or marbled skin (cutis marmorata)
Chokes	Dyspnea, substernal pain that is worsened on deep inhalation, nonproductive cough	Cyanosis, tachypnea, tachycardia
Vasomotor decompression sickness (decompression shock)	Weakness, sweating, unconsciousness	Hypotension, tachycardia, pallor, mottling, hemoconcentration, decreased urine output
Inner ear (vestibular) decompression sickness	Tinnitus, vertigo, nausea, vomiting	Ataxia, possible nystagmus and positive Romberg's test, acute sensorineural hearing loss

"bubble-resistant" individuals.[55,56,118] Because VGEs occur much more frequently than does DCS and are easily detected even in safe dives, Doppler bubble detection is used to develop models of DCS, for evaluating safe diving profiles, and for diving medicine research.

In some circumstances, VGEs can enter the arterial circulation through a right-to-left shunt, such as a patent foramen ovale (PFO), atrial septal defect, or intrapulmonary arteriovenous anastomoses,[187] or by overwhelming the pulmonary capillary filtration system. Arterialized VGEs can produce symptoms of AGE and may play a role in certain types of DCS, particularly involving the skin, inner ear, and CNS.[202,208,277,297] Further research on PFO and diving is discussed below.

Biochemical Effects of Bubbles

Intravascular bubbles cause multiple biophysical effects at the blood-bubble surface interface because bubbles are viewed by the immune system as foreign matter and incite an inflammatory reaction. One step in the process is activation of the Hageman factor, which in turn activates intrinsic clotting, kinin, and complement systems, producing platelet activation, cellular clumping, lipid embolization, increased vascular permeability, interstitial edema, and microvascular sludging. The overall effects are decreased tissue perfusion and ischemia.

The pathophysiologic changes associated with scuba diving and DCS have been extensively studied but remain poorly understood. More recent attention focuses on the roles played by cellular and biochemical changes involved in inflammation, oxidative stress, and vascular endothelial cell activation. Scuba diving without DCS results in physiologic consequences that include increased oxidative stress, impaired endothelial function, platelet activation, neutrophil activation, and increase in microparticles.[222,239,267,271,272] How these factors contribute to DCS is unclear. Gas bubbles are associated with platelet aggregation.[6] Complement activation by bubbles has been reported in vitro and in vivo in animals and proposed as a mechanism for DCS in humans.[142,287,288] Bubbles cause endothelial disruption, leading to extravasation of plasma into the interstitium, causing an increase in hematocrit level.[31,221] Leukocytes adhere to bubbles and denuded endothelium. Microparticle increase and production may play a role in neutrophil activation, resulting in vascular leak and DCS in mice and humans.[270,273,303] Nitric oxide is now thought to play a role in bubble formation.[300] Endothelial nitric oxide, an important vasodilator with antiatherogenic properties, can attenuate bubble formation and DCS incidence, most likely by reducing gaseous nuclei from which bubbles form. Activation of heat shock proteins may lead to protection from bubble-induced injury during decompression.[24,113,194]

Clinical Manifestations

The clinical manifestations of DCS are protean (Table 71-5), with neurologic and musculoskeletal systems most often affected. Symptoms and signs of DCS include joint pain, cutaneous rashes, neurologic dysfunction, pulmonary edema (chokes), and shock; death may follow. DCS is often categorized as type I and type II, with type I referring to the mild forms of DCS (cutaneous, lymphatic, and musculoskeletal) and type II including the neurologic and other serious forms (pulmonary chokes and inner ear DCS). Some investigators have advocated using type III DCS to refer to combined AGE and DCS with neurologic symptoms.[216] However, this term should not be used; *biphasic decompression illness* accurately describes the presentation of AGE and DCS occurring together.

Although categorization of DCS as types I and II is firmly entrenched in the literature, its use is not advocated. It is clinically more meaningful to refer to the body systems affected when discussing patients with DCS, especially in light of the growing awareness that all cases of DCS must be considered serious and treated vigorously.

Musculoskeletal Decompression Sickness

Musculoskeletal DCS, or *the bends,* is development of pain in and around major joints after diving or other hyperbaric or hypobaric exposures. It is the most common manifestation of DCS, occurring in approximately 70% of patients.[193,236,244] This form of DCS is often referred to as limb bends, joint bends, or pain-only bends.

The term *bends* originated at the beginning of the 20th century, when caisson workers on the Brooklyn Bridge who were suffering from DCS of the hips were noted to walk stiffly, bending forward at the hips.[236] Coworkers would describe the stricken men as walking as if they were trying to do the *Grecian bend,* a term for the forward-bending, stiff-at-the-hips way that stylish women of the day would walk because of their tight corsets. Over time, the term became shortened to just bends.

The shoulders and elbows are the joints most often affected by DCS in scuba divers, but any joint may be involved. Hips and knees are most commonly affected in saturation divers, caisson workers, and aviators. The reason for the different anatomic predilection is not known.

The pain of joint bends is usually described as "boring" or a dull ache deep within the joint, although it may also be characterized as sharp or throbbing. It is sometimes described as "tearing" or feeling like tendinitis or bursitis. It may radiate to surrounding areas. Movement of the joint can worsen the pain,

so the joint is usually held immobile. An area of vague numbness or dysesthesia may surround the affected joint, but this typically does not conform to any anatomic distribution and should not be confused with neurologic involvement. There also may be erythema around the joint, and the joint may be mildly swollen. The joint may also be tender to touch.

Sometimes divers will complain of having a "niggle" after a dive. *Niggle* is a British colloquialism referring to mild pain in a single joint that improves 10 to 15 minutes after the onset of pain and then disappears without treatment. Whether niggles should be treated with recompression is controversial, but the safest approach is to treat them with at least the protocol in U.S. Navy Treatment Table 5.

Differentiating between limb bends and trauma or other causes of joint pain may be aided by inflation of a sphygmomanometer cuff placed around the joint to 150 to 250 mm Hg.[247] If the pain is due to DCS, inflation of the cuff may immediately relieve the pain by reducing the gas volume in tendons and ligaments. This relief suggests that the mechanism of pain is gas expansion (bubbles) in tendons and ligaments, which stretches nerve endings. The pain of limb bends recurs when the cuff is deflated. The test is helpful when it is positive, but lack of a response cannot be used to rule out the presence of DCS; this must be done with a "test of pressure" in a hyperbaric chamber. Importantly, most often there may be neither abnormal physical signs (other than splinting or stiffness from pain) nor abnormal radiographic findings.

Limb bends pain alone is not immediately threatening to life or function, but indicates that bubbling may be occurring in venous blood. Often, patients who begin with musculoskeletal DCS and are not treated progress to more serious forms of DCS. In addition, it is impossible to completely exclude subtle neurologic signs in a field setting. Likewise, as discussed later, untreated DCS may lead to osteonecrosis of major joints.

Fatigue

Profound fatigue that is out of proportion to the activity performed underwater, or otherwise while under increased atmospheric pressure, may be an early manifestation of DCS. Although its cause is unknown, a feeling of severe fatigue after diving demands careful evaluation for other manifestations of DCS.

Cutaneous Decompression Sickness (Skin Bends)

DCS may present with a variety of cutaneous manifestations, including scarlatiniform, erysipeloid, or mottled rashes; pruritus; and formication. Occasionally, localized swelling or peau d'orange may result from lymphatic obstruction, and rarely, an entire limb may become edematous.

Skin manifestations are relatively uncommon and, in and of themselves, are usually not serious. However, mottling or marbling of the skin *(cutis marmorata)* is often a harbinger of more severe DCS (Figure 71-12). The exact physiologic basis of the mottled skin lesion is unknown. Skin bends should be easily distinguished from cutaneous barotrauma, "wetsuit dermatitis,"[170,250] marine envenomation, or other skin rashes often seen in divers.

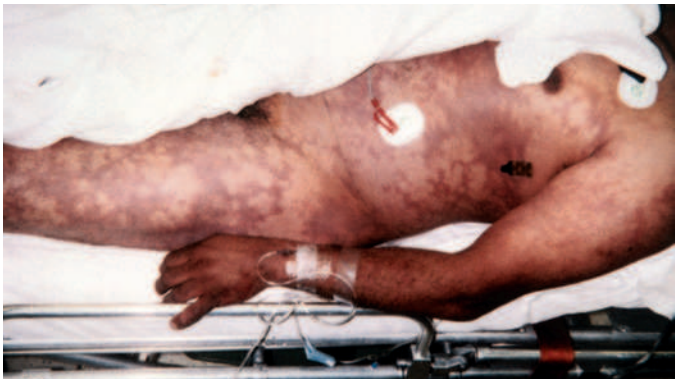

FIGURE 71-12 Cutis marmorata is mottling of the skin seen in severe cases of decompression sickness.

Itches, or "the creeps," is a type of skin bends seen during decompression in hyperbaric chamber workers when the skin is exposed to the high PN_2 in compressed air. This is a highly pruritic skin reaction most intensely felt on body parts exposed to the compressed air. The sensation is often described as feeling like ants crawling over one's body.

In hyperbaric chambers, inert gas from the external environment is absorbed directly into skin, and itches represent bubble formation in the skin during decompression. The concentrations of dissolved gases in ocean water are essentially constant at all depths, so the skin is not exposed to elevated partial pressures of inert gases underwater.

Pulmonary Decompression Sickness (Chokes)

The chokes is an unusual but very serious form of DCS characterized by burning substernal pain (especially on inhalation), cyanosis, dyspnea, and nonproductive cough. Animal studies have demonstrated gas bubbles or foam in the pulmonary arteries, right atrium, and right ventricle after unsafe decompression. The chokes probably represents massive pulmonary gas embolism with mechanical obstruction of the pulmonary vascular bed by bubbles. Typically, symptoms of pulmonary venous air embolization begin when 10% or more of the pulmonary vascular bed is obstructed. Patients with the chokes can progress rapidly to profound shock or neurologic DCS. The specific clinical and radiographic manifestations of the chokes are similar to those seen with VGEs from other causes.[162]

Symptoms of VGEs include air hunger, dyspnea, cough, and chest pain. Findings may include pallor, diaphoresis, tachypnea, tachycardia, hypotension, cyanosis, expiratory wheezing, neurologic signs, and a mill-wheel heart murmur. Victims may also exhibit increased central venous or pulmonary artery pressure, electrocardiographic changes of ischemia or cor pulmonale, decreased end-tidal carbon dioxide fraction, and precordial Doppler sounds of circulating gas bubbles. Echocardiography may show gas bubbles in the right atrium and ventricle. Rarely, air may be visualized in the main pulmonary artery on chest radiographs; this is pathognomonic for pulmonary air embolism.[146,162,219]

Neurologic Decompression Sickness

Neurologic impairment may occur as the sole manifestation of DCS or as part of a larger dysbaric syndrome. Neurologic DCS is manifested by myriad symptoms and signs because of the random nature by which DCS affects the nervous system. Although any level of the CNS may be affected, the most commonly involved site in divers is the spinal cord, specifically the lower thoracic and lumbar regions. Although much less frequently affected than the CNS, the peripheral nervous system also may be involved.[150]

Based on military experience, neurologic DCS was believed to occur in only 10% to 20% of DCS cases,[244] but neurologic manifestations of DCS have been found in 50% to 60% of scuba diving casualties treated in Hawaii[105,153] and have been reported in similarly high frequencies in other populations of sport divers.[74,84]

Classically, dysbaric spinal cord injury occurs in the lower thoracic, lumbar, and sacral portions of the cord, producing low back pain, subjective "heaviness" in the legs, paraplegia or paraparesis, lower extremity paresthesia or dysesthesia, and sometimes bladder or anal sphincter dysfunction. General malaise or fatigue is often noted as well. Involvement of the cervical and thoracic cord may cause chest or abdominal pain and weakness or sensory disturbances in the upper extremities. Absence of the bulbocavernosus reflex, elicited by gently squeezing and pulling the glans penis to seek reflex contraction of the anal sphincter, often foretells a poor prognosis, as does absence of the superficial anal reflex, which can be elicited in the male or female by stroking the perianal region.

The mechanism of spinal cord DCS is multifactorial, involving autochthonous inert gas bubble formation in the cord[116,225] and in the epidural vertebral venous plexus (Batson's plexus), with

resulting congestive infarction of the spinal cord.[132] There are other mechanisms that are not well understood.[140,225]

DCS of the brain produces a variety of symptoms, most of which are indistinguishable from AGE. These include dizziness, vertigo, altered mentation or level of consciousness, generalized weakness, and visual deficits (e.g., diplopia, scotoma, visual field defects, and blindness). Involvement of the cerebellum or inner ear may produce ataxia and loss of balance.

Inner Ear or Vestibular Decompression Sickness

The *staggers* is another classic DCS syndrome. In this case, the inner ear is primarily affected; the name derives from the unsteady gait that results from vestibular damage. Manifestations include dizziness, vertigo, nystagmus, tinnitus, nausea, and vomiting. In contrast to inner ear barotrauma, which generally has a favorable prognosis, inner ear DCS carries a high risk for residual inner ear damage despite appropriate treatment.

Inner ear DCS has been most often seen in saturation divers, when there is a rapid ascent on heliox, or when switching gases on ascent during very deep dives. However, there are increasing reports of inner ear DCS in technical and sport divers. A PFO has been associated with inner ear DCS, suggesting that shunted VGEs may play a role in inner ear injury.[202,277]

Vasomotor Decompression Sickness

Vasomotor DCS, or *decompression shock,* is a rare, life-threatening form of DCS. The pathogenesis of this shock syndrome is not completely understood. It is believed to be caused by a rapid shift of fluid from intravascular to extravascular spaces secondary to diffuse bubble embolization, ischemia, and hypoxia.[54] Hypotension may also result from massive venous air embolization of the lungs.

Despite vigorous intravenous fluid replacement, the hypotension of decompression shock may not respond until recompression is undertaken. Unfortunately, the condition is highly lethal, and most patients do not survive long enough to undergo recompression unless a hyperbaric chamber is immediately available.

LONG-TERM SEQUELAE OF DECOMPRESSION SICKNESS

Although DCS is the most overt manifestation of inadequate decompression after diving, it has now been clearly established that there may be long-term sequelae of diving related to inadequate decompression, even if the diver never manifests overt DCS. The most described problem is dysbaric osteonecrosis.

Dysbaric Osteonecrosis

Dysbaric osteonecrosis is a form of avascular or aseptic necrosis of bone associated with pressure changes. The major joints (shoulders, elbows, hips, and knees) are most often affected, although any bone can be involved.[53,104,195]

Dysbaric osteonecrosis was first recognized in compressed-air workers in the early 1900s.[224] Since then, its incidence in professional divers has been found to range from less than 1% to more than 80%, depending on the age of the diver and type of diving. Its occurrence correlates well with deep diving, decompression diving, occurrence of DCS, and missed decompression.[77,104,147,195,286] Most diving medicine experts consider dysbaric osteonecrosis a long-term sequela of inadequate decompression.

Fossil evidence of avascular necrosis has been found in marine mosasaurs and plesiosaurs of the Cretaceous period, suggesting that at least some of these extinct giant marine lizards dived deeply.[246] Likewise, although modern marine mammals were previously thought to be immune to DCS, it has been recently found that sperm whales, which dive to depths exceeding 3048 msw (10,000 fsw) and stay underwater for as long as an hour, also suffer from dysbaric osteonecrosis.

Dysbaric Retinopathy

Infrequently, DCS affects the eyes, producing a wide array of acute ophthalmic effects, including homonymous hemianopsia, cortical blindness, central retinal artery occlusion, retinal hemorrhage, nystagmus, convergence insufficiency, and optic neuropathy.[41,42] Long-term ophthalmic findings in divers have been observed.

A retinal fluorescein angiography survey of asymptomatic divers found a higher incidence of retinal pigment epithelium than in nondivers, and various capillary changes at the fovea.[237] The significance of these abnormalities is unclear because none of the divers had visual loss. Similarly, the cause of such changes is unclear, although they are postulated to be the result of small bubble microembolization.

DIAGNOSIS

As with AGE, diagnosis of DCS is clinical and based on history of exposure to increased atmospheric pressure and subsequent development of characteristic symptoms and signs. The majority of patients with DCS become symptomatic in the first hour after surfacing from a dive, with most of the remainder noticing symptoms within 3 hours after diving. The majority of all symptoms manifest within 24 hours after diving, unless they appear as a result of further decompression, such as high-altitude exposure.

A variety of laboratory abnormalities may be demonstrated in DCS, but most have little or no usefulness in the immediate management of patients. However, two tests that may be useful are urine specific gravity and hematocrit; intravascular volume depletion and hemoconcentration are common in serious DCS because of increased vascular permeability caused by endothelial damage and release of kinins. The results of these tests can help guide replacement fluid therapy. The hematocrit percentage is commonly in the high 50s or 60s in serious DCS. Low serum albumin is reported in cases of neurologic DCS due to increased vascular permeability; however, sensitivity of hypoalbuminemia as a predictor of DCS is very low.[120]

As with laboratory tests, radiographic evaluation of patients with suspected DCS may yield various findings, but the radiographs are rarely useful in acute management of the patient. Bone radiographs of patients with acute joint bends do not show abnormalities. Months to years later, they may demonstrate findings of dysbaric osteonecrosis. Noncardiogenic pulmonary edema may be seen on chest radiographs of persons with pulmonary or vasomotor DCS.

Both CT and MRI have been used to evaluate neurologic DCS injury, although conventional CT has poor sensitivity for early lesions and is unable to image spinal cord lesions.[143,155] Limited clinical data support the feasibility and efficacy of MRI in these conditions,[119,181,265,289] especially when intracranial injury is suspected, although the urgency of obtaining HBOT makes these modalities useful primarily for postrecompression evaluation of residual deficits. Neither imaging nor laboratory data should be used to confirm the diagnosis of DCS or to decide if a diver with suspected DCS needs HBOT.

TREATMENT

All persons with suspected DCS should be referred to a hyperbaric treatment facility as quickly as possible because HBOT is the primary and essential treatment for this condition. The physician must have a high index of suspicion when diagnosing DCS, because the often-diverse manifestations of DCS may present a very confusing clinical picture. The history of the dive profile is helpful if the diver knowingly violated decompression procedures, but DCS may occur on dives that should be safe according to current decompression schedules.[1] In addition, the reported depth and time of the dive are often not accurate.

Management of DCS must begin as soon as the condition is suspected.[74] Divers may be far from a hyperbaric chamber when their symptoms develop, so treatment is often initiated in the field or at a general acute care facility. Supplemental oxygen at a high flow rate (10 L/min by nonrebreather face mask) should be administered as soon as possible, beginning on the dive boat or otherwise in the field and continued during transport, emergency department evaluation, and transfer to a hyperbaric

FIGURE 71-13 Logo for the Divers Alert Network. *(Courtesy Divers Alert Network.)*

treatment facility. Supplemental oxygen enhances the rate of resolution of inert gas bubbles and treats hypoxemia. Of equal importance is maintenance of intravascular volume to ensure capillary perfusion for elimination of microvascular inert gas bubbles and tissue oxygenation. Intravenous infusion of isotonic solution should be started and run at a flow rate sufficient to maintain urine output at 1 to 2 mL/kg/hr. If there is spinal cord involvement, an indwelling urinary catheter may be needed because of sacral nerve root dysfunction and urinary retention. Intractable vomiting or vertigo should be treated with appropriate parenteral agents. Diazepam has been quite effective in providing relief from the vertigo associated with inner ear DCS. Advanced life support measures should be undertaken appropriate to the patient's clinical condition.

Arrangements must be made for transfer to the nearest hyperbaric chamber. Because of the large number and frequently changing status of hyperbaric chambers, no list is provided here. To locate an active chamber, the reader is referred to the Divers Alert Network (DAN) (Figure 71-13).

Before a patient is transferred to the hyperbaric treatment facility, it is imperative to contact the chamber to determine its availability. The chamber may be out of service or already being used to treat another patient. The physician should never send a patient without first discussing the transfer with hyperbaric treatment personnel.

If airborne evacuation is required, it is critical to obtain an aircraft that can maintain sea level cabin pressurization during flight. Examples of such aircraft are the military C9 and C-130 Hercules, Learjet, and Cessna Citation. In the case of helicopters (which cannot be pressurized), the crew must maintain the lowest possible flight altitude, preferably never greater than 305 m (1000 feet) above the starting elevation. This is always problematic in evacuations from mountain lakes. All resuscitative measures must be maintained in flight.

At the hyperbaric treatment facility, one of several standard hyperbaric treatment protocols is followed. In a multilock compressed-air chamber, the patient and an attendant can be pressurized with compressed air, and the patient given 100% oxygen by face mask.

As with AGE, hyperbaric treatment of DCS has undergone significant evolution over the past two decades and is discussed in more detail in Chapter 72.

HBOT is most often successful, but the likelihood of success is difficult to predict for any given diver. In general, the sooner after the onset of symptoms that treatment begins, the better the outcome,[9] although treatment after delays of many hours, or even days, often results in full functional recovery. In one series of 92 sport scuba divers treated after a significant delay between the offending dive and start of hyperbaric treatment, 85% had good results when standard U.S. Navy treatment tables were followed.[74] Similar results were achieved in another series of 50 patients.[158] Even despite a delay of 48 hours or longer for HBOT, 76% of divers with DCS had full recovery.[131] Such treatment is usually given according to U.S. Navy Tables 5 and 6 (Figure 71-14). Any patient with neurologic or pulmonary DCS requires treatment with U.S. Navy Table 6 (see Figure 71-14B), with extension of the hyperbaric oxygen periods, depending on how the patient responds.

Monoplace hyperbaric chambers are used to treat DCS and air embolism,[136,159] although the conventional wisdom is to use a multiplace chamber whenever possible because of the free access to the patient that is possible in these larger chambers and greater flexibility in possible treatment regimens.

U.S. Navy Treatment Table 7 is an option for serious DCS cases; however, this treatment table should be reserved for patients with major deficits because of its length and commitment of resources. The diver and attendant are held at 2.8 ATA for at least 12 hours, and longer if needed, with the patient breathing oxygen in 30-minute periods as tolerated. A final, slow, 32-hour decompression follows, regardless of the time spent at 2.8 ATA. Details of Table 7 are found in the *U.S. Navy Diving Manual*.[278]

Patients with DCS who have residual symptoms after their initial HBOT should receive repetitive treatments until symptoms have completely resolved or the patient shows no further improvement in response to two consecutive treatments. Most individuals need no more than 5 to 10 treatments. Treatments can be given daily or twice daily as shown in U.S. Navy Table 5, 6, or 9 (2-hour treatment at 2.4 ATA). A small number of patients have residual neurologic deficits that may either be permanent or improve gradually over 6 to 12 months.

Another potentially useful recommendation for dealing with recurrence of neurologic symptoms and signs after apparently successful HBOT of neurologic DCS has been advanced by Edmonds.[101] Frequent postrecompression recurrences of neurologic manifestations led him to institute multiple 30-minute,

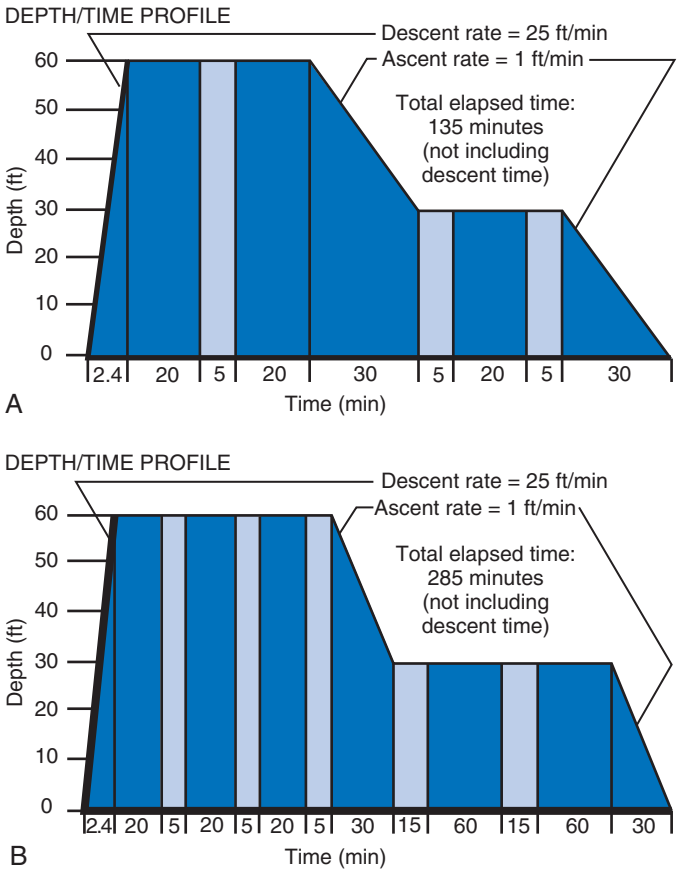

FIGURE 71-14 Examples of U.S. Navy decompression tables. *Dark shading* represents oxygen breathing; *light shading* represents air breathing. A, U.S. Navy Treatment Table 5, oxygen treatment of type 1 decompression sickness. B, U.S. Navy Treatment Table 6, oxygen treatment of type 2 decompression sickness. *(Modified from Department of the Navy:* U.S. Navy diving manual, *vol 2, rRv 2, Flagstaff, Arizona, 1988, Best Publishing.)*

sea-level, oxygen-breathing periods with 30-minute air breaks for 6 to 8 hours following treatment and observation in the hospital, rechecking the vital capacity frequently to detect and prevent pulmonary oxygen toxicity.

Remote treatment of DCS when access or transport to a recompression facility is not possible is controversial. Mild DCS (limp pain or rash) with no neurologic manifestations can be treated without recompression if HBOT is not possible. A consensus guideline for management of mild DCS in remote locations has been published.[203] In-water recompression has been used successfully for severe neurologic DCS in remote locations; however, in-water recompression carries a significant risk of drowning, hypothermia, hyperoxia, and dehydration. In-water recompression requires special training and equipment, and is beyond the scope of this chapter.[26-28,240,241,278]

Adjunctive Treatment

Corticosteroids. Although experimental proof of their efficacy is lacking, high-dose parenteral corticosteroids were widely used in the past as an adjunct to recompression treatment of both neurologic DCS and AGE. Anecdotal data suggesting that steroids were beneficial in combination with HBOT have been reported,[74,154,178] but there have been no published clinical series or controlled trials demonstrating their efficacy. In contrast, controlled studies of high-dose parenteral dexamethasone and methylprednisolone in DCS-affected dogs and pigs showed that use of glucocorticoids as an adjunct to conventional HBOT produced no benefit, and even suggested that the animals treated with steroids had inferior outcomes.[91,115]

Anticoagulants and Nonsteroidal Antiinflammatory Drugs. Because intravenous bubbles can induce platelet adherence, antiplatelet agents, such as aspirin and nonsteroidal antiinflammatory drugs (NSAIDs), have been tried prophylactically and therapeutically, but without success. Although use of tenoxicam has been shown to decrease the total number of treatments required for DCS, the outcomes of the divers in the control and treatment groups were unchanged.[19] Therefore, NSAIDs and aspirin are not routinely recommended as adjuncts for treatment of DCS. Of note, divers who are paralyzed from DCS should be treated (e.g., with subcutaneous low-molecular-weight heparin) to prevent thromboembolic disease.

PREVENTION

Ever since DCS was first recognized, efforts have been directed at prevention. Such efforts have used all manner of interventions, but to date the only proved way to prevent DCS is to limit the time a diver spends at increased pressure (depth) and to ensure that decompression from increased pressure is sufficiently slow, or staged, so that the body's burden of excess inert gas is eliminated without forming bubbles. Such depth/time ascent schedules have given rise to a variety of decompression tables.

A number of preconditioning techniques, such as exercise, vibration, antioxidants, and time spent in a heated sauna, have been investigated as protective measures against stress and DCS. Studies suggest that a single bout of moderately intense aerobic exercise 2 to 24 hours prior to diving significantly decreased the bubbles detected by ultrasound in the pulmonary artery after diving.[25,52,92] As discussed above, the presence of VGEs does not predict DCS but may be a marker of decompression stress. Other benefits of exercise may include activation of heat shock protein and increased nitric oxide production, resulting in protection of endothelial function..[93,194] Madden and colleagues[188] demonstrated that high-intensity cycling before diving decreased VGEs, circulating microparticles, and neutrophil response. They suggested that exercise may decrease preexisting gas micronuclei before the dive, resulting in decreased VGEs after diving.

Predive vibration[124] and predive sauna treatment[24] both show a decrease in VGEs. Predive antioxidants have been reported to decrease brachial artery flow–mediated dilation, suggesting that antioxidants may reduce endothelial dysfunction in divers.[223] The proposed mechanism behind these preconditioning methods remains unclear.

DECOMPRESSION SICKNESS IN BREATH-HOLD DIVERS

Conventional wisdom has always held that DCS does not occur in breath-hold divers, because they do not breathe air at an increased atmospheric pressure. However, it appears that neurologic DCS occurs among professional breath-hold divers in some settings.

A condition known as *taravana* was first described among pearl divers in the Tuamotu Islands of the South Pacific in the early 1960s.[69,70] These breath-hold divers make 40 to 60 dives per day to depths of 24 to 30 msw (80 to 100 fsw), spending about 2 minutes underwater each dive and remaining on the surface for 3 to 4 minutes between dives. These divers often complain of vertigo, nausea, euphoria, numbness, and partial or complete paralysis of the extremities after several hours of diving. In some cases, the diver loses consciousness for seconds to minutes after noting symptoms. Many of these divers have died either suddenly or after developing other symptoms. Nonfatal symptoms are usually transient, resolving in minutes to a few weeks. The symptoms immediately resolve with recompression.[229]

The symptoms of the taravana diving syndrome are similar to what has been more recently reported as occurring among some Japanese Ama divers, who have been reported to suffer dizziness, nausea, euphoria, numbness and weakness of extremities, dysarthria, and loss of consciousness after several hours of making 20 to 40 dives per hour to depths of 9 to 27 msw (30 to 90 fsw).[164,166]

With both the Tuamotu pearl divers and Japanese Ama, as well as in one instance of a U.S. Navy diver making repetitive breath-hold dives to depths of over 100 fsw in a submarine escape tank training tower, only neurologic symptoms have been observed. Joint pain and skin signs that are often seen in DCS have not been observed.

The mechanism of CNS damage after repetitive breath-hold diving is poorly understood. Venous gas bubbles were described in an Ama diver who had made 30 dives to 45 to 50 fsw in less than an hour.[242] It is possible that inert gas accumulates in the peripheral tissues of these divers consequent to the repeated back-to-back exposure to elevated atmospheric pressures. Gas bubbles are then released from peripheral fatty tissues, bypass the usual lung-filtering mechanism via pulmonary arteriovenous or cardiac intraatrial shunts, enter the systemic circulation, and cause cerebral embolization. Nothing has been reported about the prevalence of PFO in these populations, but there is no reason to believe that it is less than in other populations where it has been studied (i.e., about 25% of people). It is also possible that intravascular microbubbles (i.e., bubbles < 21 μm in diameter) that would pass through the lungs[45,46] could impair the blood-brain barrier transiently,[141] although this alone seems unlikely to explain the cerebral lesions that have been demonstrated by MRI.[166] Whatever the mechanism, symptoms and neurologic lesions that these divers suffer after repetitive breath-hold dives are now well documented.[165,301]

LONG-TERM HEALTH EFFECTS OF DIVING

Long-term untoward health effects related to diving may result from CNS injury as a result of AGE or DCS. These effects may include motor paresis or paralysis; paresthesias and other sensory disturbances; bladder, bowel, and sexual dysfunction; seizures; hearing loss; vertigo, tinnitus, and balance disorders from inner ear injury; dysbaric osteonecrosis; dysbaric retinopathy; hearing loss; and possible personality changes and loss of intellect.

A number of informal observations have been made over the years suggesting that long-time divers suffer some loss of intellect and may experience significant changes in personality, although confounding variables (e.g., use of alcohol) have always complicated such observations. There is now increasing evidence that divers may suffer subclinical CNS injury from paradoxic gas embolization and, possibly, other mechanisms.[297] For example, in MRI studies, long-time divers have been found to have more

subcortical cerebral lesions than did controls. Studies in professional divers report decreased cognitive abilities, such as decreased mental flexibility, as well as lower verbal memory intelligence and sustained attention in divers reporting memory and concentration loss[67,268]; recreational divers do not show evidence of deficiencies of general higher cognitive function.[137] Overall, there is growing evidence that long-time divers suffer untoward health effects related to repeated exposures to a hyperbaric environment and breathing compressed gas.

UNUSUAL CONDITIONS OF UNCERTAIN ORIGIN FOLLOWING DIVES

IMMERSION PULMONARY EDEMA

Acute immersion pulmonary edema has been associated with both scuba diving and swimming. Numerous case reports describe scuba divers developing pulmonary edema while at depth during a dive.[134,238,258,299] In the majority of these cases, the divers were healthy with no predisposing factors for development of pulmonary edema (e.g., no evidence of cardiac disease or hypertension). Other reports describe pulmonary edema associated with heavy exertion in surface swimmers.[184,190,256,292] Many of these reports are of healthy males in military fitness training programs who presented with dyspnea, cough, tachypnea, hypoxemia, and hemoptysis after a strenuous swim. Immersion pulmonary edema is now recognized in triathlon swimmers.[49,198]

The exact mechanism for stress failure of the pulmonary capillaries is unknown. Racehorses are known to develop extremely high pulmonary vascular pressures, leading to stress failure of capillaries.[293] Physiologic changes that occur with immersion most likely contribute to the overall pathophysiology. Immersion causes central pooling of blood, which increases preload. Decreased core body temperature from cold water causes redistribution of blood flow to thoracic vessels and vasoconstriction. Increased sympathetic outflow also leads to vasoconstriction. Hence, immersion causes increased cardiac output and increased pulmonary vascular resistance. Reduction in functional residual capacity and vital capacity occurs by blood displacement into the lungs; there is also reduction in lung compliance.

Increased pulmonary blood flow and vascular pressure most likely cause stress failure of pulmonary capillaries. In scuba divers, use of a regulator with high inspiratory resistance could lead to negative-pressure pulmonary edema.

Radiographic findings include Kerley B lines, cephalization of flow, air-space consolidation, and normal cardiac size (Figure 71-15). Treatment is conservative because hypoxemia usually resolves within 12 hours. Treatment includes supplemental oxygen and inhaled β_2-adrenergic agonists for symptomatic relief. Diuretics are often given; however, most patients improve dramatically within the first 12 hours without their use. Older divers with cardiac risk factors need to be evaluated for a cardiac event that may have precipitated acute pulmonary edema. Reversible myocardial dysfunction has been reported in 28% of a group of divers with immersion pulmonary edema and associated with age over 50 years and hypertension.[122]

There is no way to predict whether or not a scuba diver will develop acute pulmonary edema; however, low lung volumes, pulmonary hypertension, and underlying cardiopulmonary disease, particularly occult hypertension, may be common predisposing factors for immersion pulmonary edema.[121,230,256] Many divers in the case reports have returned to diving without any recurrence of symptoms; however, divers must be cautioned about recurrence. Rare fatalities are reported from immersion pulmonary edema.[60,261]

INTERNAL CAROTID ARTERY DISSECTION

Dissection of the internal carotid artery and associated thromboembolic stroke have been reported (rarely) after scuba diving. These have been initially incorrectly diagnosed as AGE.[11,126,212]

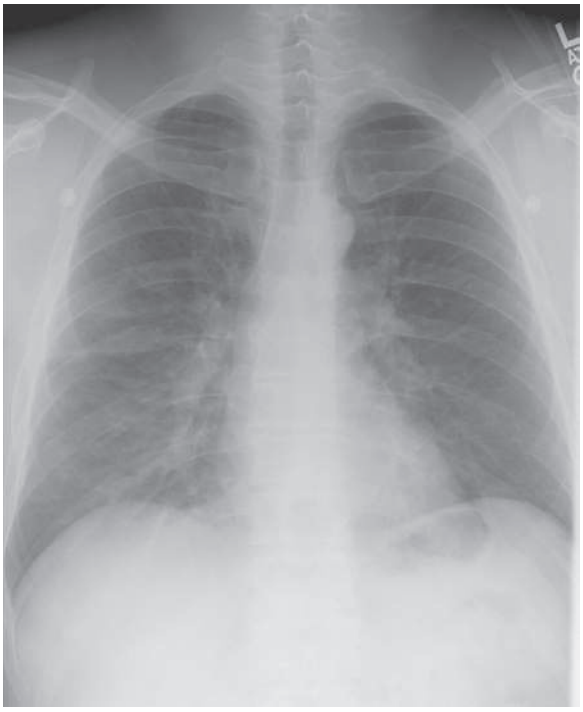

FIGURE 71-15 Chest radiograph of a diver with immersion pulmonary edema, showing bilateral patchy opacities consistent with pulmonary edema after an uneventful dive.

Although conceivably related to intravascular bubble-induced endothelial injury, the cause is a mystery.

Internal carotid artery dissection has been reported after a variety of situations involving neck trauma, hyperextension of the neck in athletic activities, and invasive diagnostic procedures, as well as spontaneously in persons having connective tissue disorders (e.g., Marfan's syndrome, fibromuscular dysplasia, and cystic medial necrosis), although its cause is unknown in most cases.

Symptoms of internal carotid artery dissection include unilateral headache, with or without ipsilateral neck pain, and other neurologic symptoms of intracranial origin. In all published cases involving divers, the condition was diagnosed after hyperbaric treatment for presumed AGE; HBOT produced no improvement in symptoms, or the condition worsened after HBOT. The preferred diagnostic modality is magnetic resonance or arteriography; ultrasound may be helpful.

Primary treatment of this condition entails anticoagulation aimed at preventing further thromboembolic complications. Most patients regain full function if appropriately treated.

MEDICAL FITNESS FOR DIVING

GENERAL CONSIDERATIONS

Although diving is an enjoyable sport that is safer than many other activities, the risks associated with diving are increased by certain physical conditions. Persons who intend to take up scuba diving should first be medically cleared. The diving examination should focus on conditions that may put the diver at increased risk for DCS, pulmonary barotrauma, and any conditions that could invoke Boyle's law or lead to loss of consciousness in the water. Special attention must be paid to the pulmonary, otolaryngologic, cardiac, and neurologic systems, as well as the person's psychological stability. The diver must be able to withstand cold stress and the physiologic effects of immersion and have sufficient exercise capacity to deal with possible stressful situations and emergencies. An increasing number of fatalities in diving are associated with cardiovascular disease, so particular attention should be directed to whether the diver can be expected

to meet the performance requirements likely to be encountered in diving.

In general, contraindications for diving fall into five general categories:

1. Persons who are unable to equalize pressure in one or more of the body's air spaces and are thus at increased risk for barotrauma.
2. Persons who have a medical or psychiatric condition that may become manifest underwater or at a remote diving site and endanger the diver's life because of the condition itself, because it occurs in the water, or because inadequate medical help is available.
3. Persons who have impaired tissue perfusion or diffusion of inert gases and thus increased risk for DCS.
4. Persons who are in poor physical condition and thus at increased risk for exertion-related medical problems (the factors compromising the physical condition may be physiologic or pharmacologic).
5. Women who are pregnant, because the fetus may be at increased risk for dysbaric injury.

MEDICAL CLEARANCE FOR DIVING

Different medical standards are in place for the various diving communities: recreational, scientific, commercial, and military. The most common questionnaire used by recreational divers is the Recreational SCUBA Training Council Medical Statement, which can be downloaded at wrstc.com/standards-downloads/. Medical standards for scientific divers fall under the auspices of AAUS and can be downloaded at aaus.org/diving_standards. The Association of Diving Contractors International, Inc. lists medical standards for commercial diving that can be downloaded at adc-int.org.

Medical clearance guidelines divide the risks of diving into severe risk, relative risk, and temporary risk. *Severe risk* includes conditions that are absolute contraindications to diving, as well as conditions that put the diver at significant risk for injury or drowning. A potential diver with any of these conditions should be discouraged from diving. *Relative risk* refers to conditions that might represent a moderately increased risk that may be acceptable to the diver. The physician must base his or her judgment on assessment of the individual patient in each of these cases. *Temporary risk* may preclude diving while a condition is being treated. The diver may dive safely once the condition has resolved.

Table 71-6 lists the general guidelines for medical clearance for diving. Physicians and medical personnel of the Divers Alert Network (DAN) are available for consultation by telephone during normal business hours (919-684-2948) for all issues related to fitness for diving.

SPECIFIC CONDITIONS OF CONCERN WITH REGARD TO DIVING

Neurologic Disorders

Any neurologic abnormality that affects a diver's exercise capacity, leads to unconsciousness underwater, or has waxing and waning neurologic symptoms that can be difficult to distinguish from neurologic DCS must be strongly considered to be a contraindication to diving. Persons with spinal cord or brain abnormalities in which perfusion is impaired may have a theoretically increased risk for DCS.

Seizures. A history of epilepsy or other seizure disorder has traditionally been an absolute contraindication for diving. Seizures occurring underwater almost always lead to fatal drowning. Even if seizures are well controlled with medication, the individual should be disqualified from diving. Factors involved with diving (stress, exercise, hyperventilation, hypothermia, hypercapnia, and sensory deprivation) may lower the seizure threshold. Elevated oxygen partial pressures are suggested to increase the likelihood of a seizure in a diver with epilepsy; however, there are no data to support this notion.[2] Investigators have proposed that individuals with well-controlled seizures may be allowed to dive only if wearing a full-face mask. If a seizure occurs underwater, it is conjectured that it is unlikely the diver will drown.[2,259]

Once an individual has discontinued antiepileptic medication for 5 years and has a normal neurologic workup (including an electroencephalogram [EEG]), the person may return to diving because the risk for having another seizure is the same as for the general population. A history of febrile seizures in childhood, seizures due to sepsis or drug ingestion, or posttraumatic seizures of childhood are not disqualifying for diving, as long as there is no ongoing seizure problem.

Head Injury. Any significant head injury that increases the risk for delayed seizures is a contraindication to diving. Such injuries include significant brain contusion, subdural hematoma, skull fracture, or loss of consciousness or amnesia for more than 24 hours. In a case of minor head injury that does not have any associated symptoms and that does not require anticonvulsant medication, scuba diving can be considered after 6 asymptomatic weeks.[4]

Unexplained Syncope. Unexplained loss of consciousness should be viewed as a contraindication to diving, for the same reasons as for seizures.

Migraine Headaches. Scintillating scotomata, paresthesias, weakness, and other neurologic symptoms associated with migraines and other types of vascular headaches may be confused with symptoms of AGE or DCS if they occur soon after diving. If such symptoms are misinterpreted, it may lead to unnecessary medical evacuation and HBOT. Depending on the specific situation, migraine headaches may be a relative contraindication to diving.

Cardiovascular Disorders

Immersion in water causes redistribution of blood from the periphery to the core. Increased cardiac preload during immersion can precipitate pulmonary edema in patients with impaired left ventricular function, increased pulmonary artery pressure, or significant valvular disease. Because an increasing proportion of scuba diving fatalities are due to coronary artery disease, individuals over 40 years of age should undergo risk assessment for coronary artery disease prior to diving.

Atrial Septal Defect. An atrial septal defect allows right-to-left intracardiac shunting of blood and increases the risk for paradoxic embolization of bubbles[298]; hence, the presence of an atrial septal defect is an absolute contraindication for diving. A ventricular septal defect does not allow right-to-left shunting and is not a contraindication for diving.

Patent Foramen Ovale. Patent foramen ovale (PFO) and diving has gained a lot of attention in the last few years. A PFO has the potential for a right-to-left shunt, and several studies suggest that a PFO may increase the risk for serious DCS.[72,123,208,253,276,285,296,297] However, the overall risk for DCS in divers with a PFO is very small, and routine screening for a PFO is not indicated.[32] It is reasonable to look for a PFO in cases of neurologic DCS, because the risk for right-to-left shunting increases with the size of the PFO, and some data suggest a significantly higher risk for recurrent serious DCS in persons having a larger (grade 2 or 3) PFO.[276]

If a PFO is identified, a diver may consider closure of a PFO to continue diving. However, this remains controversial. Honek and associates reported on 47 divers found to have a large PFO on medical screening.[144] Twenty divers who had suffered "unprovoked" decompression illness underwent transcatheter closure, and 27 divers (who either had not suffered decompression illness despite the presence of the PFO or who declined closure) were not closed. Not surprisingly, PFO closure reduces arterialization of venous inert gas bubbles after chamber dives; however, there was no difference in clinical outcomes. Billinger and colleagues[22] demonstrated reduction in decompression illness after PFO closure. Over a 5-year follow-up, the incidence of neurologic decompression illness per 10,000 dives in the closed and unclosed groups was 0.5 and 35 cases, respectively, which barely reached statistical significance because there were only five cases of decompression illness (one in the closure group and four in the nonclosure group). Closing a PFO may reduce the risk of neurologic decompression illness, but the procedure carries risks and

TABLE 71-6 Guidelines for Medical Clearance for Recreational Diving

System	Severe Risk Conditions	Relative Risk Conditions	Temporary Risk Conditions
Neurologic	Seizures Transient ischemic attack or cerebrovascular accident Serious decompression sickness with residual deficits	Complicated migraine Head injury with sequelae Herniated disk Peripheral neuropathy Multiple sclerosis Spinal cord or brain injury Intracranial tumor or aneurysm	Arterial gas embolism without residual, in which pulmonary air trapping has been excluded and the probability of recurrence is low
Cardiovascular	Intracardiac right-to-left shunt (atrial septal defect) Hypertrophic cardiomyopathy Valvular stenosis	Coronary artery bypass grafting Percutaneous transluminal coronary angioplasty or coronary artery disease History of myocardial infarction Congestive heart failure Hypertension Dysrhythmias Valvular regurgitation	Pacemaker: if the problem necessitating pacing does not preclude diving; pacemakers must be certified by the manufacturer to withstand pressure
Pulmonary	Spontaneous pneumothorax Impaired exercise performance due to respiratory disease	Asthma or reactive airway disease Exercise-induced bronchospasm Solid, cystic, or cavitating lesions Pneumothorax caused by surgery, trauma, or previous overinflation Immersion pulmonary edema Interstitial lung disease	
Gastrointestinal	Gastric outlet obstruction Chronic or recurrent small bowel obstruction Severe gastroesophageal reflux disease Paraesophageal hernia	Inflammatory bowel disease Functional bowel disorders	Unrepaired hernias of the abdominal wall Peptic ulcer disease associated with obstruction or severe reflux
Metabolic and endocrine	Pregnancy	Insulin-dependent diabetes mellitus Non–insulin-dependent diabetes mellitus	
Otolaryngologic	Open tympanic membrane perforation Tube myringotomy Middle ear or inner ear surgery Tracheostomy	Recurrent otitis externa, otitis media or sinusitis Eustachian tube dysfunction History of tympanic membrane perforation, tympanoplasty or mastoidectomy Significant conductive or sensorineural hearing loss History of round or oval window rupture	Acute upper respiratory infection Acute sinusitis Acute otitis media
Orthopedic		Amputation Scoliosis with impact on respiratory performance Aseptic necrosis	Back pain
Hematologic		Sickle cell disease Leukemia Hemophilia Polycythemia vera	
Behavioral health	Inappropriate motivation to dive Claustrophobia Acute psychosis Untreated panic disorder	Use of psychotropic medications Previous psychotic episodes	

Modified from Recreational SCUBA Training Council (RSTC) Medical Statement: wrstc.com.

should be carefully considered on a case-by-case basis. Commercial and technical divers who perform dives that are considered provocative might be appropriate candidates. Other divers may choose to adopt more conservative dive profiles.

The South Pacific Underwater Medicine Society (SPUMS) and United Kingdom Sports Diving Medical Committee (UKSDMC) have published a joint position statement on persistent foramen ovale and diving.[260] In 2015, the Undersea and Hyperbaric Medical Society (UHMS) and DAN held a 1-day workshop to establish consensus guidelines from the joint position statement on PFO and diving. Workshop proceedings are available at diversalertnetwork.org.[79a] Box 71-6 summarizes the guidelines on PFO and diving.

Coronary Artery Disease. As scuba diving has become more popular, older divers are pursuing the activity and more deaths from coronary artery disease are occurring among scuba divers in the water. In recreational divers, cardiac events are associated with approximately a quarter of diving fatalities.[80] In addition to the need for cardiac reserve during an in-water emergency, carrying tanks, donning equipment, and swimming against water currents entail significant physical stresses. Symptomatic coronary artery disease is a contraindication for diving. However, a history of myocardial infarction is not a disqualification for sport diving per se, particularly after revascularization or comparable procedures, and if the diving candidate is able to achieve 6 metabolic equivalents during an exercise stress test.[200]

Dysrhythmias. Benign dysrhythmias that do not interfere with exercise tolerance are not disqualifying. Individuals with well-controlled atrial fibrillation who are taking anticoagulation medications should be able to scuba dive without undue risk.

Hypertension. Whether a hypertensive person may safely dive is based largely on the therapy required for blood pressure

BOX 71-6 Position Statement on Patent Foramen Ovale (PFO) and Diving by the Undersea Hyperbaric Medicine Society (UHMS) and the Divers Alert Network (DAN)

Statement 1
Routine screening for patent foramen ovale (PFO) at the time of dive medical fitness assessment (either initial or periodic) is not indicated

Statement 2
Consideration should be given to testing for PFO under the following circumstance:
- A history of more than one episode of decompression sickness (DCS) with cerebral, spinal, vestibulocochlear or cutaneous manifestations. Noncutaneous manifestations of mild DCS and headache as an isolated symptom after diving are not indications for PFO investigation.

Statement 3
If testing for PFO is performed, then the following is recommended:
- That testing is undertaken by centers well practiced in the technique
- The testing must include bubble contrast, ideally combined with transthoracic echocardiogram (TTE), because this best facilitates cooperation with provocation maneuvers. Use of two-dimensional and color-flow echocardiography without bubble contrast is not adequate.
- The testing must include the use of provocation maneuvers to promote right-to-left shunting, including Valsalva release and sniffing as described in the supporting references (both undertaken when the right atrium is densely opacified by bubble contrast).

Statement 4
Interpreting a positive PFO screening result:
- A spontaneous shunt without provocation or a large, provoked shunt following diving when venous gas emboli are present is recognized as a risk factor for those forms of DCS listed in Statement 2.
- Smaller shunts are associated with a lower but poorly defined risk of DCS. The significance of minor degrees of shunting needs to be interpreted in the clinical setting that led to testing.

Statement 5
Following diagnosis of a PFO considered likely to be associated with increased DCS risk, the diver may consider the following options in consultation with a diving physician:
- Stop diving
- Dive more conservatively: There are various strategies that might be employed to reduce the risk of significant venous bubble formation after diving, or the subsequent right-to-left shunting of such bubbles across a PFO. The appropriateness of this approach, and the strategies chosen, need to be considered on an individual basis, and in discussion with a diving medicine expert. Examples include: reducing dive times to well inside accepted no-stop limits; performing only one dive per day; use of nitrox with air dive planning tools; intentional lengthening of a safety stop or decompression time at shallow stops; avoidance of heavy exercise and unnecessary lifting or straining for at least 3 hours after diving
- Close the PFO. It is emphasized, however, that closing a PFO after an episode of DCS cannot be considered to provide assurance that DCS will not occur again.

Statement 6
The options outlined in Statement 5 require careful consideration of the risks and benefits and the clinical setting that led to screening.

Statement 7
Following closure of a PFO and before returning to diving, the diver requires a repeat bubble contrast echocardiogram demonstrating shunt closure, a minimum of 3 months after the closure.

Statement 8
Diving should not be resumed until satisfactory closure of the PFO is confirmed, and the diver has ceased potent antiplatelet medication (aspirin is acceptable)

Statement 9
Venous bubbles can also enter the systemic circulation through intrapulmonary shunts, although the role of this pathway in the pathogenesis of DCS is not as well established as PFO. These shunts are normally closed at rest. They tend to open with exercise, hypoxia, and beta-adrenergic stimulation, and close with hyperoxia. It is therefore plausible that exercise, hypoxia, and adrenergic stimulation after a dive could precipitate DCS when it might not otherwise have occurred, while supplemental oxygen is likely to minimize this effect.

From Denoble PJ, Holm JR, eds. Patent foramen ovale and fitness to dive consensus workshop proceedings. Durham, NC, Divers Alert Network, 2015.

control and the presence of any other contraindications that are often associated with hypertension (e.g., coronary artery disease or diabetes). Diving has little effect on blood pressure, and when a regimen of weight control, diet, and antihypertensive medication is successful, diving usually can be allowed. Occult hypertension is thought to be a risk factor for immersion pulmonary edema.[121]

Pulmonary Disorders

Any lesion or disease that impedes airflow from the lungs increases the risk for pulmonary barotrauma and the possibility of AGE. Additionally, respiratory disease due to either structural disorders of the lungs or chest wall may impair exercise performance and is exacerbated by the effects of immersion and increased gas density while diving.

Spontaneous Pneumothorax. A history of a spontaneous pneumothorax is an absolute contraindication for diving. Individuals with a history of spontaneous pneumothorax are often found to have small blebs on the surface of the lungs that may be at increased risk for rupture while diving, causing pulmonary barotrauma and AGE. A history of previous spontaneous pneumothorax carries a significant incidence of recurrence, even after many years, and the candidate must be advised against compressed-gas diving. A pneumothorax that occurs while the diver is underwater or in a hyperbaric chamber can rapidly progress to become a life-threatening tension pneumothorax as the pleural cavity air expands (per Boyle's law) during ascent. Surgical procedures designed to prevent recurrence (such as pleurodesis or apical pleurectomy) do not correct the underlying lung abnormality.

A history of a traumatic or iatrogenic pneumothorax is not a contraindication to diving. In these cases, inspiratory/expiratory high-resolution chest CT scanning can rule out significant air trapping.

Asthma. In the past, asthma was considered an absolute contraindication for diving because of the person's increased risk for air trapping and pulmonary overinflation while diving. However, many persons with asthma dive with no apparent increased risk for pulmonary barotrauma.[217] Criteria for clearing a person with a history of asthma to dive include:

1. Asymptomatic adult with a past history of childhood asthma
2. Well-controlled asthma with known triggers with normal pulmonary function tests (with or without medication) with a reduction of less than 20% in peak mid-expiratory flow after exercise
3. No evidence of cold-induced wheezing or exercise-induced bronchospasm

A more complete discussion of the issues relating to asthma and diving can be found in the article by Van Hoesen and Neuman.[283a]

Bullous Lung Disease. Pulmonary blebs or cysts can trap air and lead to local pulmonary overpressure accidents during decompression. If a ball-valve or flutter-valve effect allows such a bleb or cyst to equalize with the elevated breathing pressure during compression or descent, but blocks escape of air during decompression, rupture could cause pulmonary barotrauma and air embolism. Similarly, individuals with significant obstructive lung disease should be disqualified from diving.

Diabetes

Like asthma, insulin-dependent diabetes mellitus (IDDM; also known as type 1 diabetes mellitus) was formerly considered a contraindication for diving because of the risk for a hypoglycemic reaction underwater that would lead to incapacitation, unconsciousness, and drowning. Hypoglycemic reactions may result from sudden bursts of energy expenditure, as may occur in dealing with emergencies. Underwater incapacitation due to hypoglycemia not only endangers the life of the diver but may also risk the lives of other persons during rescue attempts, and can lead to drowning. However, many patients with IDDM have been diving safely for years, and guidelines for divers with IDDM exist through the American Diabetes Association and the YMCA. Persons with diabetes who are well controlled on insulin, accustomed to vigorous physical exercise, knowledgeable about their condition, and who dive with prepared buddies usually can be cleared for diving. Guidelines for recreational diving with diabetes can be found on the DAN website.

Pregnancy

There is near unanimity that a woman who is pregnant, or who may be pregnant, should suspend compressed-air diving until after delivery. Animal studies have produced conflicting results in different species and laboratories, but the possibility of bubble formation in fetal or placental tissues leading to fetal demise or malformation is a concern, even during a dive that is safe for the mother.

Ear, Nose, and Throat Disorders

Any condition that inhibits or precludes the ability to clear the ears or sinuses may be disqualifying for diving. Likewise, any condition that impairs eustachian tube function may preclude an individual from diving; such conditions include frequent ear infections, mastoiditis, and cholesteatoma; a history of surgery for a middle ear disorder may also be a factor in precluding diving. A chronic perforated tympanic membrane is a contraindication for diving. A healed or repaired tympanic membrane perforation is usually not a problem for diving. Persons who have had a stapedectomy or stapedotomy should not dive, because of increased risk for oval window fistula and inner ear barotrauma. Chronic sinus disease or polyps, allergic rhinitis, or nasal septal deviation may make clearing the sinuses difficult. Individuals with inner ear disease, such as Ménière's disease, with recurrent vertigo should not dive.

Sickle-Cell Disease or Trait

The chances that a sport diver will breathe a hypoxic gas mixture are remote, but possible. Other concerns are heavy exertion in cold water or local compromise of microvascular blood flow by bubble evolution during decompression, which could lead to sickling and a vicious cycle of hypoxia, leading to further sickling.

Panic Disorders

A person prone to panic attacks may have such an attack underwater, prompting the diver to make a rapid uncontrolled ascent that may precipitate pulmonary barotrauma and AGE. The majority of dive accidents usually result from human error, not medical problems. Any condition that can lead to poor judgment and panic underwater should be disqualifying for diving.

Abdominal Hernias

There is a rare but potential risk for trapping expanding gas in a herniated loop of bowel during ascent. In general, diving should be suspended until surgical repair is completed.

Poor Physical Condition

Sport scuba diving may seem deceptively easy until an emergency occurs that requires swimming against a current, rescue of a buddy diver, or other vigorous activity. The diver should be capable of performing sudden strenuous activity before entering the water. Regular swimming or other exercise programs to ensure cardiovascular fitness are encouraged.

MEDICATIONS AND DIVING

In a survey of 531 divers, more than 50% reported taking over-the-counter medications within 6 hours of diving and 23% reported taking prescription medications (10% reported taking cardiovascular medication).[89] Many recreational divers today have underlying medical conditions that require chronic medication use. Lack of prospective experimental data on the impacts of increased pressure or a gas mixture on medication metabolism poses a challenge to physicians who desire data-driven answers. However, most diving and hyperbaric physicians approach the question of medical fitness to dive less from a medication perspective (i.e., the possible influences of depth or a gas mixture on a particular medication's actions) and more from consideration of the tangible influences of the patient's underlying medical conditions. As an example, persons with heart failure are poor candidates for diving because of their underlying heart disease, not because they take medications to control blood pressure. The critical caveat to this approach is that any medication that alters consciousness, impairs judgment, or decreases response time must be scrutinized. Although it is possible that the effect of such medications is increased secondary to the narcotic effects of nitrogen, the fact that these effects exist at all should warrant caution and second thoughts before initiating a dive.

An unusual situation in this regard, but one that is certainly plausible in today's scuba diving population, involves persons who have been treated with bleomycin (e.g., for testicular cancer). The concern is that bleomycin sensitizes the lungs to oxygen toxicity. Scuba divers who dive to depths of 18 to 36 msw (60 to 120 fsw) on compressed air would be exposed to the equivalent of 0.63 to 1.05 ATA oxygen, an amount sufficient to cause concern, because there is no known minimal safe oxygen exposure in bleomycin-treated patients.[151,304] A recent review of patients with a history of bleomycin use treated with HBOT did not demonstrate significant pulmonary complications. Although the clinical setting of HBOT exposes people to oxygen partial pressures of 2 to 3 ATA, these patients require close observation and usually undergo regular pulmonary function testing. As such, there is generalized caution among diving physicians when determining the suitability of diving for patients who have taken this medication.[275]

Another area of concern involves medications whose side effects may create diagnostic confusion in the case of DCS. An example of such a medication is mefloquine (Larium), used for malaria prophylaxis. Among its known potential side effects are vertigo, nausea, dizziness, anxiety, hallucinations, mania, sleep disturbances, and seizures. Depending on the specific circumstances, the side effects of the drug could be confused with DCS or AGE.

The side effects of some drugs may be especially problematic for divers because of the environment of diving. For example, divers in tropical locations may be exposed to intense sunlight, increasing the risk for a phototoxic reaction, which is a recognized side effect of many commonly prescribed medications. A few drugs have been studied under hyperbaric conditions and found to be at least relatively safe for divers; these include pseudoephedrine,[36,269] transdermal scopolamine,[23,294] and the antihistamine clemastine.[257] Other drugs that appear to be safe by virtue of their extensive use by divers and absence of reported untoward effects include aspirin, acetaminophen, warfarin, chlorpheniramine, and oxymetazoline.

Given the unknowns about the behavior of most medications in the diving environment, the following precautions are appropriate:

1. Whenever possible, avoid all drugs when diving.
2. Any medication that impairs mental judgment or physical capacity should be very carefully considered before being used when diving. If the medication is used, the expected benefits of its use should outweigh its risks.
3. Never take a drug for the first time before diving.
4. Always consider whether the reason for taking a drug is reason itself not to dive.
5. Always consider the known side effects of a drug and whether those would be a problem when diving.

DIVING WITH DISABILITIES

In recent years, many persons with limb amputations or other serious orthopedic impairment, spinal cord injury, cerebral palsy, or similar physical condition have sought to participate in scuba diving. Some of these persons were accomplished athletes or divers before an accident changed their physical status. For others, scuba diving is a completely new experience, offered as part of a rehabilitation program. Whatever the case, it is clear that persons having "disabilities" can enjoy diving as much as anyone else if they are properly trained, understand their condition and the limitations it causes them, and make appropriate adjustments, when necessary, for their condition.[78] When done in this context, scuba diving can open exciting new vistas for some disabled persons. Williamson and coworkers[295] demonstrated significant improvements in self-concept and body image among a group of young people with disabilities, including brain damage following head injury, congenital deafness, blindness, spinal cord dysfunction, and major limb amputations. These individuals were examined according to standard diving medicine practice regarding pulmonary and ear, nose, and throat status, and detailed psychological testing. Motivation proved to be an important predictor of success. The subjects were given extensive scuba diving training with a minimum of one-on-one instructor attention.

In any of these situations, the diver and buddy need to fully understand the concerns attendant to the condition (e.g., autonomic dysreflexia, skin breakdown, personal hygiene, and a possible increased risk for DCS) and the wisdom of taking a conservative no-decompression approach to diving.

FLYING AFTER DIVING

Diving is often done at remote destinations, and many divers travel considerable distances after packing in as many dives as possible. The question of when it is safe to fly after diving comes up often. Flying too soon after diving can seriously jeopardize decompression safety, leading to development of DCS during or after the flight because of the reduced atmospheric pressure present in most commercial aircraft.

The normal commercial aircraft cabin pressure is equivalent to an altitude exposure of approximately 1500 to 2500 m (5000 to 8000 feet), which is sufficient pressure reduction to cause dissolved nitrogen to come out of solution and form intravascular bubbles. Based on published DAN data gathered from 1987 to 1999, 17% (382/2222) of divers in the DAN injury database had their first symptom during or after flying.[117]

With continued growth of the dive-travel industry, the issue of when it is safe to fly after diving is important. Based on years of experimental work done by DAN in collaboration with Duke University, the following guidelines for flying in commercial aircraft after recreational diving have been established[255]:

1. For single no-decompression dives, a minimum preflight surface interval of 12 hours is suggested.
2. For multiple dives per day or multiple days of diving, a minimum preflight surface interval of 18 hours is suggested.
3. For dives requiring decompression stops, there is insufficient experimental or published evidence on which to base a recommendation. However, it would seem prudent that such divers should exercise caution, and a preflight surface interval substantially longer than 18 hours seems warranted. The longer the interval between diving and flying, the lower the DCS risk.

Two recent studies have looked at in-flight echocardiography in divers returning from a week of diving and found that a small percentage of divers develop venous bubbles during flight despite a preflight surface interval of 24 hours. The majority of bubbles occurred in individuals who also developed bubbles after diving.[55,56] None of these divers developed DCS.

Despite these guidelines, it is important to emphasize that no rule about flying after diving can be guaranteed to prevent DCS. These recommendations simply represent the best estimate of a safe surface interval for the majority of sport divers.

SAFE SCUBA DIVING

Sport scuba divers are fortunate that the most colorful marine life and abundant natural light exist at shallow depths. This obviates the need to dive deep. Indeed, 130 fsw should be considered a deep dive for sport diving and, for all intents and purposes, the maximum depth for recreational diving.

Recreational diving is usually done hours to days away from the nearest hyperbaric chamber, so the occurrence of DCS or AGE usually necessitates a major effort to evacuate an afflicted diver to the chamber. This often requires the use of special aircraft. Unfortunately, the delay between symptom onset and treatment may cause the damage to be poorly responsive to hyperbaric treatment. Therefore, divers should do their utmost to avoid developing AGE or DCS.

The need to take a conservative approach to depth and time is even more compelling when one considers that individual variability in DCS susceptibility, workload during the dive, water temperature, and exercise or altitude exposure following a dive may confound any set of decompression tables or dive computers. Indeed, the potentially devastating consequences of DCS, even with the most vigorous hyperbaric treatment, mandate that divers always dive with the prevention of this disease foremost in their minds.

Based on common trends seen in diving accidents and fatalities, the following suggestions for safe diving are recommended[213]:

- Ensure physical fitness to dive: train for your sport and be sure that you exercise regularly and follow a healthy diet.
- Use the buddy system.
- Follow your training: check your gauges often, respect depth and time restrictions, and do not dive beyond your training limits.
- Weight yourself properly and remember to dump your weights when appropriate.
- Ensure that your skill level and familiarity are appropriate for conditions.
- Have equipment serviced and maintained regularly.
- Account for all divers (a physical, individual response should be received from every diver before entering and after exiting the water).
- Avoid overhead environments (e.g., caves and tunnels) unless you are properly trained and equipped.
- Breath-hold divers should remember to use the buddy system and be aware of the dangers of shallow water blackout.

Diving in mountain lakes requires significant adjustments in decompression tables to account for decreased atmospheric pressure at the surface of the lake. Dive computers must be calibrated for altitude. Boni and associates[30] pointed out that for the same depth bottom time of a dive, surfacing at a lower ambient air pressure than sea level necessitates longer decompression time. Several decompression tables for altitude diving have been calculated and tested in the field.[15,71,209] The U.S. Navy Standard Decompression Tables can be used up to an altitude of 701 m (2300 feet).

Even when safe diving practices are followed, unexplained DCS cases sometimes occur.[1] Two mechanisms are postulated to account for these cases. After an otherwise safe dive, one well within the specified decompression procedure, elevated inert gas tensions exist in tissues and venous blood, and some individuals may develop VGEs. If a physiologic PFO allows intermittent retrograde flow from the right to left atrium, VGEs can become AGEs. The potential for this type of paradoxic gas embolism has been demonstrated in recent years for both PFO and other types of intracardiac and pulmonary shunts, as discussed above.

Another plausible explanation for DCS occurring after safe diving postulates that focal pulmonary barotrauma, or inadvertent breath holding during ascent, produces local air trapping during ascent and releases microbubbles into the systemic circulation. These microscopic "seed" bubbles in arterialized blood pass through the capillary bed to become bubble nuclei in venous blood, precipitating overt bubble formation and the classic manifestations of DCS.

DIVE ACCIDENT INVESTIGATION

When investigating a dive accident, whether for treatment purposes or as part of a forensic evaluation in the event of a fatal accident, the investigator begins by taking a detailed dive accident history. A number of specific details must be determined about the patient's diving activities, time of symptom onset, and nature and progression of symptoms, in addition to the medical history and other information that should be obtained for any patient. The history related to diving should specifically solicit information in the following areas[214]:

1. Type of dive and equipment used. Inquire specifically as to whether a decompression computer, diving watch, and depth gauge were used. Was compressed air or mixed gas used, and what was the source of the gas? Was an RB used?
2. Number of dives; depth, bottom, or total dive times; and surface interval or intervals between dives for all dives in the 72 hours preceding symptom onset. This information will be needed by the diving medicine consultant because it allows calculation of any omitted decompression and thus helps decide the likelihood of the patient having DCS or other problems. Unfortunately, the diver's interpretation of whether required decompression was omitted cannot be the sole source of data; this is notoriously unreliable. If a dive computer was used, it should be checked for information about any omitted decompression times; one should also ascertain whether the computer was worn on all dives.
3. Whether and how much in-water recompression was attempted. This is relevant to the likelihood of the diver having DCS. If a dive computer was used, the diver should be asked whether the specified decompression profile was followed.
4. Site of diving (e.g., ocean, lake, or quarry) and the environmental conditions (e.g., water temperature or presence of current or surge) associated with the dive. These factors enter into the differential diagnosis and may raise the possibility of the symptoms being caused by something other than a bubble-related problem. For example, DCS is more common after diving in cold water (other things being equal), and motion sickness may develop in a diver swimming back to shore on a choppy sea, even if there was no problem with seasickness before or during the dive.
5. Presence of predisposing factors for DCS. A number of factors have been associated with increased risk for DCS. These include dehydration, vigorous exercise underwater, advanced age, obesity, poor physical conditioning, local physical injury, and multiple repetitive dives in individuals who have not been acclimatized.
6. Whether the dive was complicated by running out of air, an untoward marine animal encounter, trauma, or other unexpected event. For example, low back pain suggestive of DCS may be caused by muscle strain from lifting a scuba tank or climbing into the dive boat, and tingling or numbness in an extremity may be caused by jellyfish envenomation.
7. Whether the patient flew in an airplane, went jogging, or engaged in any other particular activity after diving but before the onset of symptoms. If so, the effect of the activity on the symptoms should be ascertained. Some activities (e.g., flying in an unpressurized aircraft or vigorously exercising immediately after diving) may precipitate DCS in someone who might otherwise not be affected, and trivial dysbaric symptoms may become severe after similar activities.
8. Time of symptom onset. Symptoms that began soon after getting in the water (e.g., nausea from motion sickness), even if they worsen afterward, are not likely to be from DCS. Pulmonary symptoms that develop at depth are much more consistent with immersion pulmonary edema than with AGE.

INVESTIGATION OF DIVING FATALITIES

In the event of a fatal diving accident, information gathered from the diving accident history should be supplemented with an appropriate diving accident autopsy, thorough evaluation of the diving equipment used, and a detailed environmental history.[75] Guidelines for scuba diver death investigations have been published.[40,50,51,214,283] An excellent symposium on investigation of diving fatalities for medical examiners and diving physicians in 2014 was sponsored by the UHMS and DAN.[79] Environmental factors, such as weather, currents, wave action, visibility, water temperature, potential for entanglement, and dangerous marine life, must be considered. The diving equipment should be carefully studied for proper function and the amount of compressed air in the tanks, and the air should be analyzed for contaminants. The diver's medical and psychological histories should be sought because they may contain clues to a coincidental medical event that led to the diver's death but was unrelated to the dive. Aside from obvious psychosocial risk factors, such as alcoholism, drug abuse, or panic disorder, use of a scuba dive for suicide or homicide must be considered.

Unique aspects of the diving victim autopsy should include careful search for subcutaneous emphysema or other physical signs of pulmonary barotrauma and search for signs of marine envenomation. For example, before the surface of the body is washed, it should be examined for evidence of nematocysts from coelenterate stings. In addition, in contrast to the thoracic incision being made first, the calvarium should be opened before other incisions are made in order to prevent accidental introduction of air into the intracranial circulation. A finding of gas bubbles in intracranial vessels may result from AGE or DCS. Postmortem introduction of gas into the cerebral veins can be avoided if the calvarium is opened underwater.[75] Likewise, the initial thoracic incision must be made with care to determine whether pneumothorax is present. A careful search should be made for gas bubbles in the major blood vessels and heart. The middle ear should be examined for the presence of blood, and tympanic membranes and sinuses examined for evidence of barotrauma.

In many scuba fatalities, the pathologist may ascribe the death to drowning when there is no evidence of a diving-related malady, such as air embolism or evidence of equipment malfunction. Sudden cardiac death due to underlying cardiac disease should also be considered because these cases should be reported as natural death and not drowning.[248]

Meticulous investigation of dive accidents is important to find equipment, procedural, or medical causes that could be useful for improving the safety of diving and to gather information for legal procedures that often follow diving accidents. A unique approach to diving fatality investigation exists in the San Diego Diver Death Review Committee, which consists of personnel from the San Diego Lifeguards, San Diego Police Department, Medical Examiner's Office, Scripps Institution of Oceanography, University of California San Diego Hyperbaric Center, U.S. Coast Guard, and San Diego State University Diving Safety. The committee represents a multiagency dive team for scene investigation, equipment analysis, autopsy by the medical examiner, and input from diving medicine specialists. Representatives from all these groups review each diver death in San Diego to determine the cause and manner of death. The committee provides related information to agencies and the public for prevention and education to promote safety for the local diving community.[249]

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CHAPTER 72

Hyperbaric Medicine

KAREN B. VAN HOESEN

Hyperbaric oxygen therapy (HBOT) is a method of treating both acute life-threatening and chronic conditions by delivering oxygen at partial pressures greater than the normal sea level barometric pressure of 760 mm Hg. The patient breaths 100% oxygen intermittently or continuously while inside a chamber that is pressurized to at least 1.4 times the atmospheric pressure at sea level. The field of hyperbaric medicine has shown increasing applicability in the past 60 years. An estimated 2000 hospitals offer HBOT, and another 500 to 700 non–hospital-based programs also offer HBOT. HBOT is useful for treating diving injuries, acute carbon monoxide poisoning, necrotizing fasciitis, crush injuries, problem wounds, and radiation injuries.

Portable hyperbaric chambers are available for use in remote wilderness settings; however, their use is currently limited to diving injuries and altitude illness. HBOT has been proposed for other applications related to wilderness medicine, such as frostbite, brown recluse spider bites, and heatstroke (Box 72-1). However, few data exist to support routine use of HBOT for these. Additionally, HBOT has been suggested as an adjunct to field management of combat trauma in remote settings.

One-year accredited training fellowships in diving and hyperbaric medicine, recognized by the Accreditation Council for Graduate Medical Education, are available throughout the United States. The American Board of Preventive Medicine and the American Board of Emergency Medicine offer subspecialty board certification in undersea and hyperbaric medicine for individuals who have completed fellowship training.

HISTORY OF HYPERBARIC MEDICINE

Treatment with compressed-air therapy dates to 1662, when the British clergyman Henshaw treated acute medical disorders with increased pressures and treated chronic disorders with decreased pressures.[197] Compressed air was used to treat caisson disease, or decompression sickness (DCS), in the late 1800s. Medical use of hyperbaric oxygen (HBO) began in the 1930s with the first systematic studies on the physiologic effects of oxygen.[13] Extensive research on oxygen toxicity and safe oxygen limits was conducted during the 1940s, when oxygen was incorporated into the treatment tables of the U.S. Navy for diving injuries.

True clinical hyperbaric medicine began in the 1950s and 1960s, with the use of HBOT for anaerobic infections, including gas gangrene and clostridial infections, and for carbon monoxide poisoning.[32,198] During the 1970s, HBOT was used widely to treat a variety of conditions with little scientific basis. In 1976, the Undersea Medical Society established a multidisciplinary committee to review research and clinical data on the uses of HBOT. Every 3 years, the committee publishes a critical review of approved indications for HBOT (Table 72-1).[246] In 1986, the Undersea Medical Society changed its name to the Undersea and Hyperbaric Medical Society to incorporate the expanding field of hyperbaric medicine.

MECHANISMS OF HYPERBARIC OXYGEN

HBOT is inhalation of oxygen at a pressure greater than atmospheric pressure measured at sea level, which is 1 atmosphere (atm). An individual is placed inside a hyperbaric chamber that is pressurized with air or oxygen. The atmospheres absolute (ATA) is the sum of the atmospheric pressure and the hydrostatic pressure as measured in the chamber. For every increase of 10 meters of seawater (msw; equivalent to 33 feet of seawater [fsw]), a pressure increase of 1 atm (760 mm Hg) is present. At 20 msw (66 fsw), the absolute pressure in the chamber is 3 ATA. Increasing the atmospheric pressure in the hyperbaric environment causes both mechanical reduction in bubble size and increased partial pressure of oxygen in tissues.

EFFECTS OF HYPERBARIC OXYGEN ON OXYGEN CONTENT

The oxygen content of blood is the sum of oxygen carried by hemoglobin and oxygen dissolved in the blood plasma. At 1 atm at sea level, 98% of the oxygen content of normal blood is bound to hemoglobin. The oxygen content of blood plasma is determined by the solubility of oxygen in plasma at 37° C (98.6° F), which at a partial pressure of oxygen (P_{O_2}) of 100 mm Hg results in oxygen in solution of only 0.31 mL/dL. Once hemoglobin is fully saturated, further increases in P_{O_2} can only increase the amount of oxygen physically dissolved in the plasma. As P_{O_2} increases in inspired air, the amount of oxygen dissolved in plasma increases linearly (Figure 72-1). For every atmosphere of pressure increase, 1.8 mL/dL of oxygen is dissolved in plasma. At 3 ATA, approximately 6.8 mL/dL of oxygen can be dissolved in plasma. Because normal oxygen extraction at the tissue level is 5 mL/dL (at normal cardiac output), plasma alone can carry enough oxygen to meet the metabolic needs of tissues. In addition, this increase in oxygen-carrying capacity dramatically increases the driving force for oxygen diffusion.

EFFECTS OF AN ELEVATED PARTIAL PRESSURE OF OXYGEN

The increase in tissue oxygen concentration has important physiologic and biochemical effects in both normal and diseased tissue. HBO causes vasoconstriction and edema reduction; inhibits neutrophil adhesion; inhibits infection; upregulates growth factors and growth factor receptors; modulates inflammation; increases neovascularization, angiogenesis, and osteogenesis; and reduces ischemia-reperfusion injury. Recent studies show that HBO stimulates vasculogenic stem cell growth and release of stem cells derived from bone marrow[148,217] (Box 72-2).

Recent work by Thom[217] shows that breathing oxygen at pressures greater than 1 ATA increases production of reactive oxygen species and reactive nitrogen species, which elicit signaling molecules in transduction pathways that then generate a variety of growth factors, hormones, hypoxia-inducible factor, stem and progenitor cells, and cytokines. Through these activation pathways, HBO improves neovascularization and improves postischemia tissue survival.

Vasoconstriction

Hyperoxia-induced vasoconstriction occurs in both the arterial and venous vasculature and has been demonstrated in cerebral, retinal, renal, muscular, and myocardial circulations. Despite reduction in blood flow from vasoconstriction, cellular metabolism is not compromised by increased tissue oxygen partial pressures and increased oxygen diffusion pressure.[65] Reduced edema occurs because filtration of capillary fluid is decreased, whereas vascular outflow and improved oxygen delivery are maintained. This vasoconstriction can be beneficial in peripheral edema and intracerebral edema, and decreases edema

BOX 72-1 Wilderness Applications for Hyperbaric Oxygen Therapy

Altitude illnesses
Diving injuries
  Decompression sickness
  Arterial gas embolism
Frostbite
Brown recluse spider bite
Heatstroke
Necrotizing fasciitis acquired by marine envenomation
Field management of combat trauma

in burns and postischemic tissues.[22,161,166,167] Blood flow in the microcirculation is improved through decreased interstitial fluid pressures from edema reduction. This effect is important for treatment of brain injuries from carbon monoxide poisoning or arterial gas embolism, spinal cord DCS, and crush injuries.

Bacteriostatic and Bactericidal Effects of Hyperbaric Oxygen

HBO has been shown to have a potent inhibitory effect on the growth of various microorganisms. HBO causes a direct bactericidal effect on anaerobic bacteria through production of toxic radicals in the absence of free radical degrading enzymes, such as superoxide dismutase, catalases, and peroxidases.[98] HBO also has a bacteriostatic effect on selected facultative and aerobic organisms.[110,260] A tissue P_{O_2} of 30 to 40 mm Hg is necessary for oxidative killing of microorganisms by neutrophils. Increased P_{O_2} in infected and hypoxic tissue enhances neutrophil function and return of antimicrobial activity. A single 90-minute exposure increases the respiratory burst activity of neutrophil-like cells after exposure and an increase in the phagocytosis of *Staphylococcus aureus*.[3] HBO also augments the bactericidal action of aminoglycosides and potentiates the effect of certain sulfonamides.[80,103] Aminoglycoside and cephalosporin antibiotic transport across the bacterial cell wall requires tissue oxygen tensions above 30 mm Hg. Overall, HBO enhances antibiotic transport and augments efficacy.

Angiogenesis

HBO influences angiogenesis and vasculogenesis, the two processes that cause neovascularization. Angiogenesis is growth of new blood vessels from local endothelial cells. Vasculogenesis is recruitment and differentiation of circulating stem and progenitor cells to form new blood vessels. HBO mobilizes circulating stem and progenitor cells in both humans and mice by stimulating bone marrow endothelial nitric oxide synthase.[68,72,220] HBO also stimulates stem cell growth factor production through augmented synthesis of hypoxia-inducible factor.[93,148] Some of the mobilized cells will migrate to peripheral sites, where they function as de novo endothelial progenitor cells and contribute to wound vasculogenesis.

BOX 72-2 Mechanisms of Hyperbaric Oxygen Therapy

Mechanical decrease in bubble volume
Vasoconstriction and reduction of edema
Bactericidal and bacteriostatic effects
  Has bactericidal effect on anaerobic bacteria
  Has bacteriostatic effect on select facultative and aerobic organisms
  Enhances transport and augments antibiotic efficacy
Angiogenesis
  Mobilizes circulating stem/progenitor cells
  Stimulates stem cell growth factor production through augmented synthesis of hypoxia-inducible factors
Wound healing
  Enhances collagen synthesis
  Stimulates angiogenesis
  Improves leukocyte function of bacterial killing
  Blunts systemic inflammatory responses
  Stimulates vascular endothelial growth factor release
  Induces platelet-derived growth factor receptor appearance
Diminishes ischemia-reperfusion injury
  Induces nitric oxide
  Decreases leukocyte venular endothelial adherence
  Decreases release of toxic oxygen species
Increases osteogenesis

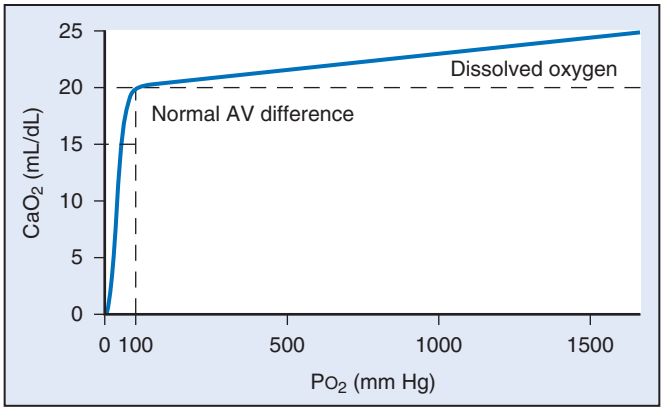

FIGURE 72-1 Effects of hyperbaric oxygen on the oxygen content of arterial blood. The blood oxygen content (CaO_2) under normal conditions is approximately 20 mL/dL and carried almost exclusively by hemoglobin. The normal oxygen extraction (arterial-venous [AV] difference) is approximately 5 mL/dL, or about one-quarter of total CaO_2. During hyperbaric oxygen therapy, dissolved oxygen in plasma can be increased to provide the entire AV difference without unloading oxygen from hemoglobin.

Wound Healing

The relationship between tissue wound hypoxia and wound healing has been well documented.[94,95] Tissue hypoxia decreases fibroblast replication, collagen deposition, angiogenesis, resistance to infection, and intracellular leukocyte bacterial killing, which are all oxygen-sensitive responses essential to normal wound healing. Intermittent elevation of tissue P_{O_2} reverses local tissue hypoxia and corrects the pathophysiology related to oxygen deficiency and impaired wound healing. HBO enhances collagen synthesis, stimulates angiogenesis, improves leukocyte function of bacterial killing, and blunts systemic inflammatory responses.[92,95,127] In addition, HBO stimulates vascular endothelial growth factor release and induces platelet-derived growth factor receptor appearance.[24,195,208] The net result of serial increased P_{O_2} from HBOT is improved local host immune responses, clearance of infection, enhanced tissue growth, angiogenesis with progressive improvement in local tissue oxygenation, and epithelialization of hypoxic wounds.

Ischemia-Reperfusion Injury

Ischemia-reperfusion injury is tissue damage that occurs after reperfusion of ischemic tissue. Ischemia-reperfusion injury is mediated by oxygen-derived free radicals, such as superoxide and hydroxyl free radicals, which are produced during prolonged periods of ischemia followed by reperfusion. These oxyradicals can lead to cell death by lipid peroxidation and generation of further free radicals. Oxyradicals are generated from xanthine oxidase and neutrophils. Neutrophil endothelial adhesion and subsequent release of free radicals play an important role in endothelial and microcirculatory damage.[107,184] Tissue reperfusion is inhibited by adherence of circulating neutrophils to vascular endothelium by $beta_2$ integrins.

HBO protects tissues from reperfusion injury. HBO provides additional oxygen so that reperfused tissues can generate scavengers such as superoxide dismutase, catalase, peroxidase, and glutathione, which detoxify destructive oxygen radicals before they damage tissues.[60] HBO antagonizes the $beta_2$ integrin system, which initiates adherence of neutrophils to postcapillary venule endothelium.[216,222] Reperfusion injury may be inhibited by HBO

TABLE 72-1 Approved Indications for Hyperbaric Oxygen Therapy

Indication	HBO Mechanism	Treatment Profile	Number of Treatments
Air or gas embolism	Bubble reduction Inert gas washout Modulation of ischemia-reperfusion injury and inflammation Treats ischemia	U.S. Navy Table 6 or 6A Repeat treatment with U.S. Navy Table 5 or 6, or 2.4 ATA	1-10 or to clinical plateau
Decompression sickness	Bubble reduction Inert gas washout Modulation of ischemia-reperfusion injury and inflammation Treats ischemia	U.S. Navy Table 6 Repeat treatment with U.S. Navy Table 5 or 6, or 2.4 ATA	1-10
Arterial insufficiencies Central retinal artery occlusion	Modulation of ischemia-reperfusion injury	2-2.8 ATA BID to clinical plateau Consider U.S. Navy Table 6 if no improvement	6
Enhancement of healing in select problem wounds	Treats ischemia Modulation of ischemia-reperfusion injury and inflammation Upregulation of growth factors, growth factor receptors, and circulation stem cells Inhibition of infection Angiogenesis	2-2.5 ATA	10-30
Carbon monoxide poisoning	Carbon monoxide washout Treats ischemia Modulation of ischemia-reperfusion injury and inflammation	2.4-3 ATA	1-3
Clostridial myositis and myonecrosis (gas gangrene)	Inhibition of toxins Suppresses organism growth Treats ischemia	3 ATA TID in first 24 hr, then BID for 2-5 days	3-10
Compromised grafts and flaps	Treats ischemia Angiogenesis Upregulation of growth factors, growth factor receptors	2-2.5 ATA BID initially until graft/flap appears stable	10-20
Crush injury, compartment syndrome, and acute ischemia	Treats ischemia Limits edema Modulation of ischemia-reperfusion injury and inflammation	2-2.5 ATA	2-14
Delayed radiation injury and soft tissue and bony necrosis	Treats ischemia Angiogenesis Upregulation of growth factors, growth factor receptors	2-2.5 ATA For prophylaxis: 20 sessions before surgery in radiated field 10 sessions after surgery	30-60
Idiopathic sudden sensorineural hearing loss	Supraphysiologic levels of oxygen in tissue	2-2.5 ATA	10-20
Intracranial abscess	Inhibition of infection Cerebral vasoconstriction and edema reduction Modulation of inflammation	2-2.5 ATA 1-2 times per day	10-20
Necrotizing soft tissue infection	Inhibition of infection Treats ischemia Modulation of inflammation	2-3 ATA BID until stabilization	5-30
Refractory osteomyelitis	Treats ischemia Inhibition of infection Stimulates osteoclasts	2-2.5 ATA	20-40
Severe anemia	Treats ischemia	2.4-3 ATA 3-4 times per day until replacement or red blood cells by regeneration or transfusion	1-20
Thermal burn injury	Inflammation modulation Upregulation of growth factors, growth factor receptors, and circulation stem cells Inhibition of infection	2-2.5 ATA TID in first 24 hours, then BID	20-30

ATA, atmospheres absolute; BID, twice a day; TID, three times a day.

through decreased leukocyte venular endothelial adherence, release of toxic oxygen species, and arteriolar vasoconstriction; hence, progressive arteriolar vasoconstriction is inhibited.[259] HBO inhibits intracellular adhesion molecule 1 expression, which plays a role in neutrophil adhesion and ischemia-reperfusion injury.[35] Additionally, HBO upregulates transforming growth factor $beta_1$, which decreases reperfusion injury by upregulating bcl-2 and inhibiting tumor necrosis factor alpha.[78] Nitric oxide plays an important role in ischemia-reperfusion injury, regulating vascular tone and neutrophil adhesion. HBO appears to increase nitric oxide production by inducing nitric oxide synthase production.[10,35,221] It is possible that the beneficial effect of HBO in ischemia-reperfusion injury is primarily mediated by nitric oxide.[34]

HBO increases the ischemic tolerance of multiple organs in animal models by induction of antioxidant enzymes and antiinflammatory proteins. HBO has been shown to be beneficial in almost all animal models of ischemia-reperfusion injury, and results in increased microvascular perfusion in skeletal muscle and improved skin flap survival.[116,252,259,261] It may be an important adjunct for treatment of tissue at risk for reperfusion injury that can accompany crush injury, limb replantation, compartment syndrome, arterial gas embolism (AGE), and DCS. New data suggest a role of HBO in preventing ischemia-reperfusion injury associated with myocardial infarction and cerebral ischemia.

TYPES OF HYPERBARIC CHAMBERS

MONOPLACE HYPERBARIC CHAMBERS

Monoplace, or single-person, chambers account for the majority of hyperbaric chambers in the United States. They accommodate a single patient, and the chamber is pressurized either with 100% oxygen or with air while the patient breathes oxygen through a mask or hood. Monoplace chambers are easier to maintain, require less space, and are less expensive than are multiplace chambers. They can be configured with noninvasive and invasive monitoring. Specially adapted ventilators and monitoring systems in some monoplace chambers allow for treatment of critically ill patients. During treatment, there is no direct access to the patient. Therefore, the chamber must be decompressed for an emergency, which can limit its use in some critically ill patients (Figure 72-2). Most monoplace chambers cannot exceed a treatment depth of 3 ATA.

MULTIPLACE HYPERBARIC CHAMBERS

Multiplace chambers hold two or more people and allow more than one patient to be treated at a given time, accompanied by attendants, including technicians, nurses, and respiratory therapists (Figure 72-3). The chamber is pressurized with air; 100% oxygen is delivered by face mask, using a head tent, or through an endotracheal tube. Multiplace chambers allow for intensive care–level monitoring, mechanical ventilators, vascular pressure monitoring, intravenous (IV) infusion pumps, blood gas analysis, and medication administration. Multiplace chambers can maintain a treatment pressure of 6 ATA and are ideal for treatment of diving injuries, particularly DCS and AGE. General anesthesia, surgery, chest tube placement, and cardiopulmonary resuscitation have been performed inside multiplace chambers. They have the disadvantage of a risk for DCS to attendants during long treatments.

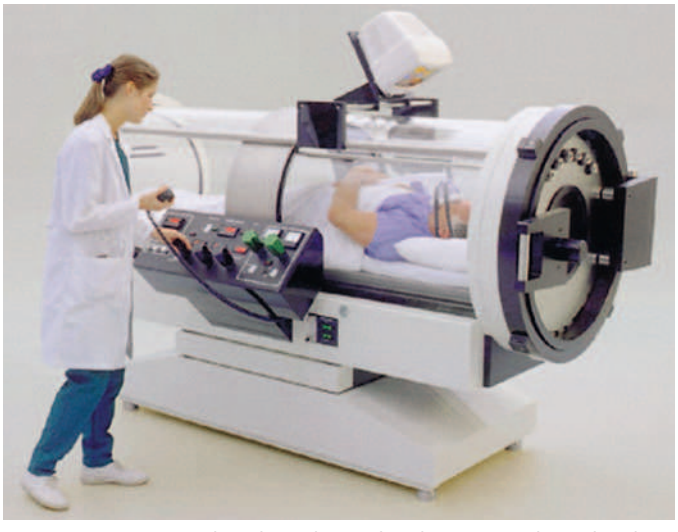

FIGURE 72-2 Monoplace hyperbaric chamber. Monoplace chambers hold a single patient, usually in an environment of pure oxygen. Attendants remain outside the chamber during treatment.

FIGURE 72-3 Multiplace hyperbaric chamber. Multiplace chambers allow more than one patient at a time to receive HBO. Attendants may accompany the patients during treatments. *(Courtesy Duke University Photography, Les Todd.)*

PORTABLE RECOMPRESSION CHAMBERS

Portable monoplace recompression chambers have been developed for use in remote areas for treatment of both diving injuries and high-altitude illnesses.

HYPERBARIC CHAMBERS FOR ALTITUDE ILLNESS

Several portable hyperbaric chambers are available for emergency treatment of severe acute mountain sickness, high-altitude pulmonary edema, and high-altitude cerebral edema, particularly when descent is not feasible or while awaiting evacuation (Figures 72-4 to 72-6). They are constructed of relatively lightweight fabrics and require no use of portable oxygen. The portable chambers are pressurized with ambient air using manual foot pumps or hand pumps, and can simulate a descent of 1500 to 2500 m (5000 to 8200 feet). Hyperbaric treatment should only be used as an emergency measure and is not a substitute for descent or evacuation.

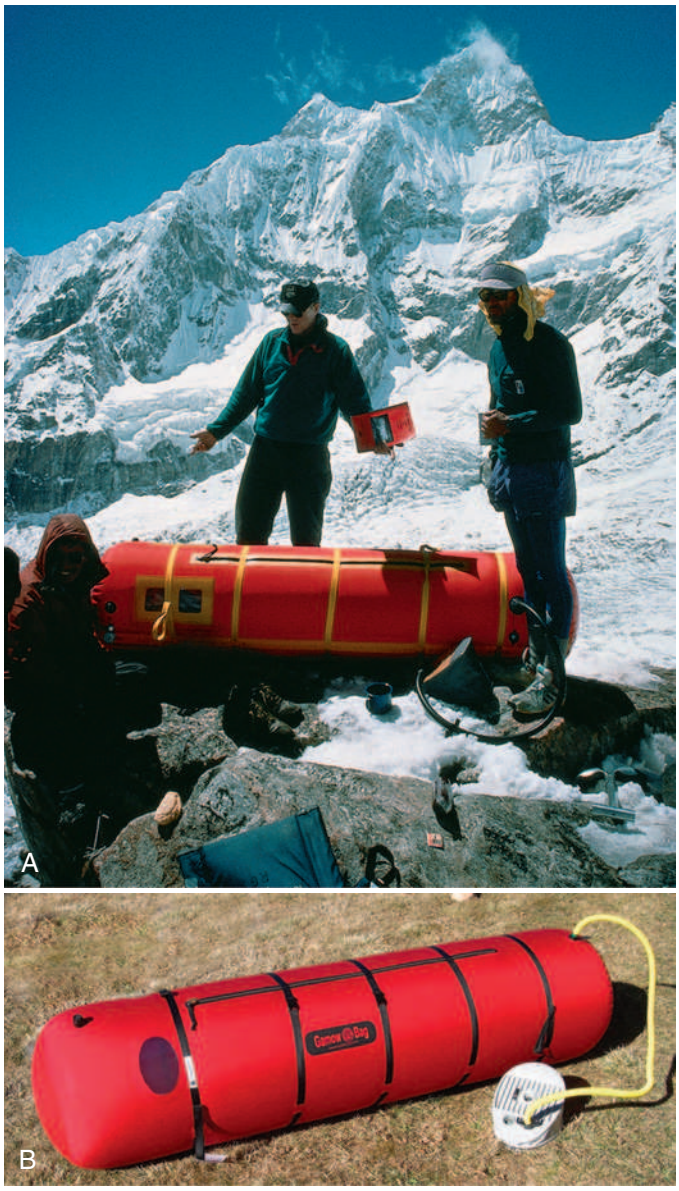

FIGURE 72-4 Chinook Medical Gear's Gamow portable altitude chamber. **A,** In the field. **B,** Close up. *(Courtesy Chinook Medical Gear, Inc.)*

The patient should be told to breathe normally and instructed on equalizing his or her ears by swallowing or by the Valsalva maneuver. The patient is placed inside the portable chamber, zipper pulled shut, and bag inflated with the foot pump to 2 psi (104 mm Hg) above ambient pressure. Two pop-off pressure values are set at 2 psi and prevent overpressurization of the bag. All portable chamber bags must be pumped continually 8 to 12 times per minute in order to flush air through the chamber to prevent carbon dioxide buildup. Patients should be treated for approximately 1 hour and then removed from the bag and reassessed. Additional cycles of descent and reassessment are continued until the patient improves and is able to descend or evacuation is available. Patients with severe high-altitude pulmonary edema may not be able to lie flat; hence, one end of the chamber can be propped up 30 to 40 cm (12 to 16 inches). In severe cases, a patient can breathe oxygen at 4 to 6 L/min during treatment by placing an oxygen bottle inside the chamber.

Different brands of portable chambers are available for use at high altitude. The Gamow bag (Figure 72-4) was invented by Dr. Igor Gamow at the University of Colorado. It is cylindrical, lightweight (the bag, pump, and carrying day pack weigh approximately 7 kg [15 lb]), and easy to use. It measures 2.5 × 0.6 m (8 × 2 feet) and can maintain an internal pressure of 2 psi (104 mm Hg or 0.13 atm). The Certec hyperbaric chamber has two bags: an outside envelope made to withstand tension and provide stability and an inside envelope of polyurethane that allows for more durability of the chamber (Figure 72-5). The Certec chamber weighs 4.8 kg (10.5 lb) and measures 2.2 × 0.65 m (7 × 2 feet); it can be pressurized to a slightly higher inflation pressure (internal pressure of 165 mm Hg or 0.22 atm or 3.2 psi) than can the Gamow bag. Compared with the Gamow

FIGURE 72-5 Certec chamber. *(Courtesy Certec Company.)*

FIGURE 72-6 Treksafe Portable Altitude Chamber. *(Courtesy Treksafe Company.)*

bag, this is equivalent to about 800 m (2625 feet) more of simulated altitude descent; however, it is unclear whether this is clinically relevant. The Portable Altitude Chamber is about the same size as the Gamow bag but allows more room at the head and shoulders and has a radial zipper at the head, allowing easier patient assessment and access (Figure 72-6). All three chambers have been used successfully at altitude; early symptom relief and improvement in peripheral oxygen saturation and cerebral oxygenation have been reported.[7,96] These chambers have no utility for treatment of diving injuries.

The Life Support Technology (LST) Group and the Center for Investigation of Altitude Medicine (CIMA) in Cusco, Peru, have developed a series of acute mountain sickness hyperbaric treatment profiles using standard hyperbaric chambers that can be pressurized to 3 ATA.[37] These profiles provide rapid pressurization to sea level and beyond, with exposure to HBO followed by a gradual decrease in ambient pressure while the patient breathes air. LST and CIMA report that these hyperbaric treatments virtually eliminate the rebound acute mountain sickness often seen with conventional treatment, in which the patient either remains at altitude or is recompressed back to 1500 to 2438 m (4921 to 7999 feet) briefly, and then returns to the same altitude where symptoms were first manifested. These are proposed protocols and are not the standard of care in treating acute mountain sickness.

HYPERBARIC CHAMBERS FOR DIVING INJURIES

Sturdier portable chambers exist for treatment of diving-related injuries. Although there are a number of hard, small recompression chambers used in commercial diving operations, these chambers are not collapsible or easily portable. Two collapsible chambers, used primarily by the U.S. Navy and commercial diving operations, are pressurized with air from a scuba cylinder while the patient breathes 100% oxygen from a face mask. The patient can be transported within the portable chamber to a large recompression facility and moved under pressure into a large multiplace chamber. Unlike the lightweight portable chambers used for treatment of altitude illnesses, these have a working pressure of 3.3 ATA or 48 psi (equivalent of 75 fsw).

The SOS Hyperlite Hyperbaric Stretcher is the most widely used collapsible chamber for treatment and transportation under pressure of a patient suffering from DCS or AGE to a permanent recompression chamber (Figure 72-7). The chamber is made of seamless Vectran-braided tube with coated nylon bladder, has two removable endplates with a communication system, and can deliver 100% oxygen to the patient. It is capable of a standard treatment depth of 3.3 ATA and weighs approximately 50 kg (110 lb). Also called the Emergency Evacuation Hyperbaric Stretcher, or Hyperlite 1, it is certified for use within the U.S. Department of Defense and is currently in use by the U.S. Coast Guard, all four branches of the U.S. military, the National Aeronautics and Space Administration, and the Russian navy. SOS Group is currently developing larger multioccupant collapsible chambers with maximum operating pressures up to 6 ATA.

FIGURE 72-7 The Hyperlite by SOS Limited is pressurized with air from a scuba cylinder. The injured diver breathes 100% oxygen from a separate oxygen bottle. *(Courtesy SOS Group.)*

Another collapsible, portable hyperbaric chamber is the Italian-made GSE chamber. A smaller version, the Hyperbaric Backpack, is also available. This chamber is 76 cm (30 inches) in diameter, 2 m (7 feet) long, and weighs 42 kg (92 lb). It is a double bag of translucent composite polyester and specified for 6 ATA. Another chamber, the Chamberlite 15, is probably the simplest unit on the market and one of the lightest in weight. It is capable of providing 100% oxygen at 2 to 2.4 ATA pressure with special adapters. This unit is constructed of foldable polyurethane, has 10 viewing ports, and weighs less than 18 kg (40 lb), making it highly portable by standard stretcher or two-person carry. The depth of treatment is limited, however, because treatment of diving injuries requires a minimum treatment depth of 2.82 ATA. It has been used in studies on carbon monoxide poisoning and may be a useful tool for emergency treatment of carbon monoxide poisoning in the field.[196]

CONTRAINDICATIONS TO HYPERBARIC OXYGEN THERAPY

ABSOLUTE CONTRAINDICATIONS

HBOT is safe, with very few contraindications. Untreated *pneumothorax* is an absolute contraindication to treatment, because it may progress to a tension pneumothorax during decompression. Once a chest tube has been placed, the patient can safely undergo HBOT. Treatment with bleomycin, disulfiram, and doxorubicin may enhance toxic effects when used concurrently with HBOT; patients taking one of these medications must be reviewed individually for the risks and benefits of HBOT.[109]

RELATIVE CONTRAINDICATIONS

Any condition that interferes with pressure equalization in the sinuses, ears, or lungs (e.g., acute or chronic sinusitis, otitis media, upper respiratory infection) is considered a relative contraindication. Nasal and oral decongestants can be used before treatment; myringotomy or placement of tympanoplasty tubes can facilitate treatment in certain situations. Use of bronchodilators and slow ascent rates can allow individuals with chronic obstructive pulmonary disease to undergo HBOT.

COMPLICATIONS OF HYPERBARIC OXYGEN THERAPY

OXYGEN TOXICITY

In 1878, Bert described seizures and death in animals exposed to 3 to 4 ATA. This central nervous system (CNS) manifestation of oxygen toxicity became known as the *Paul Bert effect*.[19] In 1899, pulmonary oxygen toxicity was described after prolonged exposures to 0.74 to 1.3 atm of oxygen.[125] Tissue injury from oxygen is mediated by reactive oxygen species that are produced by chemical reactions involving single electron transfers to molecular oxygen or its metabolites. These reactive oxygen species include superoxide anions, hydroxyl radials, and hydrogen peroxide.

Under normal oxygen conditions, cells have adequate antioxidant defenses. These defenses include antioxidant enzymes such as superoxide dismutase and catalase, and low-molecular-weight scavengers such as glutathione and vitamin E. As P_{O_2} increases, these defenses are overwhelmed and reactive oxygen species cause lipid peroxidation, protein oxidation, sulfhydryl depletion, and oxidation of pyridine nucleotides, leading to cell membrane disruption, cell injury, and death.[61]

Clark has written a thorough review of the pathophysiology of oxygen toxicity during hyperbaric exposures.[45a]

BOX 72-3 Signs and Symptoms of Central Nervous System Oxygen Toxicity

Diaphoresis	Tinnitus
Bradycardia	Nausea
Palpitations	Vomiting
Euphoria	Vertigo
Apprehension	Lip or facial twitching
Visual fields constriction	Seizures

Central Nervous System Oxygen Toxicity

CNS toxicity occurs during oxygen exposures of 1.4 atm or greater. Symptoms may include dizziness, irritability, facial twitching, tunnel vision, tinnitus, nausea, and seizures (Box 72-3). The incidence of oxygen-induced seizures is 1 to 3/10,000 patients treated at 2 to 2.5 ATA. Susceptibility to oxygen-induced seizures varies among individuals and in any single individual from day to day. Factors that increase susceptibility include exertion, increased partial pressure of carbon dioxide (P_{CO_2}), increased metabolic rate from fever, thyrotoxicosis, adrenal stress, and acute cerebral injury. Catecholamines can lower the seizure threshold and increase the risk for CNS oxygen toxicity. Oxygen-induced seizures are self-limited and resolve quickly after oxygen has been discontinued. No permanent neurologic sequelae have been reported from HBO-induced seizures, and anticonvulsants are not indicated. Intermittent exposure to brief periods of air during HBOT significantly extends the oxygen tolerance and prevents oxygen toxicity. Individuals with a history of seizures or who are taking medications that can lower the seizure threshold can still receive HBOT; oxygen breathing periods are reduced from 30 minutes to 20 minutes as a precaution.

Remove oxygen at the first sign of CNS toxicity (e.g., when feelings of apprehension are noted or when sweating, nausea, or twitching is observed). After all symptoms have resolved, oxygen therapy can be resumed. Shortening the duration of oxygen exposure can prevent the recurrence of symptoms. Treatment for an oxygen-induced seizure involves removing the oxygen mask, maintaining the airway, and preventing self-injury by the patient. Of note, the chamber pressure should be kept constant until the seizure activity ceases to prevent possible pulmonary barotrauma. There is no reason for a person who has had an oxygen-induced seizure not to receive normobaric 100% oxygen if it is medically indicated.

Pulmonary Oxygen Toxicity

Retrolental fibroplasia in premature infants and pulmonary oxygen toxicity in adults are well-known problems associated with the use of therapeutic oxygen. Pulmonary oxygen toxicity is induced by breathing an above-normal but relatively low P_{O_2} for prolonged periods. The limit for indefinite exposure without demonstrable lung damage is considered to be a P_{O_2} of approximately 0.5 ATA. On a time-dose curve, it is considered safe to breathe 100% oxygen at 1 ATA for up to about 20 hours, or at 2 ATA for up to 6 hours. This time can be lengthened significantly by using intermittent exposures, such as interspersing a 5-minute air break between every 20 minutes of oxygen breathing

Symptoms of early pulmonary oxygen toxicity include substernal burning, slight cough, mild dyspnea, and chest tightness, and can be seen in patients treated for DCS, air embolism, and gas gangrene when treatments are prolonged. These symptoms resolve after discontinuing therapy. Pulmonary oxygen toxicity is rare using current treatment tables. Patients do not experience significant alteration in pulmonary gas exchange when treated with standard HBOT protocols.[37a,248]

BAROTRAUMA

The most common side effect of HBOT is middle ear barotrauma caused by inadequate equalization of pressure in the middle ear. This may result from an upper respiratory infection, eustachian tube dysfunction, or inadequate techniques of equalization. Middle ear barotrauma typically occurs during compression, causing pain and hemorrhage within the middle ear, rarely leading to tympanic membrane rupture. It can be prevented by slow compression rates with frequent stops, proper autoinflation techniques, and use of decongestants. For individuals who have significant difficulty clearing their ears, bilateral tympanotomies can be placed. In an emergency, myringotomies can be performed. Sinus barotrauma results from blockage of the sinus ostia due to upper respiratory infection, or allergic sinusitis or rhinitis. Symptoms include pain in the maxillary or frontal sinus. Oral or nasal decongestants before treatment may allow the patient to proceed with HBOT.

CLAUSTROPHOBIA

Some patients may experience claustrophobia or confinement anxiety in both monoplace and multiplace chambers. Benzodiazepines can be given before treatment to help relieve anxiety.

VISUAL REFRACTIVE CHANGES

Patients who undergo more than 20 HBOT treatments often develop progressive myopia that is temporary and reverses completely within a few weeks after discontinuing HBOT. The exact mechanism is not known but appears to be lenticular in origin.[41,128,170] Treatment with HBOT beyond 150 treatments is associated with an increased risk for irreversible refractive changes or development of new cataracts. In the United States, HBOT treatments rarely exceed 60 in number.

PRACTICAL ASPECTS OF HYPERBARIC TREATMENT

EVALUATION OF THE PATIENT FOR HYPERBARIC OXYGEN THERAPY

For elective treatments, patients are assessed for appropriateness of HBOT and contraindications to treatment. Patients should be screened for a history of seizures, pulmonary disease including asthma or chronic obstructive pulmonary disease, implanted devices such as cardiac pacemakers or intrathecal pumps, and claustrophobia. Eustachian tube function and ability to clear the ears can be assessed by examining the tympanic membrane with an otoscope and asking the patient to hold his or her nose and swallow or perform a simple Valsalva maneuver. If the tympanic membrane moves with either maneuver, the eustachian tube is patent.

In emergency situations, patients may be unconscious and thus cannot be screened for contraindications to HBOT. If the patient is not alert, it may be necessary to endotracheally intubate for airway protection; it is safer to sedate combative or semicomatose patients and mechanically ventilate them than to restrain and attempt to control an agitated patient in the chamber. Patients who are sedated and intubated generally do not need myringotomies, because passive inflation of the middle ear space during hyperbaric compression will occur. If necessary, an emergency myringotomy can be performed. Under direct visualization of the tympanic membrane, a perforation is made in the anterior-inferior quadrant of the tympanic membrane with a 21-gauge spinal needle. Patients with diving injuries and carbon monoxide poisoning should be maintained on 100% oxygen by face mask at 10 to 15 L until treatment is initiated in the hyperbaric chamber.

PREPARATION FOR HYPERBARIC TREATMENT

Preparation of a hyperbaric patient requires strict attention to certain safety issues and protocols. For fire safety reasons, patients must wear hospital-supplied clothing of either 100% cotton or a blend of 50/50 polyester-cotton. Undergarments must be 100% cotton. Silk, wool, waffle weave, and nylon and other synthetic materials are prohibited. Watches must be inspected for pressure compatibility. Excessive cosmetics, lotions, and perfumes should be removed. Patients must be inspected for matches, lighters,

BOX 72-4 Guidelines for Equipment Use in Hyperbaric Chambers

Equipment Not Allowed to Be Used in Chamber

Telemetry device
Defibrillator
Brush-driven motor (noninvasive blood pressure machines)
Laser printer (electrocardiogram strips)
Pneumatic stockings
Patient-controlled anesthesia pump
Non–pressure-tested intravenous pump
Transport ventilator
Unnecessary equipment, such as pulse oximetry device
Off-gassing batteries and equipment supplied by > 12 volts
48-watt power source

Equipment Allowed Inside Chamber

Electrocardiogram monitor
Bard Harvard pump
HBO-approved intravenous pump
HBO-approved ventilator
Doppler device
Permanent pacemakers (check with manufacturer for whether they have been pressure tested)
Swan-Ganz and arterial line
Pressure bag
Drains, Foley catheter, nasogastric tube, endotracheal tube cuff
Glass bottles inspected by HBO staff and vented if necessary

HBO, hyperbaric oxygen.

and smoking materials, all of which are prohibited. The hyperbaric staff should follow established protocols at their facility. In general, staff must (1) ensure eustachian tube patency, (2) remove all medical devices not required during hyperbaric treatment, (3) cap all IV catheters that are not in use, (4) empty a Foley bag or have the patient void before chamber entry, (5) have labeled medications available, (6) confirm a good signal on all monitors and check that monitors and IV pumps are fully charged, and (7) ensure that the ventilator is functioning correctly on the surface. A list of equipment that is prohibited and allowed inside a hyperbaric chamber is presented in Box 72-4.

PEDIATRIC CONSIDERATIONS

Generally, indications for HBOT in the pediatric population are similar to those in adult patients. Critically ill pediatric patients can safely receive HBOT when attended by experienced personnel.[104] Accidental carbon monoxide poisoning and serious burns are common afflictions in pediatric age groups. Invasive procedures can result in iatrogenic cerebral gas embolism in children. Figure 72-8 shows a 7-day-old infant with clostridial omphalitis from an umbilical vein catheter that developed into gas gangrene of the abdominal wall. The child was treated successfully with HBOT at the University of California, San Diego, Hyperbaric Center.[179] This center has also successfully treated a child with acute hepatic artery thrombosis after liver transplantation (Figure 72-9).[77]

Special attention must be paid to reduce evaporative, conductive, and convective heat losses in the chamber, because children have an increased risk for hypothermia. An oxygen hood can be used for most children older than 3 to 4 years. In younger children, the neckdam can be pulled down to waist level, which allows the child's hands to be free inside the hood (Figure 72-10). Although there are no studies on the safe limits of exposure to HBO for children, in practical experience there is no increased risk for developing pulmonary or neurologic oxygen toxicity. Additionally, there are no significant differences in side effects or morbidity rates with HBOT for children compared with adults.[186] One theoretical problem with HBOT exists for the child with a ductus arteriosus–dependent congenital heart disorder. The increased P_{O_2} could result in ductal constriction, limiting cardiac output, so this must be considered when treating an infant with congenital heart disease.[234]

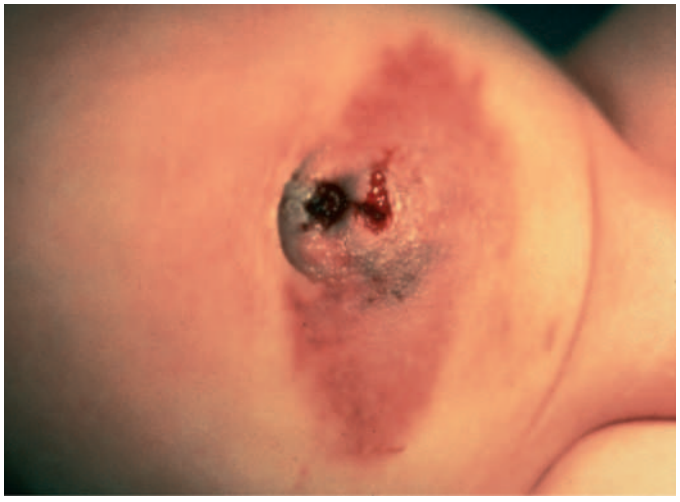

FIGURE 72-8 A 7-day-old infant with gas gangrene of the abdominal wall due to *Clostridium perfringens* treated with HBOT with a good outcome.

APPROVED CLINICAL APPLICATIONS OF HYPERBARIC OXYGEN THERAPY

ARTERIAL GAS EMBOLISM

AGE can result from pulmonary barotrauma while diving (see Chapter 71). Pulmonary barotrauma typically occurs when a diver ascends rapidly while holding the breath because of panic or running out of air. Rapid expansion of gas causes pulmonary overdistention and disruption of the alveolar capillary membrane. Air enters the pulmonary veins, travels to the heart, and enters the systemic circulation. Air embolism may result from causes other than diving, many of which are iatrogenic. Pulmonary barotrauma can occur from blast injuries, mechanical ventilation, penetrating chest trauma, and bronchoscopy. As discussed in Chapter 71, venous gas embolism occurs after scuba diving and in large volumes can enter the arterial circulation via a patent foramen ovale, atrial septal defect, or intrapulmonary arteriovenous anastomosis, or by overwhelming the pulmonary capillary network.

Box 72-5 summarizes the surgical and invasive procedures and nonsurgical causes of gas embolism.[235] Documented AGE, presumably from venous gas embolism, has occurred during

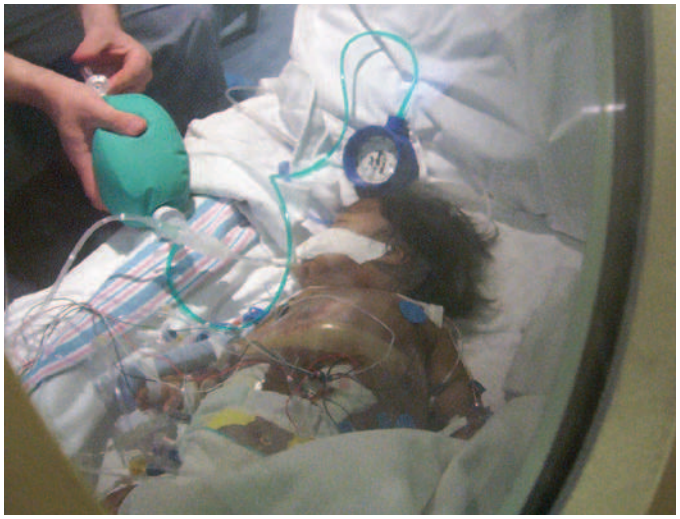

FIGURE 72-9 An 11-month-old boy undergoing HBOT for acute hepatic artery thrombosis after liver transplantation.

FIGURE 72-10 Child being treated in a hyperbaric chamber with the neckdam of the oxygen hood around his waist.

BOX 72-5 Surgical or Invasive Procedures Associated with Gas Embolism

Neurosurgical
Sitting position craniotomies
Posterior fossa procedures
Spinal fusion
Cervical laminectomy

Cardiac
Cardiovascular surgery
Cardiopulmonary bypass grafting
Angioplasty

Pulmonary
Lung biopsy
Thoracentesis

Orthopedic
Total hip arthroplasty
Arthroscopy

Gastrointestinal
Laparoscopy
Laparoscopic cholecystectomy
Retrograde cholangiopancreatography
Orthotopic liver transplantation
Percutaneous hepatic puncture

Gynecologic
Therapeutic abortion
Hysteroscopy
Cesarean delivery

Urologic
Transurethral prostatectomy

From Van Hoesen K, Neuman TS: Gas embolism: venous and arterial gas embolism. In Neuman TS, Thom SR, editors: Physiology and medicine of hyperbaric oxygen therapy, Philadelphia, 2008, Saunders/Elsevier, p 257.

central venous catheterization, mechanical ventilation, cardiopulmonary bypass, angioplasty, lung biopsy, percutaneous hepatic puncture, liver transplant, hemodialysis, gastrointestinal endoscopy, hydrogen peroxide irrigation and ingestion, spine surgery, oral sex, sexual intercourse after childbirth, transurethral prostatectomy, laparoscopy, arthroscopy, hysteroscopy, and cesarean delivery.* Abnormal neurologic findings after cardiovascular or neurologic surgery or dialysis should make one consider AGE.

*References 8, 9, 18, 46, 49, 57, 89, 101, 108, 141, 152, 155, 226, 231, 233, 240, 250.

AGE manifests with a sudden alteration in consciousness, confusion, focal neurologic deficits, cardiac arrhythmias, and death. AGE requires immediate recompression. HBOT reduces the volume of gas bubbles, thereby restoring the blood flow, and improves oxygen delivery to ischemic, hypoperfused tissues. HBOT reduces cerebral edema and decreases ischemia-reperfusion injury. Treatment for AGE had traditionally been done according to U.S. Navy Table 6A, with an initial excursion to 165 feet (6 ATA) for 30 minutes to enhance bubble compression (Figure 72-11). Animal studies showed no additional benefit from compression to 6 ATA compared with 2.82 ATA.[120] Current recommendations include initial recompression to 2.82 ATA

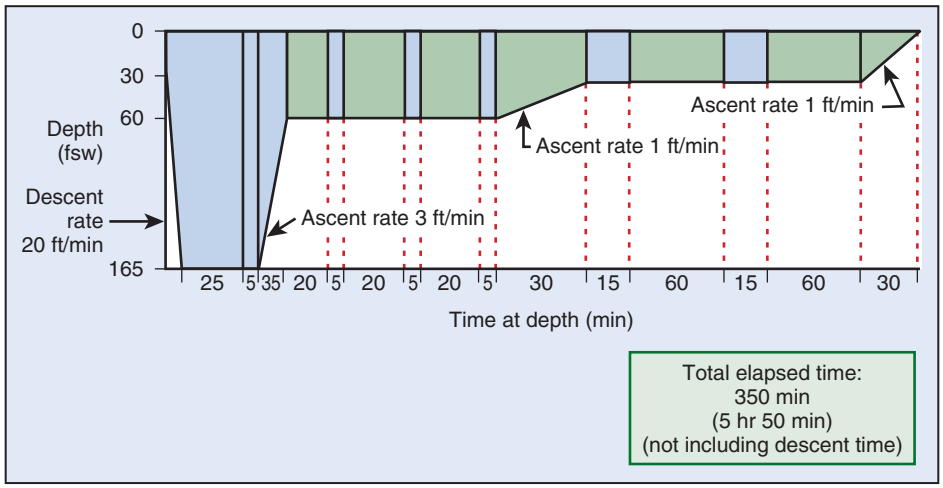

FIGURE 72-11 U.S. Navy Treatment Table 6A. fsw, feet of seawater. *(Redrawn from* U.S. Navy Diving Manual.*)*

while breathing 100% oxygen. If there is no improvement or clinical deterioration, deeper recompression to 6 ATA can be instituted. The diver breathes a mixture of 50% nitrogen and 50% oxygen below 2.82 ATA. Repetitive treatments are recommended until there is no further stepwise improvement.[149]

The Divers Alert Network has an on-call diving physician available 24 hours a day, 7 days a week, who can assist in triage and arrangement of transport and treatment for all diving injuries. In the United States, the network can be reached at 919-684-9111.

DECOMPRESSION SICKNESS

Decompression sickness (DCS) results from generation of bubbles of inert gas in the tissues and blood that interfere with organ function by occluding blood flow and other biochemical events (see Chapter 71). DCS may result from rapid ascent from diving, flying after diving, ascent in an unpressurized aircraft, or exposure in a hyperbaric or hypobaric chamber. Definitive treatment of DCS is immediate recompression and HBOT. HBOT causes immediate reduction in bubble volume, increase in the diffusion gradient for inert gas from the bubble into surrounding tissues, oxygenation of ischemic tissue, and reduction in cerebral edema. Other beneficial effects of HBOT that probably play a role in treating DCS are reduction in neutrophil adhesion to the capillary endothelium and prevention of ischemia-reperfusion injury.[131]

Various hyperbaric treatment regimens for DCS have been described. However, there are no human prospective, randomized studies for treatment of DCS. In general, U.S. Navy Table 6 has become the standard of treatment for pain-only and neurologic DCS.[214] Occasionally, U.S. Navy Table 5 is still used for pain-only DCS, but many diving physicians have discontinued its use.[75] U.S. Navy Tables 5 and 6 are shown in Chapter 71, Figure 71-14. These tables consist of compression to 60 fsw (2.8 ATA), initially with intermittent oxygen breathing. Theoretical advantages of using helium and oxygen in the Comex 30 table have been reported.[111] Longer oxygen treatment tables exist that feature extended decompression profiles for refractory cases of DCS involving the CNS.

The choice of treatment table and number of treatments required depend on the clinical severity of illness, response to treatment, and residual symptoms after initial recompression. If the delay to treatment is not excessive, the majority of patients with DCS have complete resolution of symptoms after a single hyperbaric treatment. Patients with severe DCS involving the spinal cord or a prolonged delay to recompression may have incomplete resolution of symptoms and often require repetitive treatments. Although delay to treatment worsens the outcome for severely injured divers, currently available data have not established a maximum time after which recompression is ineffective.[4,53] In one series of 76 divers, even despite a delay of 48 hours or longer for HBOT, 76% of divers with DCS had full recovery.[79] Patients with DCS should continue treatments until symptoms have completely resolved or until they show no further improvement in response to two consecutive treatments. Most individuals need no more than 5 to 10 repetitive treatments. Repetitive treatments can be given daily or twice daily as described in U.S. Navy Table 5, 6, or 9 (2-hour treatment at 2.4 ATA). A small number of patients have residual neurologic deficits that may either be permanent or improve gradually over 6 to 12 months.

ARTERIAL INSUFFICIENCIES

Central Retinal Artery Occlusion

Central retinal artery occlusion (CRAO) is an emergency condition of the eye that presents as sudden painless vision loss. Causes of CRAO include atherosclerosis-related thrombus, embolism, vasospasm, and giant cell arteritis. The prognosis is poor and patients are often left with permanent vision loss. The central retinal artery enters the globe within the substance of the optic nerve and serves the inner layers of the retina. The long posterior ciliary arteries supply blood to the choroid and outer layers of the retina. In CRAO, retinal arterial flow is interrupted and the inner retinal layers become ischemic. Eventually, recanalization occurs and blood flow is reestablished. The retina typically has suffered significant ischemia and cell death, however, so vision remains impaired. Conventional therapy for CRAO includes ocular massage, anterior chamber paracentesis, pentoxifylline, and oral diuretics; however, these modalities rarely restore vision.

Patients with CRAO present with sudden painless loss of vision, resulting in a clinical assessment of "light perception" to "counting fingers." Complete loss of vision with no light perception is more indicative of ophthalmic artery occlusion, with no blood flow in the choroidal vessels. In 15% to 30% of individuals, a cilioretinal artery is present that supplies the area around the macula; hence, central vision may be preserved in these individuals with CRAO. Ocular findings in patients with CRAO may include an afferent papillary defect and "boxcarring" of arterioles. The retina typically appears pale yellow or white due to ischemia; a cherry red spot may develop in the macula.

In cases of elevated partial pressures of oxygen, the choroidal circulation may supply enough oxygen to the inner retinal layers by diffusion to maintain viability of the inner retina.[121] Supplemental oxygen at normobaric pressures has been successful at reversing retinal ischemia in CRAO.[36,56,204] HBO has also been successful in restoring vision in cases of CRAO if initiated within 24 hours or less of onset of vision loss.[14,121,145,175] Although there are reports of patients responding to HBOT after 24 hours, the majority do not have return of vision if therapy is initiated after a delay of 24 or more hours. Murphy-Lavoie and colleagues[157] summarized 29 studies in the literature of retinal artery occlusion and HBOT and found that overall, 65% of cases showed improvement when treated with HBO.

Patients with signs and symptoms consistent with CRAO with onset within 24 hours should be placed on 100% oxygen at 1 ATA immediately. If vision improves significantly with normobaric oxygen within 15 minutes, the patient should be admitted and given intermittent normobaric oxygen for 15 minutes every hour. If there is no response within 15 minutes, HBOT should be initiated. Adjunctive therapies to lower intraocular pressure or cause retinal vasodilation can be started while HBOT is being arranged. Recommended protocols for HBOT range from 2 to 2.8 ATA for 90 minutes twice daily for a minimum of 3 days.[156] U.S. Navy Table 6 has also been considered for treatment if there is no improvement at 2.8 ATA after 20 minutes. HBOT should be continued twice daily until there is no further visual improvement after 3 consecutive days. If a patient has return of vision during HBOT, inpatient monitoring and intermittent supplemental oxygen should be considered.[157]

Enhancement of Healing in Selected Problem Wounds

Problem or chronic wounds are usually present in a compromised host and fail to respond to medical and surgical management. The incidence and prevalence of these wounds are increasing. Such wounds include venous leg ulcers, pressure ulcers, arterial ulcers, and foot ulcers in patients with diabetes or significant vascular disease. Numerous factors impair wound healing in diabetic foot ulcers, including impaired autonomic responses, neuropathy leading to trauma, impaired microvascular perfusion, local tissue hypoxia, and increased rates of wound infection. Using HBOT to restore tissue P_{O_2} into the normal range can stimulate fibroblast proliferation, collagen synthesis, neutrophil oxidative killing of microorganisms, and angiogenesis.

The most common problem wounds treated with HBOT are diabetic foot wounds. Liu and associates[124] published a systematic review of the effectiveness of HBOT for chronic diabetic foot ulcers that evaluated 13 trials, including 7 prospective randomized trials. HBOT improved the rate of healing and lowered the risk of major infection in patients with diabetic foot ulcers. A 2012 systematic review showed similar results.[69] In 2014, Worth and associates analyzed 17 published studies (clinical trials, retrospective case series, and comparative case series) showing positive responses with HBOT.[253] They also reviewed arterial insufficiency ulcers, venous stasis ulcers, and pressure ulcers. HBOT is not indicated in routine pressure ulcer management or for primary management of venous stasis ulcers. Primary

treatment of arterial insufficiency ulcers is to improve blood flow using angioplasty, surgical revascularization, or other interventional techniques.

Other treatments to hasten recovery include negative pressure wound therapy (wound vacuum-assisted closure), bioengineered tissue grafts, topical debriding agents, platelet-derived growth factor, and surgical closure in combination with HBOT. Not all wounds should be treated with HBOT. For example, HBOT is not a substitute for surgical revascularization in arterial insufficiency, for which a comprehensive, multidisciplinary approach is advised.

HBOT is administered at 2 to 2.5 ATA for 90 to 120 minutes once or twice daily. Patients hospitalized with limb-threatening infections or significant peripheral arterial occlusive disease may require twice-daily treatments until stabilized. Many of these wounds are slow to respond and may require 30 to 60 HBO treatments. Appropriate wound care is extremely important to facilitate healing, and many hyperbaric chambers are associated with wound care centers.

Figure 72-12 shows a 58-year-old diabetic man with Wagner grade 3 plantar foot wound with necrosis and cellulitis. He refused amputation and was treated with 30 hyperbaric treatments. He responded with granulation of the wound tissues and eventual epithelialization and closure.

CARBON MONOXIDE AND CYANIDE POISONING

Carbon monoxide poisoning is a significant health problem in the United States, accounting for more than 50,000 emergency department visits annually. Poisonings occur primarily from fire smoke, exhaust from internal combustion engines, and inhalation of fumes from faulty gas furnaces. Carbon monoxide poisoning has also occurred in recreational boaters and campers who use internal combustion engines and fossil fuel heaters in enclosed spaces.

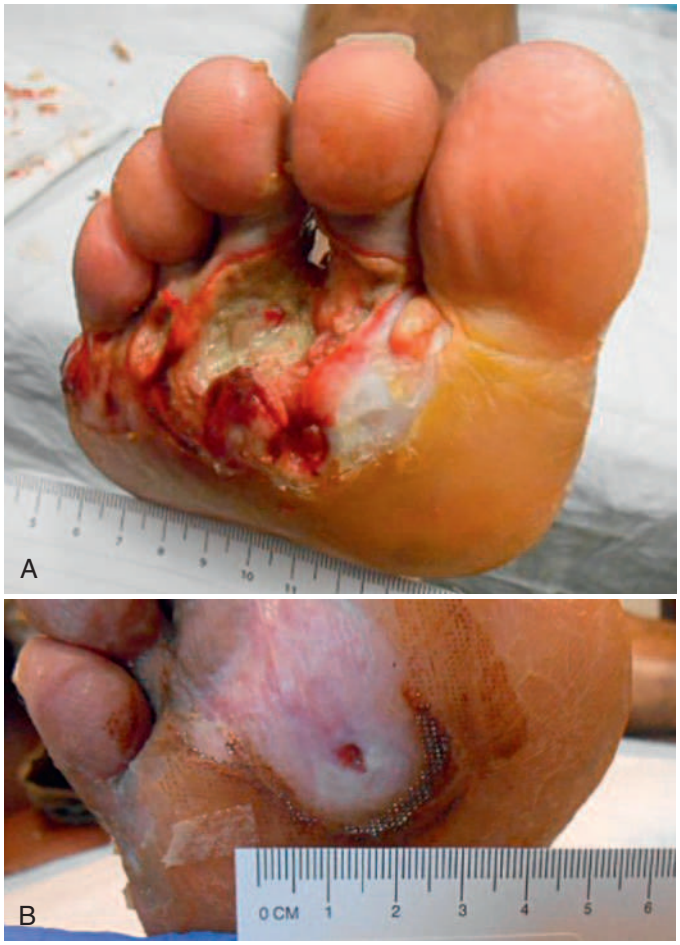

FIGURE 72-12 Diabetic foot wound. **A,** A 58-year-old diabetic man with Wagner grade 3 plantar foot wound. **B,** After 30 HBO treatments, he had almost complete closure of his wound, eliminating the need for amputation. *(Courtesy of Caesar Anderson, MD).*

Carbon monoxide toxicity is mediated by a number of mechanisms that lead to hypoxic stress. Carbon monoxide binds to hemoglobin with an affinity 200 times that of oxygen, resulting in a shift of the hemoglobin-oxygen dissociation curve to the left and tissue hypoxia. Additionally, carbon monoxide binds to myoglobin and cytochrome oxidase, interfering with intracellular respiration and increasing oxidative stress, both of which contribute to cell death. Carbon monoxide causes the production of reactive oxygen species, leading to neuronal necrosis and apoptosis.[177] In addition to hypoxic stress, carbon monoxide poisoning leads to a complex cascade of biochemical events involving inflammatory and immunologic processes.[215,216,218,219,222] A poor correlation exists between tissue hypoxia and blood carboxyhemoglobin levels.

The most common signs and symptoms of carbon monoxide poisoning are headache, nausea, vomiting, dizziness, malaise, and confusion. Chest pain and shortness of breath may occur. Severe exposures result in loss of consciousness and death.[82,245] Carbon monoxide poisoning can result in delayed neurologic sequelae, characterized by deterioration in cognitive and motor function that appears days to weeks after the initial insult. Delayed neurologic sequelae are reported in up to 46% of acute carbon monoxide poisoning victims.[223,247]

Supplemental oxygen is the standard treatment for carbon monoxide poisoning. Oxygen hastens dissociation of carbon monoxide from hemoglobin and provides enhanced tissue oxygenation. Use of HBOT for treatment of carbon monoxide poisoning is based on the following mechanisms:

- HBOT accelerates the rate of carbon monoxide dissociation from hemoglobin; the carboxyhemoglobin half-life is decreased from 5.5 hours breathing air to 23 minutes breathing 100% oxygen at 3 ATA.[29,169]
- HBOT accelerates dissociation of carbon monoxide from cytochrome oxidase, thereby improving oxidative phosphorylation at 3 ATA.[31]
- HBOT prevents carbon monoxide–mediated brain lipid peroxidation and leukocyte-mediated inflammatory changes in the brain by inhibition of leukocyte beta$_2$ interferons.[215,216]
- HBOT reduces brain inflammation and improves mitochondrial oxidative processes.[218]
- HBOT decreases cerebral edema and maintains adequate cerebral oxygen delivery.
- HBOT reduces necrosis and protects against accelerated apoptosis.[33]

Animal models of carbon monoxide poisoning and HBOT demonstrate more rapid improvement in cardiovascular status, lower mortality rates, and a lower incidence of neurologic sequelae.[100,171,227]

HBOT is recommended for patients with a carboxyhemoglobin level greater than 25% or with signs of serious carbon monoxide poisoning, including prolonged unconsciousness, neurologic symptoms, cardiovascular dysfunction, or severe acidosis, irrespective of the carboxyhemoglobin level. HBOT should be used more liberally in pregnant patients because of enhanced fetal vulnerability to carbon monoxide and hypoxia.[234] Controversy exists surrounding HBOT for mild to moderate cases of carbon monoxide poisoning and whether HBOT can prevent delayed neurologic sequelae. Six randomized, controlled clinical trials during acute carbon monoxide poisoning had conflicting results; however, the studies varied in quality, study design, and outcomes. The studies that showed no benefit from HBOT had long treatment delays and poor follow-up.[55,135,180,189,223,247] The largest randomized clinical trial with the best study design and follow-up demonstrated a significant benefit of HBOT in decreasing the incidence of delayed neurologic sequelae.[247] A recent review article by Hampson and colleagues[84] discusses practice recommendations in acute carbon monoxide poisoning, including recommendations for HBOT. The recommended treatment protocol for carbon monoxide poisoning is 2.4 to 3 ATA for 90

to 120 minutes[247] for a total of three treatments at 6- to 12-hour intervals.[83]

Using HBOT to treat cyanide poisoning is less clear. Hydrogen cyanide often accompanies carbon monoxide poisoning; the toxic effects of cyanide and carbon monoxide are synergistic.[165] Treatment for hydrogen cyanide poisoning includes hydroxocobalamin with or without sodium thiosulfate. Amyl nitrite and sodium nitrite induce methemoglobin to bind hydrogen cyanide, which can further impair the oxygen-carrying capacity in the presence of carbon monoxide. HBO may directly reduce the toxicity of cyanide and maintain adequate oxygen delivery to the brain during the peak insult from hydrogen cyanide. In recent work in animal models after cyanide poisoning, HBO was beneficial in increasing cerebral tissue oxygen partial pressures and in reducing respiratory distress and cyanosis.[118] There are no controlled clinical trials of HBOT for pure cyanide poisoning or carbon monoxide poisoning complicated by cyanide. The use of HBOT is supported only by anecdotal cases; the clinical literature is limited.[25,71,117,123,190]

CLOSTRIDIAL MYONECROSIS (GAS GANGRENE)

Invasive clostridial infection usually results from injury or contamination of wounds. Clostridial myositis accompanied by myonecrosis (gas gangrene) is an acute, rapidly progressive invasive infection of muscles characterized by toxemia, edema, tissue death, and gas production. *Clostridium* is an anaerobic, spore-forming, gram-positive encapsulated bacillus. The most common species isolated is *C. perfringens* (isolated in 80% to 90% of wounds); others include *C. novyi, C. septicum,* and *C. histolyticum. C. perfringens* is a facultative anaerobe that can multiply freely in tissues with an oxygen tension of 30 mm Hg and demonstrates restricted growth at oxygen tensions of 70 mm Hg. Although more than 20 toxins are produced, the most prevalent and lethal is alpha-toxin, which causes hemolysis and tissue necrosis. HBOT does not kill clostridial organisms; it has a bacteriostatic effect and at oxygen tensions of 250 mm Hg inhibits alpha-toxin production.[237]

Treatment for clostridial infections consists of surgical debridement, antibiotics, and HBOT as soon as possible to stop alpha-toxin production and to save potentially viable tissue. Multiple retrospective clinical studies report improved overall survival rates and a decreased rate of amputation. The lowest rates of morbidity and mortality are achieved with initial conservative surgery and rapid initiation of HBOT.[5,73,90,112,173,181]* This approach not only saves lives but also saves limbs and tissues because no major amputations or excisions are done prematurely and demarcation of dead tissue is allowed.

HBOT for gas gangrene should be given at 3 ATA every 8 hours for the first 24 hours and then twice per day at 2.4 to 3 ATA for 2 to 5 days. When daily assessment reveals no further evidence of ongoing tissue necrosis, HBOT can be stopped. Figure 72-13 shows a young female who fell while riding her horse and sustained gas gangrene due to *C. perfringens.* After aggressive surgical debridement, antibiotics, and HBOT, a graft was placed to cover her wound, and she made an excellent recovery.

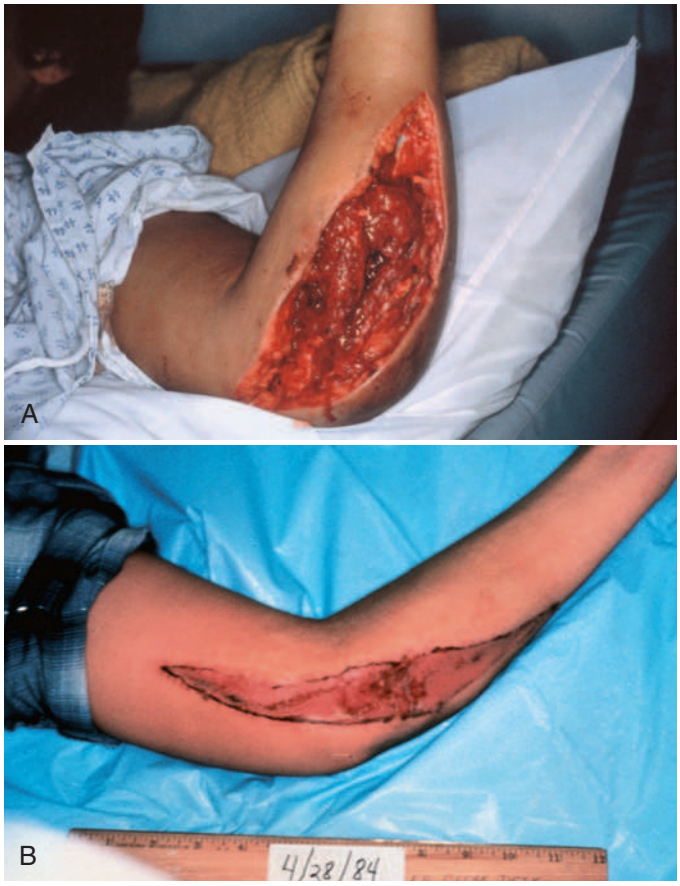

FIGURE 72-13 Gas gangrene due to *Clostridium perfringens.* **A,** The patient presented with myonecrosis from gas gangrene and was aggressively treated with surgical debridement, antibiotics, and HBOT. **B,** Once her wound was stabilized, a graft was placed to cover the wound.

COMPROMISED GRAFTS AND FLAPS

HBOT is extremely useful in flap salvage when tissue is compromised by decreased perfusion, irradiation, or hypoxia. HBOT can help maximize viability of compromised grafts and flaps by improving tissue oxygenation and increasing flap capillary density. HBOT enhances graft and flap survival by decreasing the hypoxic insult, enhancing fibroblast function and collagen synthesis, stimulating angiogenesis, and inhibiting ischemia-reperfusion injury. Since 1966, experimental animal studies have shown improved skin flap survival in animals treated with HBOT compared with controls.[11,142,160,191] In an ischemic flap model in rats, HBOT increased microvascular blood flow during reperfusion, compared with untreated ischemic controls, by reducing neutrophil endothelial adherence in venules and blocking progressive arteriolar vasoconstriction associated with reperfusion injury.[259] Flap survival with HBOT averaged 90%, compared with other studies with failure rates as high as 67% in compromised tissues.[27,172,244] Baynosa and Zamboni[11,12] published extensive critical reviews of HBOT and its applications to different types of compromised flaps and grafts in both animal and clinical human studies that demonstrates the benefit of adjunctive HBOT for multiple types of grafts and flaps with various causes of compromise. Prompt initiation of HBOT as soon as flap or graft compromise is identified maximizes tissue viability and ultimately graft or flap salvage.

HBOT is most effective when started as soon as signs of flap compromise appear. Initial treatments should be twice daily at a pressure of 2 to 2.5 ATA for 90 to 120 minutes. Once the graft or flap appears more viable, HBOT can be given once a day until the flap appears stable. The average number of treatments varies from 10 to 20. In patients who have had previous graft or flap failure, 20 treatments prior to flap placement can prepare the site with granulation tissue, followed by 10 to 20 treatments after flap or graft placement to ensure tissue survival. Figure 72-14 shows a dusky, ischemic flap after surgery; it improved dramatically with a single HBOT treatment.

CRUSH INJURIES AND SKELETAL MUSCLE COMPARTMENT SYNDROMES

HBOT is a therapeutic adjunct to treatment of crush injury and skeletal muscle compartment syndrome. Posttraumatic cytogenic and vasogenic edema reduce tissue oxygenation, contributing to hypoxia and ischemia. There may be secondary tissue destruction caused by reperfusion injury. HBOT (1) increases tissue oxygen tension in hypoxic tissue during the early postinjury period when oxygen demand is greatest; (2) causes vasoconstriction

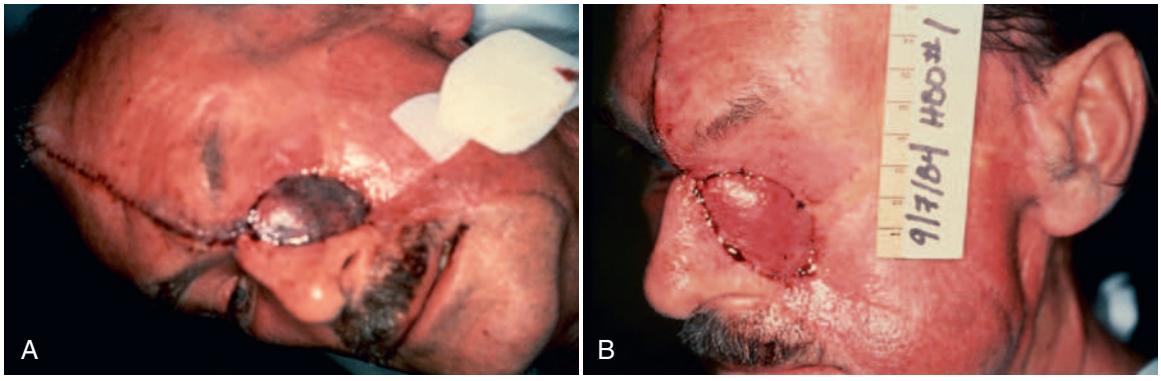

FIGURE 72-14 HBOT and compromised flap. **A,** Shortly after surgery, the flap appeared dusky and ischemic. **B,** The patient was treated with one round of HBOT and had immediate improvement in color and blood flow to the wound.

and edema reduction while maintaining hyperoxygenation; (3) improves blood flow in the microcirculation by decreasing interstitial fluid pressures attributed to edema reduction; and (4) mitigates reperfusion injury that accompanies crush injuries and skeletal muscle compartment syndrome.

Numerous animal studies and clinical series support use of HBOT for crush injuries and compartment syndrome.[51,70,166,207,208,259] A randomized, double-blinded clinical trial in crush injuries showed a statistically significant 94% healing rate in the HBOT group compared with 59% in controls.[26] A complete summary of the supporting literature and an evidence-based review is provided by Strauss.[206]

Early initiation of HBOT within 4 to 6 hours of injury or ischemia is essential. HBOT is administered at 2 to 3 ATA for 90 to 120 minutes. The number of treatments depends on the pathophysiology. Patients with crush injuries require HBOT three times a day for the first 2 days, two times a day for the next 2 days, and daily for an additional 2 days. Suspected reperfusion injuries after reimplantations, placement of free flaps, or transient ischemia only require one or two treatments. Signs of impending compartment syndrome include increasing pain, particularly with passive stretch, a tense compartment, and hyperesthesia or weakness in the distal extremity. If compartment pressures indicate that fasciotomy is not required, HBOT should be given three times within 24 hours. If clinical indications and compartment pressure measurements dictate immediate fasciotomy, HBOT can be used after surgery to reduce morbidity from residual injury due to swelling and ischemia.

DELAYED RADIATION INJURIES (SOFT TISSUE AND BONY NECROSIS)

Radiation kills normal tissue and causes loss of vascularity and cellularity. Delayed radiation complications are seen after a latent period of 6 months or more and may develop many years after exposure. Radiation leads to progressive obliterative endarteritis with resultant tissue ischemia and fibrosis. Dental extractions and other surgical procedures are associated with high complication rates when performed on heavily irradiated tissues.

HBOT stimulates fibroplasia, angiogenesis, and increased cellularity in radiated tissue by mechanisms similar to those described above for wound healing.[150] HBO can correct diminished stem and progenitor cell mobilization caused by irradiation and chemotherapy.[72] HBOT has been used prophylactically before oral surgery in a radiated tissue field and to treat delayed radiation tissue injury. HBOT has been used successfully for many years in treatment and prevention of mandibular osteoradionecrosis.

From randomized, prospective trials of bone grafts to irradiated tissue in humans, a standard protocol has been developed for use of HBOT when surgery is performed in irradiated tissue.[132,133] This protocol of 20 to 30 preoperative HBOT treatments followed by 10 postoperative treatments has been shown to be effective for head and neck therapy after radiation surgery, including bone graft reconstruction, soft tissue vascular flaps, and tooth extraction. In a recent review by Hampson and associates,[82] 73% of 43 patients treated for mandibular necrosis showed complete resolution and 21% had significant improvement. HBOT is also beneficial for prophylaxis of osteoradionecrosis. Figure 72-15 demonstrates improvement in osteoradionecrosis with use of HBOT.

This clinical success has led scientists to apply HBOT to radiation injuries at other sites. HBOT has been shown to be beneficial in other forms of soft tissue radionecrosis, including radiation proctitis, enteritis, and cystitis.[20,28,40,59,84] Additionally, HBOT is useful in treatment of laryngeal and tracheal radionecrosis, and for soft tissue necrosis of the head, neck, and chest wall, the latter particularly after mastectomy.[38,62] A more recent application has been use of HBOT to treat neurologic injuries secondary to radiation, including transverse myelitis, brain necrosis, optic neuritis, and brachial and sacral plexopathy.[58] Although evidence of

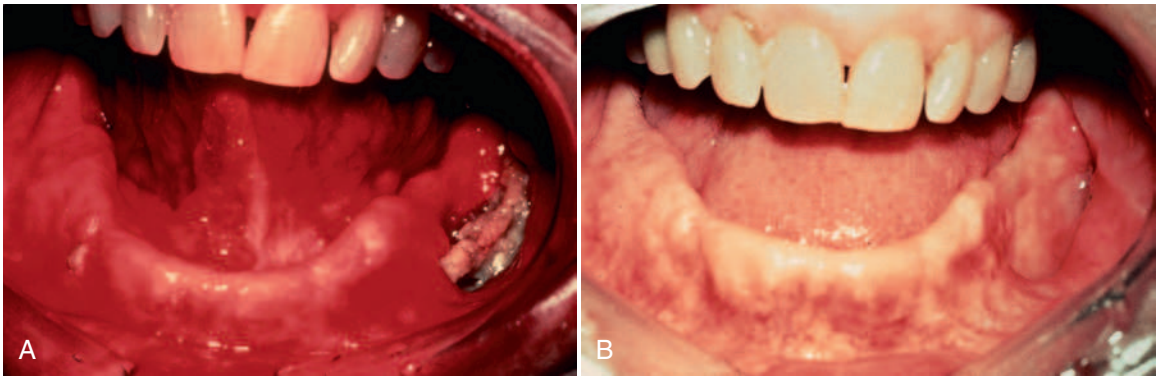

FIGURE 72-15 Patient with osteoradionecrosis of the mandible before **(A)** and after **(B)** HBOT.

beneficial HBOT for radiation-induced neurologic injuries is anecdotal, because of the severe consequences of injury to the CNS, HBOT should be given serious consideration.

HBOT for radiation injury is administered at 2 to 2.5 ATA daily for 20 to 40 treatments. Some patients benefit from additional treatments, so utilization review should occur at 60 treatments. To prevent osteoradionecrosis in patients who require extraction of teeth in previously irradiated jaws, the patients should receive 20 treatments before extraction, followed by 10 treatments after extraction. If osteoradionecrosis is already present, the treatment protocol changes to 30 preextraction treatments, or surgical resection followed by 10 postresection treatments.

A frequent concern of practitioners referring patients for HBOT with radiation injury is the fear that HBOT will accelerate malignant cancer growth or cause a dormant malignant tumor to be reactivated. Feldmeier[58] extensively reviewed this subject and found that both clinical reports and animal studies show no enhancement of cancer growth or recurrence rates.

IDIOPATHIC SUDDEN SENSORINEURAL HEARING LOSS

Idiopathic sudden sensorineural hearing loss is loss of at least 30 dB over at least three contiguous frequencies within 3 days. In addition to sudden unilateral hearing loss, individuals present with tinnitus, vertigo, and a sensation of aural fullness. Although the cause of this type of hearing loss is unclear, possible pathophysiologic mechanisms include vascular occlusion, cochlear membrane damage, ischemia, viral infections, labyrinthine membrane breaks, diseases associated with immunity, and trauma. Perilymph oxygen tension decreases significantly with sudden sensorineural hearing loss. HBOT increases the perilymph PO_2 by 9.4-fold.[114] Additional benefits of HBOT in the treatment of idiopathic sudden sensorineural hearing loss may include antiinflammatory effects, blunting of ischemia-reperfusion injury, and edema reduction.

Murphy-Lavoie and colleagues[158] and Piper and associates[178] have published the most comprehensive reviews of HBOT treatment efficacy for idiopathic sudden sensorineural hearing loss. There are more than 100 publications evaluating the use of HBOT for the condition, including 8 randomized controlled trials. The majority of these studies have shown that HBOT has significant efficacy as an adjunct to medical therapy.

The recommended treatment is 100% oxygen at 2 to 2.5 ATA for 90 minutes daily for 10 to 20 treatments. The best outcomes occur by combining oral corticosteroids with HBOT, particularly in patients with profound hearing loss treated within 2 weeks from symptom onset.

INTRACRANIAL ABSCESS

HBOT is an adjunct to treatment for intracranial abscess. The overall mortality rate for intracranial abscess is 2.7% to 25% in patients with underlying immune deficiency or neoplasm.[254] In certain patients with complications or in patients who pose therapeutic problems, HBOT has been beneficial when used as an adjunct. Over 109 cases of intracranial abscess treated with adjunctive HBO have been reported in the literature, with an overall mortality rate of 0% to 3.4%.[115,136,211]

Proposed mechanisms of HBOT in intracranial abscess include reduction in brain swelling, inhibition of anaerobic organisms found in the abscess, enhancement of neutrophil-mediated phagocytosis of infecting organisms, and treatment of concomitant skull osteomyelitis. HBOT has been recommended as a complement to currently accepted standard procedures in patients with multiple abscesses or abscesses in a deep or dominant location; in a compromised host with an abscess; in patients who are poor surgical candidates or for whom surgery is contraindicated; or in patients in whom there has been no response to standard surgical and antibiotic treatment or who have deteriorated further after undergoing standard treatment.[6] Because of significant mortality rates and long-term sequelae, it is unlikely that a rigid, human, double-blind controlled study can be done.

For the treatment of intracranial abscess, HBOT is administered at 2 to 2.5 ATA, with 60 to 90 minutes of oxygen administration per treatment. Initially, HBOT should be administered twice daily depending on the condition of the patient. The optimal number of treatments is unknown; treatments should be based on the patient's clinical response and radiologic findings. In reported series, the average number of HBOT sessions was 14. Utilization review is recommended after 20 treatments.

NECROTIZING SOFT TISSUE INFECTIONS

HBOT is recommended as an adjunct to surgical debridements, antibiotic treatment, and goal-directed critical care therapy for necrotizing soft tissue infections (including crepitant anaerobic cellulitis, progressive bacterial gangrene, necrotizing fasciitis, Fournier's gangrene, nonclostridial myonecrosis, and zygomycotic gangrenous cellulitis), particularly for compromised hosts when the mortality rate is expected to be high. Detailed descriptions of each of these infections are beyond the scope of this chapter, but can be found in a recent summary.[97] These infections are typically mixed aerobic-anaerobic infections of subcutaneous tissues, fascia, and muscle. HBOT adversely affects anaerobic bacterial growth and improves neutrophil function. The hyperoxygenated tissue zone surrounding the infected area may be of significance in preventing extension of invading microorganisms.

Studies continue to demonstrate the beneficial effect of HBOT in management of necrotizing soft tissue infections. Patients treated with HBOT show a statistically significant lower mortality rate, decreased incidence of amputation, and improved long-term outcome.[47,97,112,181,183,194,200,249] There are no randomized, double-blinded, controlled clinical trials comparing the outcomes of patients treated with HBOT with patients not treated with it, because it would be unethical to perform such trials when morbidity and mortality rates have improved in patients treated with HBOT compared with those not treated with HBOT before it became available. HBOT can be both cost-effective and saving of life and limb, particularly for the sickest patients.

HBOT for necrotizing fasciitis is given at 2.4 to 3 ATA for 90 to 120 minutes twice daily until the patient has stabilized and infection is controlled. If the infection is severe, the recommendation is to give 3 ATA three times in the first 24 hours, followed by twice-daily treatments for up to 30 treatments.

REFRACTORY OSTEOMYELITIS

Refractory osteomyelitis recurs or persists after standard therapy with aggressive surgical debridement and antibiotics. HBOT can elevate tissue oxygen tension in infected bone to normal or above-normal levels.[130] Osteoclast function of removing necrotic bone is an oxygen-dependent process; osteoclasts do not function properly in hypoxic bone. HBOT has a stimulatory effect on osteoclast function.[144,150] Experimental animal models have demonstrated the benefit of HBO for refractory cases of osteomyelitis.[144,229] No prospective, randomized clinical human trial examining the effect of HBOT on refractory osteomyelitis exists, however. The overwhelming majority of published animal data, human case series, and prospective trials support using HBOT as a beneficial adjunct for management of refractory osteomyelitis.[87] The highest reported cure rates were obtained when HBOT was combined with culture-directed antibiotics and concurrent surgical debridement. Of patients who failed to respond to repetitive surgery and antibiotic care, 63% to 86% have had infections successfully arrested with addition of HBOT.[2,39,138] HBOT is indicated in patients who fail to respond to surgical debridement and appropriate antibiotics, or in whom osteomyelitis recurs after appropriate management.

In addition to long-bone osteomyelitis, specific cases of refractory osteomyelitis deserve special consideration because of their potential for life-threatening infections or their central neuraxial locations. HBOT has been recommended for mandibular, sternal, vertebral, and cranial osteomyelitis and for malignant external otitis. In one circumstance, it was used to treat progressive and potentially fatal *Pseudomonas aeruginosa* osteomyelitis involving

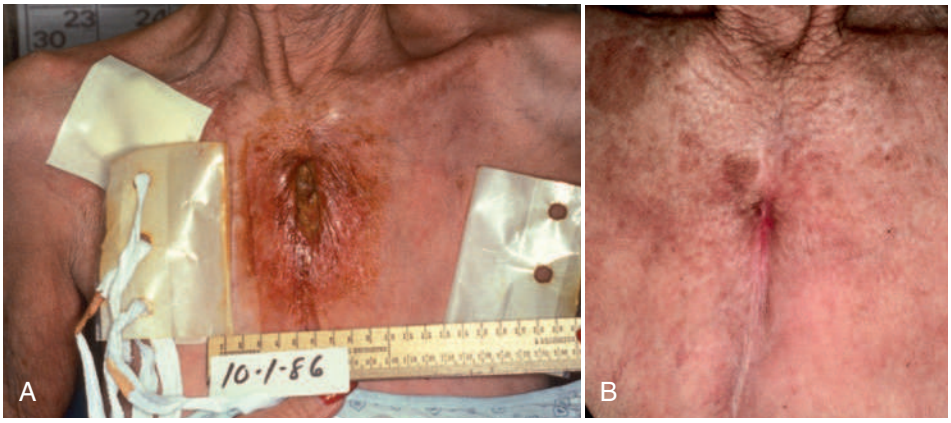

FIGURE 72-16 **A,** Woman with sternal osteomyelitis following coronary artery bypass graft that failed repeated debridements and two 6-week courses of IV antibiotics. **B,** After 40 sessions of HBOT, she had complete closure of the wound and resolution of osteomyelitis.

the external auditory meatus with osteomyelitis of the temporal bone.[1,48,159,174,187,258]

HBOT is used as an adjunct to standard surgical and antibiotic treatment for refractory osteomyelitis. Figure 72-16 shows a woman who developed sternal osteomyelitis after coronary artery bypass grafting. She was treated with two debridements and two separate 6-week courses of IV antibiotics. There was no improvement in her wound, and she was eventually referred for HBOT. After 40 treatments, she had complete resolution of her sternal infection and closure of the wound. HBOT is administered at 2 to 2.5 ATA for 90 to 120 minutes once per day, for an average of 20 to 40 treatments.

SEVERE ANEMIA

Severe anemia occurs when sufficient red cell mass is lost to compromise tissue oxygenation in a patient who, for medical, personal, or religious reasons, cannot or will not receive a blood transfusion. HBOT can dissolve enough oxygen to meet the oxygen requirements of tissues in the severely anemic patient until the bone marrow restores hemoglobin and red cell mass. Animal studies demonstrate the beneficial effect of HBOT in hemorrhagic shock models.[23,134,255] HBOT has been used successfully to correct accumulating oxygen debt in untransfusible patients and to reverse organ dysfunction in severe anemia associated with hemorrhagic shock.[74,76,143] The best published series of use of HBOT for treatment of exceptional anemia is reported by Hart; 26 patients with severe, exceptional blood loss anemia, defined as class IV hemorrhage, demonstrated a 70% survival rate.[88] Van Meter published a systematic review of HBOT for treatment of severe anemia.[236] HBOT may be used concurrently with erythropoietin, fluorocarbons, or stroma-free polymerized hemoglobin.

THERMAL BURNS

HBOT is used as an adjunct for treatment of thermal burns and has been shown to improve morbidity and mortality rates, reduce the length of the hospital stay, and decrease the need for surgery. The rationale for use of HBOT is to minimize edema, enhance host defenses, preserve marginally viable tissue, reduce ischemia-reperfusion injury, enhance leukocyte killing, preserve adenosine triphosphate (ATP), stimulate angiogenesis, and improve wound healing. Experimental data in animals have shown that HBOT reduces generalized edema and wound size.[102,167] Additionally, HBOT has a beneficial effect on angiogenesis and epithelial regeneration.[21] Biopsies of control animals showed progression to full-thickness injury, and biopsies of animals treated with HBOT showed preservation of dermal elements and capillary patency.[203]

Clinical experience in human burn patients suggests improved wound healing, reduction in mortality rates, decrease in the need for surgical procedures and resuscitative fluid requirements, and reduced hospital stay attributed to adjunctive HBOT.[42-44,81] In a prospective, randomized, controlled, double-blind trial comparing HBOT with sham controls in a human burn model, Niezgoda and colleagues[161] demonstrated significant reductions of wound size, hyperemia, and wound exudates in the HBOT group. Current recommendations include serious burns greater than 20% of the total body surface area or involving the hands, face, feet, or perineum that are deep partial- or full-thickness injuries. Although use of HBOT for burn treatment is approved by the Undersea and Hyperbaric Medical Society, some burn centers around the country do not use HBOT except for carbon monoxide poisoning. A 2004 Cochrane Review found insufficient evidence to support or refute use of HBOT in the management of thermal burns.[241] In a recent evidence-based review, Cianci and colleagues[45] analyzed 22 clinical series; 20 of these reports demonstrated benefit of the use of HBOT in thermal injury.

HBOT should be initiated as soon as possible after injury. Initially, three treatments at 2 to 2.5 ATA for 90 minutes of oxygen delivery within the first 24 hours are recommended, and then twice-daily treatments for 10 to 14 days for 20 to 30 treatments. Further treatments are often used to optimize graft uptake. Patients rarely need more than 50 treatments. Careful attention to fluid management is critical, because initial fluid requirements of burn patients can be several liters per hour. Ambient temperature in the chamber should be comfortable because thermal instability can be problematic, particularly within 1 to 2 hours of burn wound dressing changes. In cases of concurrent inhalation injury, patients can be maintained on ventilator support during treatment.

TRENDS IN HYPERBARIC OXYGEN THERAPY RESEARCH

ACUTE MYOCARDIAL ISCHEMIA

In animal studies, HBOT reduces the ischemic effect of coronary artery occlusion.[202,224] Thomas and coworkers demonstrated that a combination of thrombolytic therapy and HBOT is more effective in reducing the size of a myocardial infarction than either treatment alone.[224] In a rabbit model of aortic atherosclerosis, HBOT not only halted the progression of the disease but also accelerated the regression of atherosclerotic lesions.[113] HBOT decreased the size of necrosis in rat myocardium after irreversible occlusion of the left coronary artery.[52] HBOT may lessen or inhibit reperfusion injury by protecting oxidative metabolism in reperfusion-stunned myocardium. The Hyperbaric Oxygen and Thrombolysis in Myocardial Infarction study showed that treatment with HBO in combination with thrombolysis resulted in attenuated creatine phosphokinase rise, more rapid resolution of pain, and improved ejection fraction.[201]

Dekleva and colleagues[50] demonstrated that HBOT in conjunction with streptokinase in acute myocardial infarction reduced left ventricular volumes with associated increases in ejection fraction. In a controlled trial, patients who underwent percutaneous coronary intervention for acute myocardial infarction or unstable angina and received HBOT in the early peri-PCI period had lower clinical restenosis rate and less frequent development of late anginal symptoms.[193] The authors postulate that HBOT may induce expression of antioxidant enzymes that offer protection against atherosclerosis and reduce oxidation products in high-density lipoproteins. Additionally, fibrinolysis derived from endothelial cells and blood flow are enhanced by HBOT, which could result in reduction in recurrent thrombosis.[225] The results are provocative; larger randomized trials are indicated.

Bennett and colleagues compared all studies of acute coronary syndrome treatments that included HBOT; this metaanalysis consisted of six trials with 665 patients.[15] They found a significant decrease in mortality rates with HBOT ($P = .02$). There was evidence from individual trials of reduction in the risk for major adverse coronary events ($P = .03$) and certain dysrhythmias ($P = .01$) following HBOT. The review also showed that HBOT following acute coronary syndrome reduced the amount of time until pain relief following the onset of angina ($P < .0001$). Evidence from these few trials suggests that HBOT is associated with reduction in risk of death, volume of damaged muscle, risk of major adverse coronary events, and time to relief from ischemic pain. However, because of study flaws, low numbers of patients, and inconsistency of timing of HBOT for myocardial injury, the authors caution that the results be carefully interpreted. Further studies are required to define the mechanistic role of HBOT in myocardial ischemia.

ACUTE CEREBRAL ISCHEMIA

The effectiveness of HBOT in cerebral ischemia is controversial. It is difficult to compare studies because of different stroke models in different species with different HBOT protocols. Recent work demonstrates that HBOT appears to be protective in various models of focal cerebral ischemia.[199,210,256] In three recent reports using magnetic resonance imaging in an animal model, HBOT given 40 minutes to 6 hours after stroke onset was neuroprotective; HBOT improved infarct volume reduction and neurologic outcome.[126,188,238] Early HBOT may stop the process of ischemic infarct growth by rapidly restoring oxygen and energy to ischemic but still viable brain tissue. Other proposed mechanisms of HBOT include reduction of extracellular dopamine and inhibition of cyclooxygenase-2 overexpression in the cerebral cortex,[256,257] as well as effects on antiinflammatory and antiapoptotic responses.

Although animal studies suggest a possible role of HBOT in treating acute cerebral ischemia, clinical studies of humans have failed to show significant benefit from HBO, probably in part due to the long time window between symptom onset and HBOT.[17,162,185] Bennett and colleagues[17] reviewed 11 randomized controlled trials involving 705 patients and found no difference in case fatality rates at 6 months in those receiving HBOT or controls. Although some measures of disability and functional performance indicated improvement following HBOT, the authors found no good evidence that HBOT improved clinical outcomes.

TRAUMATIC BRAIN INJURY

Traumatic brain injury (TBI) can result in short- or long-term problems with memory, equilibrium, vision, headaches, decision making, judgment, fatigue, sleep, irritability, and emotional lability. TBI is one of the defining injuries of modern military conflict; an estimated 10% to 20% of U.S. service members suffer mild TBI from wars in the Middle East. TBI also occurs in nonmilitary activities that include motor vehicle accidents and sport and recreational accidents (e.g., bicycling, skiing). Incident rates vary widely; a rate of 130/100,000 individuals per year is a conservative estimate. HBOT has been proposed as a treatment for patients with TBI. Possible mechanisms by which HBOT may have a beneficial effect include modulation of ischemia-reperfusion; reduction of brain inflammation and edema; improvement of oxygen availability to idling, but viable, neurons in the brain; and increase in metabolic performance of chronically impaired neurons leading to improved integrative plasticity.[141a]

There are multiple case reports of off-label use of HBOT for TBI that describe improvement in cognitive function; however, these reports have been criticized because they are not controlled trials. Randomized controlled trials report conflicting outcomes. In a 2012 Cochrane Review, Bennett and associates[16] reviewed seven studies involving 571 people (285 receiving HBOT); none of the studies were blinded. Although a few studies showed that HBOT might reduce the risk of death, there was little evidence that HBOT improved outcomes.

Wolf and colleagues[251] studied 50 U.S. Air Force personnel diagnosed with TBI in a double-blinded, randomized controlled study and found that compared with controls, HBOT did not reduce symptoms following concussion or symptoms of posttraumatic stress disorder. In a multicenter, double-blinded, sham-controlled clinical trial of 72 military service members with mild TBI, HBOT showed no benefit compared with sham compressions, although both groups improved compared with those who received care for postconcussive symptoms alone.[147] HBOT is currently not the standard of care for patients with TBI.

ORGAN TRANSPLANTATION

HBOT has been suggested to limit the ischemia-reperfusion injury that can complicate organ transplantation.[34,153] In animal studies, use of HBOT in transplantation has shown a positive response. HBOT influences ischemia-reperfusion injury and consequent acute cellular rejection. Several clinical studies have been published on using HBOT with liver transplantation. All such studies relate either to its use in the posttransplantation period for management of hepatic artery thrombosis[54,77,140] or its use in acute liver failure prior to transplantation. One major potential advantage of HBOT is that it affects the processes of ischemia-reperfusion injury and acute cellular rejection through numerous cellular and molecular mechanisms. HBOT has been shown to stimulate hepatocyte proliferation after liver resection in animal and human studies.[209,228] In addition, it may play a role as a liver support adjunct for fulminant hepatic failure, posttransplantation graft dysfunction, and acute hepatic artery thrombosis. More research is required on the influence of HBOT in liver transplantation, specifically its effect on the immune response.

APPLICATIONS FOR HYPERBARIC OXYGEN THERAPY IN WILDERNESS MEDICINE

HBOT plays a significant role in wilderness medicine for treatment of diving-related illnesses and altitude illness. HBOT has been investigated in other wilderness-related entities, such as frostbite, brown recluse spider bite, and, more recently, heatstroke. HBOT has also been successful in treating necrotizing soft tissue infection from a stingray puncture and *Vibrio vulnificus* septicemia and cellulitis from eating raw fish.[182,243] HBOT has been tried in an experimental rat model of sea nettle envenomation. Although it had no effect on mortality rate, HBOT protected against venom-induced decreases in brain blood flow and maintained oxygenation in envenomed animals.[151]

FROSTBITE

The first case of HBOT use with cold injury was reported by Ledingham in 1963, followed by a number of reports for its use in the 1970s. Numerous case reports are available, but controlled trial data are lacking. Initial experiments in rabbits showed that HBOT had no effect on tissue loss and variation of injury.[66,67,85] However, other studies have shown benefit. If HBOT was administered immediately after rewarming, the mean tissue loss was decreased in rabbits. This benefit was significantly reduced by delays in treatment, particularly beyond 24 hours.[67,168] Research

into the pathophysiology of frostbite has revealed marked similarities to inflammatory processes seen in thermal burns, ischemia-reperfusion injury, and crush injury.[154] Hence, HBOT has theoretical advantages as an adjunct in the treatment of frostbite. An 11-year-old boy who suffered deep frostbite on six fingers was treated with HBOT and had complete recovery after 14 days.[242] In another report, HBOT was shown to improve skin microcirculation by increasing the number of nutrient capillaries in frostbitten areas on the toes.[63] A young female who suffered frostbite to her fingers and was treated with HBOT had complete return of function, with superficial tissue loss to one finger only.[64] A case of frostbite due to contact with liquid helium gas was successfully treated with HBOT with good recovery.[192] Kemper and associates[105] reported a case of deep frostbite of the toes treated with HBOT after a 21-day delay with good outcome. They reviewed 17 human case reports of frostbite treated with HBOT, all showing positive effects with no amputations.

The ideal time to initiate HBOT for frostbite is during the rewarming period because of the reperfusion aspect of the injury. Although there is no standard protocol, HBOT can be given at 2 to 2.5 ATA for 90 to 120 minutes three times in the first 24 hours, followed by twice-daily treatments until there is no longer threatened tissue loss. If a frostbitten limb has already been rewarmed and is at risk for significant tissue loss, HBOT is still indicated to help hasten demarcation, decrease the risk for infection, enhance the survival of the damaged tissue in the gradient of injury, and improve flap survival.

BROWN RECLUSE SPIDER BITE

The brown recluse spider *(Loxosceles reclusa)* is known to cause necrotic skin lesions in humans. Most bites resolve spontaneously, but a few progress to severe local necrosis and tissue loss (see Chapter 43). Treatment of brown recluse spider bites with HBOT remains controversial. HBOT has been proposed to directly inhibit neutrophil adherence to the endothelium and to decrease venom-induced ischemia-reperfusion injury, as well as perhaps inactivate sphingomyelinase or other components of the venom.[146] HBOT may minimize the wound size by increasing oxygen tension within the wound and thereby increasing angiogenesis and fibroblast proliferation.

Animal models show conflicting data. In experimental models using rabbits, there was neither reduction in lesion size nor histologic improvement from HBOT.[176] Similarly, there was no decrease in lesion size in a swine model.[91] Two reports have shown benefit in controlled animal studies. One showed histologic improvement in a rabbit model, but found no difference in lesion size.[205] A randomized, controlled trial using rabbits demonstrated that HBOT reduced the size of lesions, even when treatment was delayed by 2 days.[139] A single treatment given immediately was as effective as were multiple treatments.

Several case studies in humans suggest that HBOT may be beneficial in managing dermatonecrosis associated with *L. reclusa*. Svendsen first described the use of HBOT for treatment of brown recluse spider bites.[212] He treated six patients who had clinically deteriorating lesions with HBOT 2 to 6 days after the bite with twice-daily treatments for 3 days. All lesions healed well without surgery, skin sloughing, or significant scarring. Kendall and Caniglia reported 47 cases with good outcomes; only 1 of 48 patients required skin grafting.[106] Maynor and colleagues treated 14 patients, all of whom healed without scarring, disability, or a need for skin grafting.[137] Two cases of potentially devastating bites, one to the glans penis and another to the periorbital region on a child's face, were both treated with HBOT and had good outcomes.[30,99]

Tutrone and colleagues published a review of the treatment of brown recluse spider bites with HBOT[232] and found it may be effective at reducing scarring and complications. However, there are no randomized, controlled trials of HBOT in human cases of *Loxosceles* envenomation and no standardized HBOT protocols. HBOT can be given at 2 to 2.5 ATA for 90 to 120 minutes on a daily basis for 2 to 10 treatments. Systemic symptoms, size of the dermatonecrotic lesion, and area of surrounding erythema should be assessed on a daily basis.

HEATSTROKE

Heatstroke is characterized by significant hyperthermia, altered mental status, and varying degrees of multiple-organ failure. In rodents, heatstroke causes vasoplegic shock, intracranial hypertension, and cerebral ischemia and injury.[122] In experimental studies on rats, HBOT was beneficial in resuscitating rats with experimental heatstroke. HBOT reduced heatstroke-induced arterial hypotension, hypoxia, plasma tumor necrosis factor-alpha overproduction, and cerebral ischemia. HBOT improved survival rates during heatstroke by augmenting mean arterial pressure and local cerebral blood flow and by decreasing multiple-organ dysfunction.[164,230] In a diabetic rat model of heatstroke, HBOT increased survival times by reducing heat-induced activated inflammation and ischemic and oxidative damage in the hypothalamus and other brain regions.[119] In another rat model for heatstroke, activated protein C or HBOT was equally effective in reducing heat-induced inflammation, a hypercoagulable state, and multiple-organ injury. Combined activated protein C and HBOT reduced these heatstroke reactions better than did activated protein C or HBO alone.[213]

Additionally, HBOT has been used to successfully treat a heatstroke patient with multiple-organ dysfunction.[163] HBOT is not standard therapy for heatstroke, however. Further work is needed to support its use as an adjunct to conventional treatment options in severe cases.

FIELD TREATMENT OF COMBAT TRAUMA

Because the indications for HBOT closely overlap those preset in combat casualties, HBOT has been increasingly used by U.S. and NATO forces for treatment of combat-related trauma.[86,129,239] Hart reviewed preliminary wound statistics from Operation Iraqi Freedom.[86] Approximately 70% of injured soldiers sustained extremity trauma. Of 560 surgical procedures performed, 31% were complicated by persistent tissue necrosis, wound infection, graft failure, or delayed wound healing. HBOT is effective in correcting tissue ischemia and hypoxia, as an adjunctive treatment for compartment syndrome, and for controlling wound infections and augmenting healing. Field use of HBOT for combat injuries could limit the extent of surgical debridements, improve tissue flap and graft survival rates, decrease wound infection rates, and improve healing rates for complex wounds. HBOT could be used as a temporizing measure pending operating room availability.

Several portable hyperbaric chambers have been suggested for use in field treatment. The Hyperlite Hyperbaric Chamber or Emergency Evacuation Hyperbaric Stretcher is ideal for remote locations without hospital support. The U.S. Navy's Transportable Recompression Chamber System is a multiplace chamber designed for treatment and evacuation of injured divers, but can be used for any emergency hyperbaric treatment and moved to almost any location. The Fly Away Recompression Chamber is a much larger system (61 m^3 [200 $feet^3$]), consisting of a double-lock recompression chamber capable of treating divers or any patient in need of HBOT. A multiplace hyperbaric chamber could be put aboard a designated hospital ship for treatment of combat wounds.

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CHAPTER 73

Injuries From Nonvenomous Aquatic Animals

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The expanses of oceans, estuaries, and fresh waters that cover the earth are the greatest wilderness. Seventy-one percent (or 362 million km^2 [139,768,981 miles^2]) of the earth's surface is composed of ocean, the volume of which exceeds 523 km^3 (325 million miles^3). Underneath the surface lie huge mountain ranges, deep valleys, and many active volcanoes. Nearly one-half of the sea floor is composed of an abyssal plain, which lies at an average depth of 4 km (2.5 miles) and is largely devoid of life forms. Within the undersea realm exist four-fifths of all living organisms. Hundreds of thousands of marine species have been taxonomically described and thousands are as yet undiscovered.

The opportunity for direct encounters with aquatic organisms continues to increase because of enhanced recreational, industrial, scientific, and military oceanic and freshwater activities related to ever-rising human populations. The most common cause of injuries is handling of animals that bite and sting in self-defense, followed by provoked encounters and then unprovoked encounters.

Nearly 80% of the world's population resides in coastal regions. In the United States, 50% of the population lives within 80 km (50 miles) of a coastline. It is estimated that 127 million U.S. citizens live along the coasts. A significant proportion of this population is directly involved as entrants into the aquatic world. Therefore, it is imperative that clinicians be familiar with hazards unique to the aquatic environment.

Although noxious marine organisms are concentrated predominantly in warm temperate and tropical seas, particularly in the Indo-Pacific region, hazardous animals may be found as far north as 50 degrees latitude. Saltwater aquariums in private homes and public settings, intercontinental seafood shipping, and increasing accessibility of air travel to aquatic recreationists, most notably scuba/skin divers and surfers, contribute to the risks.

Like the rainforest, the ocean depths have the potential to reveal virtually limitless active pharmaceutical agents, including antihelmintic, anticoagulant, antifungal, antimalarial, antiprotozoal, antituberculosis, antiinflammatory, and antiviral compounds.[129] Genetically engineered reproduction of the adhesive protein of the popularly consumed marine mussel *Mytilus edulis* has created a tissue adhesive agent that may one day prove superior to cyanoacrylic compounds. The annelid sandcastle worm *(Phragmatopoma californica)* manufactures a glue used to construct a protective home of sand and shell fragments. This is being investigated as a tissue adhesive for fragmented human bones. Toxins isolated from ascidians (tunicates, or sea squirts) include cyclic peptides, some of which (ecteinascidin-743, aplidine) have undergone evaluation for cancer chemotherapy; others (e.g., thiocoraline and kahalalide F) may follow. Investigative techniques continue to improve. In pursuit of anatomic information that can elucidate the biology and ecology of fish, evolution, cellular physiology, and aquatic models of human disease, nuclear radiologists have performed in vivo nuclear magnetic resonance imaging (MRI) and spectroscopy of anesthetized (tricaine methanesulfonate [MS222]) aquatic organisms.[26] Most marine organisms rely on antimicrobial components of their innate immune defenses to combat pathogens. From this unique perspective, scientists seek to identify novel antimicrobials, among which the most promising are marine cationic antimicrobial peptides, defined as small (10 to 40 amino acids) peptides containing a prevalence of positively charged residues (lysine and arginine).[147] As examples, these have been found in teleost fishes (pardaxin, pleurocidin, hepcidin), tunicates (styelin, clavanin), chelicerates (big defensin, tachyplesin), crustaceans (callinectin), gastropods (dolabellanin), and mollusks (mytilin). Their activities include synergy to induce cell lysis, modulating the host immune response, chemotaxis, macrophage development, production or inhibition of cytokines, and so forth. Sponges (phylum Porifera) and sharks (order Selachii) have become particular foci of biomedical screening over the past three decades.

Despite the wondrous nature of the deep, danger exists. The ubiquity of hazardous creatures and their propensity to appear at inopportune times make it imperative to be aware of them, to respect their territorial rights, and to avoid unpleasant contact with them.

DIVISIONS AND DEFINITIONS

Dangerous aquatic animals are divided into four groups: (1) those that bite, rip, puncture, or deliver an electric shock without envenomation; (2) those that sting (envenom),[173] discussed in Chapters 74 and 75; (3) those that are poisonous on ingestion (see Chapter 77); and (4) those that induce allergies (see Chapter 78). Aquatic skin disorders are discussed in Chapter 76.

IN DEFENSE OF THE FISH

As in all nature (except for humans), indiscriminate aggression is rarely involved when injuries are inflicted by aquatic animals. Most injuries result from gestures of warning or self-defense; aquatic creatures rarely attack humans without provocation. Attacks are made in defense of young, in territorial dispute when mating activities are interrupted, or during active procurement of food. *Caution* is the key word when dealing with potentially injurious aquatic creatures.

GENERAL PRINCIPLES OF FIRST AID

The physician must adhere to fundamental principles of medical rescue. Although many injuries and envenomations have unique clinical presentations, the cornerstone of therapy is immediate attention to the airway, breathing, and circulation. Along with specific interventions directed against a particular venom or poison, the rescuer must simultaneously be certain that the victim maintains a patent airway, breathes spontaneously or with assistance, and is supported by an adequate blood pressure. Because marine attacks and envenomations often affect swimmers and divers, the rescuer should anticipate drowning (see Chapter 69), immersion hypothermia (see Chapter 8), and decompression sickness or arterial air embolism (see Chapters 71 and 72). Any victim rescued from the ocean should be thoroughly examined for signs of a bite, puncture, or sting if such is a possibility.

WOUND MANAGEMENT

Whether the injury is a bite, abrasion, or puncture, meticulous attention to basic wound management is necessary to facilitate healing and minimize posttraumatic infection.

WOUND IRRIGATION

All wounds acquired in the natural aquatic environment should be vigorously irrigated with sterile diluent, preferably normal saline (0.9% sodium chloride) solution. Seawater is not recommended as an irrigant, because it carries a hypothetical infection risk. Sterile water or hypotonic saline is acceptable. Tap water (preferably disinfected) is a suitable alternate irrigant if a sterile solution cannot be obtained in a timely manner.[5] Irrigation should be performed before and after debridement. A 19-gauge needle or 18-gauge plastic intravenous (IV) catheter attached to a syringe that delivers a pressure of 7031 to 14,061 kg/m^2 (10 to 20 psi) will dislodge most bacteria without forcing irrigation fluid into tissue along the wound edges or deeper along dissecting tissue planes. Convenient ring-handle syringes with blunt irrigation tips and IV tubing that connects to standard IV bags are useful. At least 100 to 250 mL of irrigant should be flushed through each wound. If a laceration is from a stingray, proteinaceous (and possibly) heat-labile venom may be present in the wound. Therefore, if the wound is still painful at the time of irrigation, the irrigant may be warmed to a maximum temperature of 45° C (113° F).[9]

Antiseptic may be added to the irrigant if the wound appears to be highly contaminated. Povidone-iodine solution in a concentration of 1% to 5% may be used with a contact time of 1 to 5 minutes.[199] When antiseptic irrigation is completed, the wound should be thoroughly irrigated with normal saline or tap water to minimize tissue toxicity from the antiseptic. Antiseptics that are particularly harmful to tissues include full-strength hydrogen peroxide, povidone-iodine scrub solution, hexachlorophene detergent, and silver nitrate.

Scrubbing should be used to remove debris that cannot be irrigated from the wound. Sharp surgical debridement is preferable to sponge scrubbing, which may increase infection rates, particularly when applied with harsh antiseptic solutions. Poloxamer 188 (Pluronic F-68), a nontoxic, nonionic surfactant skin wound cleanser (found in Shur-Clens 20%), does not offer any significant advantage over traditional sterile saline irrigation.

WOUND DEBRIDEMENT

Debridement is more effective than irrigation at removing bacteria and debris. Crushed or devitalized tissue should be removed with sharp dissection to provide clean wound edges and encourage brisk healing with minimal infection risk. The limitations are those imposed by anatomy, specifically skin tautness or the presence of vital structures. Anesthesia of wound edges may be attained by regional nerve block or local infiltration with lidocaine or bupivacaine, which do not damage local tissue defenses. A topical anesthetic mixture containing epinephrine may be less desirable because of the vasoconstrictive effects and theoretical infection-potentiating effects. Definitive wound exploration, debridement, and repair should be undertaken in an appropriate sterile environment. It is often impractical to explore complex wounds in the emergency department, and some wounds may necessitate surgical exploration in the operating room. Operating loupes can be used as needed to inspect the wound for residual foreign material, such as sand, seaweed, tooth or spine fragments, or integumentary sheath shards. Standard radiographs, static soft tissue techniques, computed tomography, ultrasound, MRI, or fluoroscopy should be used preoperatively or perioperatively to localize spines or teeth.

WOUND CLOSURE

The decision to close a wound must weigh the risk of infection. The incidence of infection is high in wounds acquired in natural bodies of water because such wounds may be contaminated with venom or potentially virulent microorganisms, or both; because early adequate irrigation and debridement are often unavailable; and because definitive care is often delayed. Tight wound closure restricts drainage and promotes bacterial proliferation, particularly from anaerobes, which are common contaminants. Wounds at high risk in this regard include those on the hands, wrists, or feet; punctures and crush injuries; wounds into areas of fat with poor vascularity; and wounds to victims who are immunosuppressed. Whenever possible, the use of sutures to close dead space in contaminated wounds should be minimized because the absorbable sutures act as foreign bodies.

PROPHYLAXIS AGAINST TETANUS

Any wound that disrupts the skin can become contaminated with *Clostridium tetani*. Anaerobic bacteria, predominantly of the genus *Clostridium,* have been isolated in shark tissue and as part of the oral flora of alligators and crocodiles. Proper immunization with tetanus toxoid virtually eliminates the risk of disease. Although it was previously accepted that the protective level of toxin-neutralizing antibody is 0.01 antitoxin unit/mL, it appears that clinical tetanus can develop despite an antibody level many times that amount. Therefore, it is imperative to provide an early and adequate booster injection. If the victim is older than age 50 years, is from an underdeveloped country, or cannot provide a definite history of tetanus immunization, it is likely that circulating toxin-neutralizing antibody will be suboptimal. Prophylaxis should be provided according to the scheme shown in Chapter 21 in Table 21-6. Tdap (tetanus, reduced diphtheria, and acellular pertussis vaccine) should be used instead of Td for routine tetanus boosters and wound management in adolescents and adults.

BACTERIOLOGY OF THE AQUATIC ENVIRONMENT

Wounds acquired in the aquatic environment are soaked in natural source water and sometimes contaminated with sediment. Penetration of the skin by the spines or teeth of animals, the razor edges of coral or shellfish, or mechanical objects such as the blades of a boat propeller may inoculate pathogenic organisms into a wound. Sports activities, such as surfing, snorkeling, diving, and fishing, lead to ubiquitous abrasions and minor lacerations that heal slowly and with marked soft tissue inflammation. Fishing boats, bearing decks and other structures that seldom are adequately cleaned, are particularly rich sources of pathogens. Wounds acquired in the aquatic environment tend to become infected and may be refractory to standard antimicrobial therapy. Not infrequently, indolent or extensive soft tissue infections develop in the normal or immunocompromised host.[15,151] A clinician faced with a serious infection caused by an aquatic injury frequently needs to administer broad-spectrum antibiotics to a patient before definitive laboratory identification of pathogenic organisms has been obtained.

MARINE BACTERIOLOGY

Marine Environment

Ocean water provides a saline milieu for microbes. The salt dissolved in ocean water (3.2% to 3.5%) is 78% sodium chloride (sodium 10.752 g/kg; chlorine 19.345 g/kg). Other constituents include sulfate (2.791 g/kg), magnesium (1.295 g/kg), potassium (0.39 g/kg), bicarbonate (0.145 g/kg), bromine (0.066 g/kg), boric acid (0.027 g/kg), strontium (0.013 g/kg), and fluorine (0.0013 g/kg). The temperature of the surface waters varies with latitude, currents, and seasons. Tropical waters are warmer and maintain a more constant temperature than do temperate and subtropical waters, which are subject to substantial meteorologic variation. Shallow and turbulent coastal waters are generally richer in nutrients than is the open ocean, which is reflected in the diversity of life that can be identified in the intertidal zone. Although the greatest number and diversity of bacteria are found near the ocean surface, diverse bacteria and fungi are found in marine silts, sediments, and sand and within the oral cavities of marine organisms. In ocean waters having marked differences in salinity/density, the greatest concentration of bacteria is noted at the thermocline, where changes in both temperature and salinity are usually found.[159] Marked vertical salinity stratification is the norm in estuaries where freshwater inflow "floats" over the top of a denser wedge of marine waters. This effect is less active in

other coastal waters, where tide- and wind-driven perturbations may create a more even distribution of sediments, microbes, salinity, and temperature. Microbes are most abundant in areas that have the greatest numbers of higher life forms. Growth requirements are species specific with respect to the use of organic carbon and nitrogen sources, requirements for various amino acids, vitamins and cofactors, sodium, potassium, magnesium, phosphate, sulfate, chloride, and calcium. Most marine bacteria are facultative anaerobes, which can thrive in oxygen-rich and oxygen-poor environments. Few are obligatory aerobes or anaerobes. Some marine bacteria are highly proteolytic, and the proportion of proteolytic bacteria seems to be greater in the oceans than on land or in freshwater habitats.[159] It has been observed that sharks may harbor bacteria that are resistant to many drugs; this phenomenon could be a result of natural immunity, or it may be that some of these animals have encountered synthetic drugs that found their way into the sea via effluents.

Diversity of Organisms

Unique conditions of nutrient and inorganic mineral supply, temperature, and pressure have allowed evolution of unique, highly adapted marine microbes.[215,216] In addition, numerous other bacteria, microalgae, protozoa, fungi, yeasts, and viruses have been identified in or cultured from seawater, marine sediments, marine life, and marine-acquired or marine-contaminated infected wounds or body fluids of septic victims. In their natural environment, the bacteria serve to scavenge and transform organic matter in the intricate cycles of the food and growth chains. Some of these bacteria are listed in Box 73-1.[157] Enteric pathogenic bacteria have been isolated from sharks.[76] A shark attack victim in South Africa who sustained serious injuries to his lower extremities was reported to have developed a fulminant infection attributed to *Bacillus cereus* shown to be sensitive to fluoroquinolones, amikacin, clindamycin, vancomycin, and tetracyclines and resistant to penicillin and cephalosporins (including third-generation cephalosporins). In another report, one shark attack victim in Australia grew both *Vibrio parahaemolyticus* and *Aeromonas caviae* from his wounds, whereas another grew *Vibrio alginolyticus* and *Aeromonas hydrophila* from his wounds.[162] It is now fairly well known that *Vibrio* species and *Aeromonas* species are potential pathogens residing in ocean water and fresh water.

Marine bacteria are generally halophilic (thrive in saline conditions), heterotrophic (require exogenous carbon and nitrogen-containing organic supplements), motile, and gram-negative rod forms. *Halomonas venusta,* a halophilic, nonfermentative, gram-negative rod, was reported as a human pathogen in a wound that originated from a fish bite.[200] Previous opinions that enteric pathogens (associated with the intestines of warm-blooded animals) deposited into marine environments ultimately succumb to sedimentation, predation, parasitism, sunlight, temperature, osmotic stress, toxic chemicals, or high salt concentrations may be untrue.[75] Pathogens may accumulate in surface water in association with lipoidal particulates, from which they are rapidly dispersed toward the shore by wave and wind activity. In addition, dredging, storms, upwellings, and other benthic disturbances may churn enteric organisms into the path of wastewater nutrients. In the United States, coastal and Great Lakes beaches regularly have bacteria counts above the Environmental Protection Agency's threshold values for safety. Sewage spills and intentional industrial effluent release contribute to harmful contamination, notably including enteric bacteria.

BOX 73-1 Bacteria and Fungus Isolated from Marine Water, Sediments, Marine Animals, and Marine-Acquired Wounds

Achromobacter
Acinetobacter lwoffii
Actinomyces
Aerobacter aerogenes
Aeromonas hydrophila
Aeromonas salmonicida
Aeromonas sobria
Alcaligenes faecalis
Alteromonas espejiana
Alteromonas haloplanktis
Alteromonas macleodii
Alteromonas undina
Bacillus cereus
Bacillus subtilis
Bacteroides fragilis
Branhamella catarrhalis
Chromobacterium violaceum
Citrobacter
Clostridium botulinum
Clostridium perfringens
Clostridium tetani
Corynebacterium
Edwardsiella tarda
Enterobacter aerogenes
Erysipelothrix rhusiopathiae
Escherichia coli
Flavobacterium
Fusarium solani
Grimontia (Vibrio) hollisae
Klebsiella pneumoniae
Legionella pneumophila
Micrococcus luteus
Micrococcus sedentarius
Moraxella lacunata
Mycobacterium marinum
Neisseria catarrhalis
Pasteurella multocida
Photobacterium (Vibrio) damsela
Propionibacterium acnes
Proteus mirabilis
Proteus vulgaris
Providencia stuartii
Pseudomonas aeruginosa
Pseudomonas cepacia
Pseudomonas maltophilia
Pseudomonas putrefaciens
Pseudomonas stutzeri
Salmonella enteritidis
Serratia
Staphylococcus aureus
Staphylococcus epidermidis
Streptococcus
Vibrio alginolyticus
Vibrio carchariae
Vibrio cholerae
Vibrio fluvialis
Vibrio furnissii
Vibrio harveyi
Vibrio mimicus
Vibrio parahaemolyticus
Vibrio splendidus I
Vibrio vulnificus

WOUND INFECTIONS CAUSED BY *VIBRIO* SPECIES

Vibrio organisms can cause gastroenteric disease (gastroenteric *Vibrio* infections are discussed in Chapter 82) and soft tissue infections, particularly in immunocompromised hosts. Extraintestinal infections may be associated with bacteremia and death. *Vibrio* species are the most potentially virulent halophilic organisms that flourish in the marine environment. The teeth of a great white shark were swabbed and yielded *V. alginolyticus, V. fluvialis,* and *V. parahaemolyticus*.[30] Mako shark tooth culture has yielded *V. damsela, V. furnissii,* and *V. splendidus I*.[6] *V. parahaemolyticus* has also been identified in freshwater habitats.[10] Water that is brackish (salinity of 15 to 25 parts per thousand [ppt]) allows growth of *Vibrio* species if appropriate nutrients are present; *V. vulnificus* infection has been documented after exposure to waters with salinities of 2 and 4 ppt. The optimal season for exposure appears to be summer, when water temperatures encourage bacterial proliferation. In most studies reported, infections seem to cluster during summer months; this may be related to increased numbers of people at the seashore.[63] This has been corroborated to some degree by the observation that *V. parahaemolyticus* cultured from marine mammals was recovered only in warmer months of the year in the northeastern United States or in animals from subtropical regions. Sharks appear to develop some immunity to autochthonous *Vibrio* species, as suggested by detection of a binding protein similar to the immunoglobulin M (IgM) subclass of immunoglobulin. Allochthonous (for the shark) *Vibrio* species, such as *V. carchariae,* may be the agents of elasmobranch disease when the animal is under stress. Other species, such as *V. anguillarum* and *V. tapetis,* are pathogens of aquatic vertebrates or invertebrates.[12]

Vibrio species are halophilic, gram-negative rods that are facultative anaerobes capable of using D-glucose as their sole or principal source of carbon and energy.[51] These organisms are part of the normal flora of coastal waters not only in the United States but also in many exotic locations frequented by recreational and industrial divers and seafarers. *Vibrio* species are mesophilic organisms and grow best at temperatures of 24° to 40°C (75.2° to 104°F), with essentially no growth below 8° to 10°C (46.4° to 50°F). Certain other "marine bacteria" are facultative psychrophiles (thrive in cold temperatures) or barophiles (thrive in high pressures), or both. *Vibrio* species seem to require less sodium for maximal growth than do other more fastidious marine organisms, a factor that explains their presence in estuarine waters and allows explosive reproduction in the 0.9% saline environment of the human body. At least 11 of the 34 recognized

Vibrio species have been associated with human disease.[51] Wound infections have been documented to yield *V. cholerae* O group 1 and non-O1, *V. parahaemolyticus, V. vulnificus, V. alginolyticus,* and *V. damsela*. Septicemia, with or without an obvious source, has been attributed to infections with *V. cholerae* non-01, *V. parahaemolyticus, V. alginolyticus, V. vulnificus,* and *V. metschnikovii*. *Vibrio* may infect fish, causing significant mortality rates in fish culture facilities. The affliction manifests with lethargy, loss of appetite, skin sores, exophthalmia, and gastrointestinal hemorrhage.

Vibrio parahaemolyticus

Vibrio parahaemolyticus is a halophilic gram-negative rod. The organisms are found in waters along the entire coastline of the United States. Generally, the incidence of clinical disease is greatest in warm summer months when the organism is commonly found in zooplankton. *V. parahaemolyticus* absorbs onto chitin and to minute crustacean copepods that feed on sediment. It has been postulated that unusual warm coastal currents (such as El Niño) may contribute to increased proliferation of *Vibrio* species. The optimal growth temperature of *V. parahaemolyticus* is 35° to 37°C (95° to 98.6°F); under ideal conditions, the generation time has been estimated at less than 10 minutes, with explosive population growth from 10 to 10^6 organisms in 3 to 4 hours.

Extraintestinal wound infections are most common in persons who suffer chronic liver disease or immunosuppression. Although more than 95% of *V. parahaemolyticus* strains associated with human illness are positive for the Kanagawa reaction (production of a cell-free, heat-stable hemolysin on high-salt-mannitol [Wagatsuma] agar), the relationship of this reaction to pathogenicity is not yet clear. Furthermore, most marine strains are not Kanagawa positive. Virulence factors include proteases, beta-hemolysins (thermostable direct hemolysin [tdh] and tdh-related hemolysin [trh]), adhesins, and the expression of virulence genes, including the toxR operons.[12,174] Some primary soft tissue infections previously attributed to *V. parahaemolyticus* may theoretically be attributed to misidentified *V. vulnificus*.

Vibrio vulnificus

Vibrio vulnificus (formerly known as a "lactose [fermenting]-positive" vibrio) is a halophilic gram-negative bacillus. *V. vulnificus* (Latin for "wounding") is found in virtually all U.S. coastal waters and has been reported to cause infection worldwide.[11] It prefers salinity of 0.7% to 1.6%; and although it prefers a habitat of warm (at least 20°C [68°F]) seawater, it can be found in much colder water. It does not appear to be associated with fecal contamination of seawater. It has been shown to exist in Chesapeake Bay with bacterial counts comparable to those reported from the Gulf of Mexico.[213]

V. vulnificus may or may not have an acidic polysaccharide capsule (opaque colony), which confers protection against bactericidal activity of human serum and phagocytosis and thus renders the organism more virulent in animals. At extremely low frequency, some strains can shift between unencapsulated (avirulent; translucent colony) and capsulated (virulent) serotypes. The encapsulated isolates show exquisite (positive) sensitivity to iron. Virulent isolates can use 100% but not 30% saturated (normal for humans) transferrin as an iron source, as well as iron in hemoglobin and hemoglobin-haptoglobin complexes. *V. vulnificus* exhibits enhanced growth and virulence in the presence of increased serum iron concentration or saturated transferrin-binding sites.[2] *V. vulnificus* is classified into three biotypes: biotype 1 is pathogenic for humans and biotype 2 is pathogenic for fish.[51,53] Biotype 3 causes soft tissue infections and septicemia following contact with fish from freshwater ponds; it was first noted in Israel. Within biotype 1, various genetically distinct subgroups identified by randomly amplified polymorphic deoxyribonucleic acid (DNA) polymerase chain reaction (PCR) appear to be especially virulent.[8] Furthermore, complete genomic sequencing of *V. vulnificus* YJ016, a biotype 1 strain, reveals gene clusters related to pathogenicity (cell adhesion, colonization, cytotoxicity, and tissue destruction), including capsular polysaccharide, siderophore biosynthesis and transport, and heme receptor and transport.[53] In vivo antigen technology can identify virulence genes produced and expressed in humans.[102] The ability of biotype 1 to multiply and produce a toxic metalloprotease in human serum may be a prominent virulence factor.[206]

Infection worsens rapidly after the initiation of symptoms and has been noted most frequently in men older than age 40 years with preexisting hepatic dysfunction (particularly cirrhosis), end-stage renal impairment, leukopenia, or impaired immunity (malignancy, leukemia, hypogammaglobulinemia, human immunodeficiency virus [HIV] infection, diabetes, bone marrow suppression, long-term corticosteroids), although it has been reported in young, previously healthy individuals.[22,94,107,109,134] One case followed application of fish blood by a healer as a traditional remedy to a chronic leg ulcer in an obese patient suffering from recurrent erysipelas.[184] Preexisting liver disease is a predictor of death, with 50% of such individuals in one series succumbing to the illness.[87] Persons with high serum iron levels (from chronic cirrhosis, hepatitis, thalassemia major, hemochromatosis, multiple transfusions [such as are given for aplastic anemia]) or achlorhydria (low gastric acid; may be iatrogenically induced with H_2 blockers) may be at greater risk for fulminant bacteremia.[2,178,193] This has been attributed in part to the protective effect of gastric acid, the iron requirement of the organism, and the effects of liver disease (decreased polymorphonuclear leukocyte and macrophage activity, flawed opsonization, and shunting of portal blood around the liver). It has been proposed that an effective host response against *V. vulnificus* and similar iron-sensitive pathogens (e.g., *Listeria monocytogenes, Klebsiella* spp., and *Yersinia* spp.) is in part augmented by hepcidin, a cysteine-rich cationic antimicrobial peptide central to iron metabolism.[8,204] *V. vulnificus* produces a siderophore *(vulnibactin)* and a protease that may enhance pathogenicity.[2] Other pathogenicity factors may be the polysaccharide capsule, hemolysin, type IV pili and other proteases, including a serine protease and a 45-kDa metalloprotease regulated through quorum sensing at a lower temperature than core body temperature.[134,205]

The syndrome consists of flu-like malaise, fever, vomiting, diarrhea, chills, hypotension, and early skin vesiculation that evolve into necrotizing dermatitis and fasciitis, with vasculitis and myositis (Figures 73-1 and 73-2).[214] Hematogenous seeding of vibrios to secondary cutaneous lesions is probable. Primary wound infections (≈ 30% of cases) rapidly show marked edema, with erythema, vesicles, and hemorrhagic or contused-appearing bullae, progressing to necrosis.[63] This may require radical surgical debridement or amputation. Up to 25% of these victims may have sepsis. When *V. vulnificus* is recovered from the blood of a victim with sepsis attributable to a wound infection, the case fatality rate may exceed 30%.[87] Necrotizing fasciitis from this bacteria has also been described to produce a fatal toxic shock–like syndrome.[63] Extracellular elastin-lysing proteases elaborated by the organism, as well as a potent collagenase, probably contribute

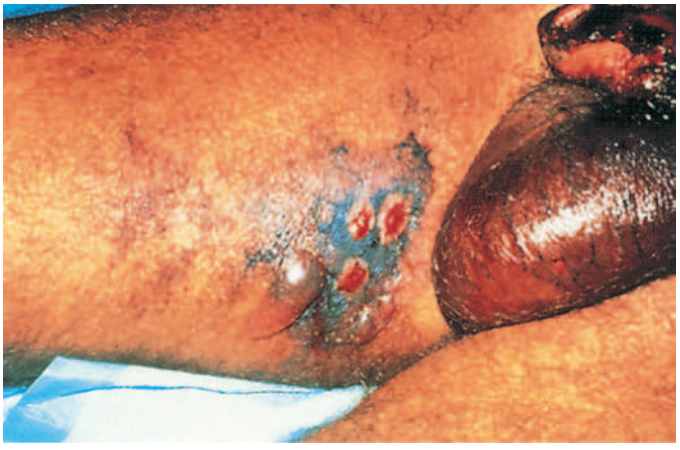

FIGURE 73-1 Ecthyma gangrenosum associated with *Vibrio vulnificus* sepsis. *(Courtesy Edward J. Bottone, MD, Department of Microbiology, Mt Sinai Hospital, New York.)*

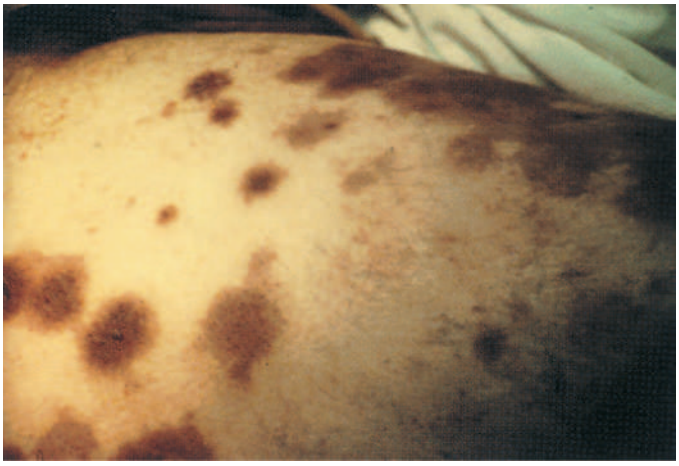

FIGURE 73-2 Torso of a victim with *Vibrio vulnificus* sepsis.

to rapid invasion of healthy tissue. *V. vulnificus* also produces a cytotoxin-hemolysin and phospholipases. Cytolysin produced by most pathogenic strains of *V. vulnificus* is extremely toxic to mice when injected intravenously and results in severe perivascular edema and neutrophil infiltration in lung tissues.[98,145] The precise roles of these and other factors (pili, mucinase, chondroitinase, hyaluronidase) in the in vivo pathogenicity of the organism have yet to be determined. Bleeding complications (which may include gastrointestinal hemorrhage and disseminated intravascular coagulation) are common and may be attributed in part to thrombocytopenia. Gastroenteritis is more common (15% to 20%) with the septicemic presentation than with primary wound infection and may exist as an isolated entity (≈ 10% of cases), although it is debated that illness has been erroneously attributed to the asymptomatically carried organism. *Vibrio vulnificus* endometritis has been reported after an episode of intercourse in the waters of Galveston Bay, Texas.[188] Another series of nine cases of *Vibrio* infections, in most cases the species being *vulnificus,* was reported associated with finning injuries of the hands.[50] Other presentations of *V. vulnificus* infections have included meningitis, necrotizing fasciitis following lightning strike in a windsurfer, spontaneous bacterial peritonitis, corneal ulcers, epiglottitis, and infections of the testes, spleen, and heart valves.[195]

The explosive nature of the syndrome can lead to gram-negative sepsis and death, reportedly in up to 50% of cases. The mortality rate may be as high as 90% in victims who become hypotensive within 12 hours of initial examination by a physician. For wound infections from all *Vibrio* species, the organism may only be recovered from blood specimens in less than 20% of victims.[87] Appropriate antibiotics should be administered as soon as the infection is suspected (see later). In one report, *V. vulnificus* sepsis was treated with antibiotic therapy, debridement of necrotic tissues, and direct hemoperfusion using polymyxin B immobilized fiber, which served as an artificial reticuloendothelial system and removed endotoxin from the circulating blood.[163] In an immunocompetent victim who acquired a *V. vulnificus* hand infection from peeling shrimp, treatment with oral ciprofloxacin was successful. In a series of seven patients treated for primary skin and soft tissue infections secondary to *V. vulnificus,* prompt operative exploration and debridement were correlated with a decrease in the intensive care unit and hospital length of stay, particularly if the surgery occurred within 72 hours from the time of infection.[83] The authors noted that all patients had necrosis of underlying subcutaneous tissue, whereas some did not demonstrate skin necrosis.

Vibrio mimicus

Vibrio mimicus is a motile, nonhalophilic, gram-negative, oxidase-positive rod with a single flagellum. It can be distinguished from *V. cholerae* by its inability to ferment sucrose, inability to metabolize acetylmethyl carbonyl, sensitivity to polymyxin, and negative lipase test. An ear infection may follow exposure to ocean water. Isolates are sensitive to tetracycline. Physicians who collect stool samples for culture to identify suspected *V. mimicus* must alert the laboratory to use appropriate culture media (thiosulfate-citrate–bile salts–sucrose [TCBS] agar).

Vibrio alginolyticus

Vibrio alginolyticus, found in seawater, has been implicated in soft tissue infections (e.g., those caused by coral cuts or surfing scrapes), sinusitis, and otitis, particularly after previous ear infections or a tympanic membrane perforation. Although bacteremia has been reported in immunosuppressed patients and patients with burns, *V. alginolyticus* does not generally carry the virulent potential of *V. vulnificus*.[128] Typical symptoms include cellulitis, with seropurulent exudate. Its distinguishing microbiologic features are stated to be sucrose and lactose fermentation, growth in 1% tryptone broth plus 10% NaCL, positive Voges-Proskauer reaction, negative urease reaction, and susceptibility to vibriostatic compound O/129. However, because these indices may vary, identification may be difficult. Antibiotic resistance may be a feature of *V. alginolyticus* infection.

Photobacterium damsela

Photobacterium (formerly *Vibrio* or *Listonella*) *damsela,* formerly enteric group EF-5 and so-named because it is pathogenic for the damselfish, causes wound infections similar to those attributed to vibrios. Rapidly progressive infection leading to muscle necrosis and fasciitis or to sepsis and death may transpire in an immunosuppressed victim or person with normal immunity.[72,74,185] This may be related to an extracellular cytolysin (damselysin) or other unidentified enzymes or virulence factors.

Vibrio cholerae

Vibrio cholerae is associated with severe gastroenteritis (see Chapter 82). With regard to tissue infection, a case of necrotizing fasciitis and septic shock caused by *V. cholerae* non-O1 (not agglutinated in cholera polyvalent O1 antiserum) acquired in San Diego, California, has been described.[202] The victim suffered from preexisting diabetes mellitus complicated by chronic plantar ulceration of the affected limb. *V. cholerae* may cause severe disease signs in Japanese sweetfish (ko-ayu or *Plecoglossus altivelis*), certain shrimp (e.g., *Penaeus monodon*), and ornamental fish in India.[13,82,182]

Growth in Culture

Although plating on standard clinical laboratory media may detect only 0.1% to 1% of the total number of microorganisms found in seawater or marine sediment, most marine bacteria that are pathogenic to humans can be readily recovered on standard media. Although pathogenic *Vibrio* species can grow on conventional blood agar media, other marine bacteria may require saline-supplemented media and incubation at 25° C (77° F) instead of the standard 35° to 37° C (95° to 98.6° F). In culture, marine bacteria may grow at a slower rate than terrestrial bacteria, which delays identification. Pleomorphism in culture may be attributed to adaptation to small concentrations of nutrients in seawater. Most organisms require sodium, potassium, magnesium, phosphate, and sulfate for growth; a few require calcium or chloride.

All *Vibrio* species grow in routine blood culture mediums and on nonselective mediums, such as blood agar. TCBS agar is selective and recommended for detection of marine *Vibrio* organisms, although cellobiose–polymyxin B–colistin (CPC) agar may be as good or better.[20,120] An alternative is Monsur taurocholate-tellurite-gelatin agar. A large clinical laboratory near the ocean might consider routinely using TCBS or CPC agar. Pathogenic vibrios generally grow on MacConkey agar. All species except *V. cholerae* and *V. mimicus* require sodium chloride for growth. Enrichment broth (alkaline peptone water with 1% NaCl) is recommended for isolation of vibrios from convalescent and treated patients. Another enrichment broth that may be more effective is 5% peptone, 1% NaCl, and 0.08% cellobiose (PNC) at pH 8.0.[91] A comparison of strategies for detection and recovery of *V. vulnificus* from marine samples of the western Mediterranean coast determined that the best strategy consisted of the

combination of culture-based methods (3-hour enrichment in alkaline-saline peptone water at 40° C [104° F], followed by culture on CPC agar) and DNA-based procedures (specific PCR amplification of the presumptive colonies with primers Dvu 9V and Dvu 45R).[6]

Key characteristics that aid in separation of *Vibrio* species from other medically significant bacteria (Enterobacteriaceae, *Pseudomonas, Aeromonas, Plesiomonas*) are motility by polar flagella, production of oxidase, fermentative metabolism, requirement of sodium chloride for growth, and susceptibility to the O/129 vibriostatic compound. *V. vulnificus* can be cultured from blood, wounds (bullae), and stool. The laboratory must be cautioned to use selective culture media with a high salt content (3% NaCl) for prompt identification. Suggestive features include positive fermentation of glucose, positive catalase and oxidase tests, positive indole test, positive reaction for both lysine and ornithine decarboxylase, positive *o*-nitrophenyl-β-D-galactopyranoside, and inability to ferment sucrose. A useful identification scheme for pathogenic *Vibrio* species is found in the chapter on *Vibrio* in the most recent edition of the American Society for Microbiology's *Manual of Clinical Microbiology*.

Because growth of *V. vulnificus* in culture generally requires 48 hours, current research is directed at a more rapid diagnostic test. Direct identification of *V. vulnificus* in clinical specimens by nested PCR has been accomplished using serum specimens and bulla aspirates from septicemic patients, as well as from fish, sediments, and water.[7,115] Simultaneous detection of five marine fish pathogens *(V. vulnificus, Listonella anguillarum, Photobacterium damsela, Aeromonas salmonicida,* and *V. parahaemolyticus)* has been accomplished using multiplex PCR and a DNA microarray.[73] This technique has not yet been applied to diagnosis of pathogens afflicting humans.

Mycobacteria must be cultured in media such as Middlebrook 7H10 or 7H11 agar or Lowenstein-Jensen medium; fungi require a medium such as Sabouraud dextrose or brain-heart infusion/Sabhi agar. Antibiotic susceptibility testing can be performed using established procedures, except for addition of NaCl 2.3% to the Mueller-Hinton broth or agar used for disk diffusion. Certain commercial test kits may not accurately identify marine organisms. In the setting of wound infection or sepsis, the clinician should alert the laboratory that a marine-acquired organism may be present. If a laboratory does not have time or resources to perform a complete identification, the bacteria may be sent to a reference laboratory. Marine bacteria are kept in the American Type Culture Collection. Because of the diversity of species, complete agreement has not yet been reached on comprehensive taxonomic criteria for identification.

Antibiotic Therapy

The objectives for management of infections from marine microorganisms are to recognize the clinical condition, culture the organism, and provide antimicrobial therapy. Management of marine-acquired infections should include therapy against *Vibrio* species. Antibiotic selection should be guided by the most current recommendations. Historically, third-generation cephalosporins (cefoperazone, cefotaxime, or ceftazidime) provide variable coverage in vitro; first- and second-generation products (cefazolin, cephalothin, cephapirin, cefamandole, cefonicid, ceforanide, or cefoxitin) appear to be less effective in vitro. The organism has been reported in some cases to be resistant in vitro to third-generation cephalosporins, mezlocillin, aztreonam, and piperacillin.[149] A combination of cefotaxime and minocycline seems to be synergistic and extremely effective against *V. vulnificus* in vitro.[56] Oral cultures taken from two captive moray eels at the John G. Shedd Aquarium in Chicago demonstrated *V. fluvialis, Photobacterium damsela, V. vulnificus,* and *Pseudomonas putrefaciens* to be sensitive to cefuroxime, ciprofloxacin, tetracycline, and trimethoprim-sulfamethoxazole.[69] Imipenem-cilastatin is generally efficacious against gram-negative marine bacteria, as are trimethoprim-sulfamethoxazole and tetracycline. Gentamicin, tobramycin, and chloramphenicol have tested favorably against *P. putrefaciens* and *Vibrio* strains. Nonfermentative bacteria (such as *Alteromonas, Pseudomonas,* and *Deleya* species) appear to be sensitive to most antibiotics. In a mouse model, combination therapy with minocycline and cefotaxime was more effective than either drug alone.[55]

Quantitative wound culture has no advantage before the appearance of a wound infection. Pending a prospective evaluation of prophylactic antibiotics in the management of marine wounds, the following recommendations are based on the indolent nature and malignant potential of soft tissue infections caused by *Vibrio* species:

Minor abrasions or lacerations (such as coral cuts or superficial sea urchin puncture wounds) do not require prophylactic antibiotics in the host with normal immunity. Persons who are chronically ill (as with diabetes, hemophilia, or thalassemia) or immunologically impaired (as with leukemia or acquired immunodeficiency syndrome [AIDS], or undergoing chemotherapy or prolonged corticosteroid therapy), or who suffer from serious liver disease (such as hepatitis, cirrhosis, or hemochromatosis), particularly those with elevated serum iron levels, should be placed immediately after the injury on a regimen of oral ciprofloxacin, trimethoprim-sulfamethoxazole, or tetracycline (or doxycycline), because these persons appear to have an increased risk of serious wound infection and bacteremia. Cefuroxime may be a useful alternative. Penicillin, ampicillin, and erythromycin are not acceptable alternatives.[135,137] Norfloxacin may be less efficacious against certain vibrios.[136,153] Other quinolones (ofloxacin, enoxacin, pefloxacin, fleroxacin, lomefloxacin) have not been extensively tested against *Vibrio;* they may be useful alternatives, but this awaits definitive evaluation. Appearance of an infection indicates the need for prompt debridement and antibiotic therapy. If an infection develops, antibiotic coverage should be chosen that will also be efficacious against *Staphylococcus* and *Streptococcus*, because these are still quite common perpetrators of infection. In general, the fluoroquinolones, which are particularly effective for treating gram-negative bacillary infections, may become less and less useful against resistant staphylococci.[192] If *Staphylococcus* is a β-lactamase–producing strain, a semisynthetic penicillin (nafcillin or oxacillin) should be chosen, with a cephalosporin such as cefazolin or cephalothin used if there is a history of delayed-type penicillin allergy. Vancomycin is recommended in the event of methicillin resistance.[122]

Serious injuries (from an infection perspective) include large lacerations, serious burns, deep puncture wounds, or a retained foreign body. Examples are shark or barracuda bites, stingray spine wounds, deep sea urchin punctures, scorpaenid (scorpionfish) spine envenomations that enter a joint space, and full-thickness coral cuts. If the victim requires hospitalization and surgery for standard wound management, recommended antibiotics include gentamicin, tobramycin, amikacin, ciprofloxacin, or trimethoprim-sulfamethoxazole. Cefoperazone and cefotaxime may or may not be effective. There is a recommendation in the literature advocating use of ceftazidime in combination with tetracycline (or doxycycline).[108] Chloramphenicol is an alternative agent less commonly used because of hematologic side effects. Imipenem-cilastatin or meropenem may be used in a circumstance of sepsis or treatment failure. Meropenem has shown excellent in vitro activity against the Vibrionaceae and may be useful for eradicating infections produced by these organisms.[57] Patients who simultaneously receive imipenem or ciprofloxacin and theophylline may have an increased tendency to seizures.[165] If the victim is managed as an outpatient, the drugs of choice to cover *Vibrio* are ciprofloxacin, trimethoprim-sulfamethoxazole, or tetracycline. Cefuroxime is an alternative. It is a clinical decision whether oral therapy should be preceded by a single IV or intramuscular (IM) loading dose of a similar or different antibiotic, commonly an aminoglycoside.

Infected wounds should be cultured for aerobes and anaerobes. Pending culture and sensitivity results, the patient should be managed with antibiotics as described previously. In a person who has been wounded in a marine environment and has rapidly progressive cellulitis or myositis, *V. parahaemolyticus* or *V. vulnificus* infection should be suspected, particularly in the presence of chronic liver disease. If a wound infection is minor and has the appearance of a classic erysipeloid reaction *(Erysipelothrix rhusiopathiae),* penicillin, cephalexin, or ciprofloxacin should be administered (see Chapter 76). *E. rhusiopathiae* attributed to a

pet goldfish has been reported to cause necrotizing fasciitis in a diabetic patient.[168] It is also cultured in episodes of infective endocarditis, although the origins of the infections are often not identified.[117,196]

If sepsis is severe, additional aggressive measures beyond surgery and antibiotics, such as administration of recombinant human activated protein C, may be required.[4] Hyperbaric oxygen therapy has been used as adjunctive therapy in *V. vulnificus* septicemia and cellulitis, but there is no standard recommendation for this modality for this indication.[203]

FRESHWATER BACTERIOLOGY

Diversity of Organisms

The natural freshwater environment of ponds, lakes, streams, rivers, lagoons, harbors, estuaries, and artificial bodies of water is probably as hazardous as the ocean from a microbiologic standpoint. Waterskiing accidents, propeller wounds, fishhook punctures, lacerations from broken glass and sharp rocks, fish fin punctures, and crush injuries during white-water expeditions are commonplace. A large number of bacteria have been identified in fresh water and associated sediments and animals. In fringe areas of the ocean that carry brackish water (NaCl content < 3%), marine bacteria, salt-tolerant freshwater bacteria, and brackish-specific bacteria, such as *Agrobacterium sanguineum,* are noted. The combined effects of human and animal traffic and waste disposal increase the risk for coliform contamination. In Great Britain, antibiotic-resistant *Escherichia coli* have been documented in rivers and coastal waters.[170] Coxsackievirus A16 has been isolated from children stricken ill after bathing in contaminated lake water.[64] Of particular note is the presence of virulent species, such as *Chromobacterium violaceum, V. parahaemolyticus,* and *A. hydrophila,* associated with serious and indolent wound infections.[201] The last can be cultured from natural bodies of water, as well as from the mouths of domesticated aquarium fish, such as the piranha.[158] Biologic control agents, such as guppy fish bred in wells to control mosquito proliferation, can carry bacterial pathogens, such as *Pseudomonas.*[49]

One investigation sampled water, inanimate objects, and animals from freshwater environments in California, Tennessee, and Florida.[10] Bacteria isolated were predominantly gram-negative and included *A. hydrophila, Flavobacterium breve, Pseudomonas* species, *V. parahaemolyticus, Serratia* species, *Enterobacter* species, *Plesiomonas shigelloides, Bacillus* species, *Acinetobacter calcoaceticus,* and *Alcaligenes denitrificans.*

Primary amebic meningoencephalitis is caused by infection with *Naegleria fowleri,* a thermophilic, free-living ameba found in freshwater environments. The infection is associated with human activities that allow entry of water into the nose, from where the ameba migrate to the brain via the olfactory nerve.

Wound Infections Caused by *Aeromonas* Species

Aeromonas hydrophila (Latin for "gas producing" and "water loving") is a gram-negative, facultatively anaerobic, polarly flagellated, non–spore-forming, motile rod and member of the family Vibrionaceae that commonly inhabits soil, freshwater streams, and lakes.[3,14,112,198] *Aeromonas* species are widely distributed and found at wide ranges of temperature and pH. Five species *(A. hydrophila, A. sobria, A. schubertii, A. veronii,* and *A. caviae)* of the nine that have been recovered from clinical material have been linked with human disease; there are 13 or more distinct genotypes.[1] *A. hydrophila* is pathogenic to amphibians, reptiles, and fish. Soft tissue and gastroenteric infections predominate in humans. Virulence factors elaborated by *Aeromonas* species include hemolysin, cytotoxin, enterotoxin, cholera toxin–like factor, and hemagglutinins.[27,197] *Aeromonas* species are sometimes misidentified as members of the genus *Vibrio* by commonly used screening tests.[1]

A wound, particularly of the puncture variety, immersed in contaminated water may become cellulitic within 24 hours, with erythema, edema, and a purulent discharge.[166,211] The lower extremity is most frequently involved. This usually occurs from stepping on a foreign object or being punctured underwater. The appearance may be indistinguishable from typical streptococcal cellulitis, with localized pain, lymphangitis, fever, and chills. Untreated or managed with antibiotics to which the organism is not susceptible, this may rarely progress to a severe gas-forming soft tissue reaction, bulla formation, necrotizing myositis, or osteomyelitis. An appearance similar to ecthyma gangrenosum caused by *Pseudomonas aeruginosa* has been reported in *Aeromonas* septicemia.

Fever, hypotension, jaundice, and chills are common manifestations of septicemia.[105] Additional clinical manifestations include abdominal pain or tenderness, altered consciousness, acute renal failure, bacteremic pneumonia, and coagulopathy.[99] In a manner analogous to the pathogenicity of virulent *Vibrio* species, the chronically ill or immunocompromised host (e.g., chronic liver disease, neoplasm, diabetes, uremia, corticosteroid therapy, extensive burns) is probably at greater risk of a severe infection or complication, such as meningitis, endocarditis, or septicemia.[96] Freshwater aspiration may result in *A. hydrophila* pneumonitis and bacteremia.

There are numerous case reports of infections linked to *Aeromonas* species. Infection has been reported following the bite of an alligator. A 15-year-old boy suffered an *A. hydrophila* wound infection after a bite from his pet piranha. For unknown reasons, there is a marked preponderance of male victims. This may represent the phenotypic variation of critical bacterial adhesins or more likely may simply reflect activity patterns of male humans. Corneal ulcer caused by *A. sobria* was reported after abrasion by a freshwater reed.[47] Medicinal leeches can harbor *Aeromonas* in their gut flora; soft tissue infections related to this phenomenon have been reported.[175] The genus *Plesiomonas* also belongs to the family Vibrionaceae; it has been linked to aquarium-associated infection complicated by watery diarrhea and fever.

Because of the microbiologic similarity of *Aeromonas* on biochemical testing to members of the Enterobacteriaceae, such as *E. coli* or *Serratia* species, it is important to alert the laboratory to the clinical setting. In the microbiology laboratory, *Aeromonas* species may be identified on the basis of positive oxidase reaction, no growth on TCBS agar, growth on MacConkey agar, and resistance to the vibriostatic compound O/129.[105]

Gram's stain of the purulent discharge may demonstrate gram-negative bacilli, singly, paired, or in short chains. Given the appropriate clinical setting (after a wound acquired in the freshwater environment), this should not be casually attributed to contamination.[98] *A. hydrophila* is generally sensitive to chloramphenicol, aztreonam, gentamicin, amikacin, tobramycin, trimethoprim-sulfamethoxazole, cefotaxime, cefuroxime, moxalactam, imipenem, ceftazidime, ciprofloxacin, and norfloxacin. For a severe infection, initial therapy that includes an aminoglycoside provides coverage against concomitant *Pseudomonas* or *Serratia* infection. As has been demonstrated with *Vibrio* species, first-generation cephalosporins, penicillin, ampicillin, and ampicillin-sulbactam are not efficacious, perhaps because of production of β-lactamase by the organism. *Aeromonas* species are capable of producing chromosomally encoded β-lactamases induced by β-lactam antibiotics. This leads to resistance to penicillins, cephalosporins, and monobactams. The β-lactamase inhibitors, such as clavulanate, are not effective against these β-lactamases, so that amoxicillin-clavulanate may not kill *Aeromonas.*[146] The optimal therapy for invasive infections caused by cefotaxime-resistant *A. hydrophila* is not known, but a recent study of in vitro and in vivo (mice) activities of fluoroquinolones suggest that ciprofloxacin may be as effective as cefotaxime–minocycline.[104] In this study, ciprofloxacin and levofloxacin showed greater activity than did gatifloxacin, moxifloxacin, and lomefloxacin.

Initial therapy of a severe soft tissue infection related to *Aeromonas* should include aggressive wound debridement to mitigate the potentially invasive nature of the organism. In one case of severe cellulitis unresponsive to debridement, fasciotomy, and antibiotic therapy, treatment with hyperbaric oxygen was felt to contribute to successful infection control.[127]

Infections Caused by a Fish Pathogen, *Streptococcus iniae*

Streptococcus iniae is a pathogen of fish, noted to cause subcutaneous abscesses in Amazon freshwater dolphins (Mammalia:

Inia geoffrensis) kept in captivity.[207] Epizootic fatal meningoencephalitis in fish species caused by streptococci has been observed in outbreaks affecting tilapia, yellowtail, rainbow trout, and coho salmon. *S. iniae* has emerged as a serious pathogen of farmed barramundi in Australia.[29] Persons who handle these fish are at risk for bacteremic illness, which can manifest as cellulitis or sepsis. Endocarditis, meningitis, and arthritis have been noted to accompany *S. iniae* infection.

The tilapia (*Oreochromis* and *Tilapia* species) are also known as St Peter's fish or Hawaiian sunfish. The surfaces of these commonly aquacultured fishes may be colonized with *S. iniae*. Persons of Asian descent have been identified as prone to infection, probably because they often prepare this fish with the intent to dine. Typically, the victim recalls puncturing the skin of the hand with a fin, bone, or implement of preparation. Cellulitis with lymphangitis and fever is common, without skin necrosis or bulla formation.[207]

In culture, *S. iniae* shows β-hemolysis. However, it may appear to be α-hemolytic because the narrow zone of β-hemolysis is ringed by a more prominent zone of α-hemolysis. Therefore, it may be misidentified as a *viridans* streptococcus and thus considered a contaminant. A reasonable approach to antibiotic therapy includes penicillin, cefazolin, ceftriaxone, erythromycin, clindamycin, or trimethoprim-sulfamethoxazole. In one series, ciprofloxacin showed slightly less efficacy in vitro.

Infection Caused by *Desmodesmus armatus*

Two cases of soft tissue infection cause by the chlorophyll-containing alga *Desmodesmus armatus* were reported.[110a] In immunocompetent adults sustaining a deep puncture wound to the foot in one case and open fracture-dislocation of the knee in the other case, infection investigated by fungal cultures of the soft tissues revealed green colonies consistent with a chlorophyllic organism that was identified as *D. armatus*. Both patients underwent surgical debridement and healed without recurrence. In these cases, no antifungal drug therapy was administered.

A General Approach to Antibiotic Therapy

Management of infections acquired in fresh water should include therapy against *Aeromonas* species. First-generation cephalosporins provide inadequate coverage against growth of fresh water bacteria. Third-generation cephalosporins provide excellent coverage, whereas second-generation products are less effective. Ceftriaxone may not be efficacious against *Aeromonas* species. Ciprofloxacin, imipenem, ceftazidime, gentamicin, and trimethoprim-sulfamethoxazole are reasonable antibiotics against gram-negative microorganisms. Trimethoprim or ampicillin alone may be inefficacious.

Whether to begin antimicrobial therapy before establishment of a wound infection is controversial. Pending prospective evaluation of prophylactic antibiotics in wounds acquired in fresh water, the following recommendations are based on the potentially serious nature of soft tissue infections caused by *Aeromonas* species:

Minor abrasions or lacerations do not require the administration of prophylactic antibiotics in the host with normal immunity. Persons who have chronic illness, immunologic impairment, or serious liver disease, particularly those with elevated serum iron levels, should be placed immediately on a regimen of oral ciprofloxacin or norfloxacin (first choice), trimethoprim-sulfamethoxazole (second choice), or doxycycline-tetracycline (third choice; use only in the setting of allergy to the first two choices, because resistance has been observed), because these persons appear to have an increased risk of serious wound infection and bacteremia. Penicillin, ampicillin, erythromycin, and trimethoprim do not appear to be acceptable alternatives. The appearance of an infection indicates the need for prompt debridement and antibiotic therapy. If an infection develops, antibiotic coverage that will also be efficacious against *Staphylococcus* and *Streptococcus* should be chosen, because these are likely the most common perpetrators of infection.

If the victim requires surgery and hospitalization for wound management, recommended antibiotics include ciprofloxacin, gentamicin, or trimethoprim-sulfamethoxazole. Imipenem-cilastatin is an extremely powerful antibiotic that should be used in a circumstance of sepsis or treatment failure. If the victim is to be managed as an outpatient, the oral drug of choice is norfloxacin (or ciprofloxacin), trimethoprim-sulfamethoxazole, or tetracycline or doxycycline. It is a clinical decision whether oral therapy should be preceded by a single IV or IM loading dose of a similar or different antibiotic, commonly an aminoglycoside.

Infected wounds should be cultured. Pending culture and sensitivity results, the patient should be managed with antibiotics as outlined previously. If fever or rapidly progressive cellulitis characterized by bullae and large areas of necrosis develops, *A. hydrophila* infection should be suspected. Less rapidly progressive *Aeromonas* infections may have the appearance of streptococcal cellulitis.[100]

SHARKS

Myth and folklore surround sharks, the most feared of sea creatures. Although dreaded, sharks are among the most graceful and magnificent denizens of the deep (Figures 73-3 and 73-4). Sharks may be found in all seas but the Southern Ocean and occur in some tropical rivers and riverine lakes. Sharks range in size from the dwarf and cylindrical dogfishes *Etmopterus perryi* and *E. carteri* (21 cm [8.3 inches]) to the plankton-feeding whale shark *Rhincodon typus* (17-21 m [56-69 feet] and 22,700 kg [50,000 lb]) (Figures 73-5 and 73-6).

Attacks by these occasionally savage animals have always held enormous fascination for scientists, adventurers, and clinicians. The problem was highlighted for the U.S. military in 1945 during World War II, when crew members from the USS *Indianapolis* perished in shark-inhabited waters. On July 30, the heavy cruiser was sunk by a Japanese torpedo, resulting in hundreds of deaths plus reports of an estimated additional 60 to 80 shark attack fatalities of survivors left adrift for 5 days. This estimate is certainly a high figure inflated by historical misinterpretation of scavenge bites on already dead servicemen and some exaggeration, but nevertheless, the approximately two dozen bites on live and dying seamen involved in this tragedy represents the largest documented incidence of mass shark attacks (GHB interviews with survivors).

The bull shark *Carcharhinus leucas* (Figure 73-7) is a frequent visitor and occasional resident of tropical and warm-temperate rivers and a regular denizen of reefs and other nearshore marine habitats. It commonly penetrates 161 km (100 miles) or more up freshwater rivers, such as the Ganges, Nile, and Zambezi, and

FIGURE 73-3 Schooling sharks. *(Copyright Stephen Frink.)*

FIGURE 73-4 Caribbean reef sharks. *(Courtesy Jennifer Hayes.)*

has been seen in the Amazon River at Iquitos, Peru, 4000 km from the Atlantic Ocean, and as far inland as Illinois in the Mississippi River.[140,187] These sharks also live in Lake Nicaragua and have had their aggressive behavior attributed in part to high levels of testosterone. During the summer of 2001, the "Summer

FIGURE 73-5 Whale shark. *(Copyright Stephen Frink.)*

FIGURE 73-6 Snorkeler soars above a whale shark. *(Copyright Carl Roessler.)*

of the Shark" in the U.S. media, a bull shark was believed to have attacked an 8-year-old child off the Gulf Coast of Florida.

The International Shark Attack File (ISAF) has its origin from the Shark Research Panel created by the Office of Naval Research (ONR) in 1958. The File was initiated by Perry W. Gilbert and Leonard P. Schultz in 1958 for the Smithsonian Institution, American Institute of Biological Sciences, Cornell University, and the ONR. In 1967, the data were sent to Mote Marine Laboratory in Sarasota, Florida, where H. David Baldridge analyzed 1165 reported attacks and case histories and prepared a special technical report, "Shark Attack Against Man," for the U.S. Navy Bureau of Medicine and Surgery in 1973. After a period of maintenance at the National Underwater Accident Data Center at the University of Rhode Island, in 1988 the Shark Attack File moved to the University of Florida at Gainesville, where it is maintained by the Florida Museum of Natural History under the direction of George H. Burgess.[36] It remains an authoritative collection of analyzed data, containing a series of more than 6000 individual investigations from the mid-1500s to the present. Smaller regional scientific records of shark attacks are maintained by the Taronga Zoo (Australia), California Department of Fish and Game, Hawaii Department of Land and Natural Resources, Natal Sharks Board (South Africa), and University of Sao Pãulo (Brazil). All of these organizations serve as cooperators with ISAF, feeding results of regional investigations into the ISAF database. Hundreds of cooperating scientific observers located throughout the world act in a similar capacity, ensuring broad international coverage.

The world's shark populations are in danger from overfishing, particularly in light of their slow growth rate, late sexual maturation, relatively lengthy gestation periods, and low number of offspring. Each year, 26 to 73 million sharks representing 1.21 to 2.29 million metric tons (2.7 to 5 billion pounds), or about 10 million sharks for each shark-related human fatality, are killed in

FIGURE 73-7 Bull shark. *(Courtesy Marty Snyderman.)*

FIGURE 73-8 Shark slaughter. *(Copyright Stephen Frink.)*

fisheries.[59] One-half of this total represents incidental bycatch (nontargeted captures in fishing nets or on longlines fishing for other species).[23] The National Marine Fisheries Service estimates that 20 million metric tons of marine wildlife are killed and thrown back into the sea as bycatch (Figure 73-8). Such non-landed bycatch likely increase the above estimate of shark deaths, which was calculated using East Asian shark fin–trade landings. Sharks killed by commercial fishing in U.S. waters average 20,000 metric tons (44,092,000 lb) per year. Great declines in shark populations along the eastern coast of the United States occurred over the last two decades of the 20th century (now moderated through U.S. fishery regulation), but declines continue through much of the (unregulated) world. Some commercially targeted species declined by as much as 80%; the most dramatic declines were seen in dusky sharks. The flesh of most sharks is deemed of low value in American markets, and innumerable animals were ground into fertilizer or simply discarded at sea.

The fishery interest in sharks centers on the fins, including those of the blue, hammerhead (Figures 73-9 to 73-11), silky (Figure 73-12), mako, and thresher sharks. These are of great value in Eastern Asia, where they are made into shark fin soup, a traditional dish that signifies high economic status and is reputed to be an aphrodisiac. Interest in fins has also spawned the heinous and wasteful practice of finning, in which a shark is captured, its fins are sliced off (Figures 73-13 to 73-15), and then it is returned to the water (Figure 73-16).[124] Shark fin soup, which dates back from the Chinese Sung Dynasty in 960 AD, is sold for upward of $150 per bowl. The prepared fins themselves may sell for more than $800 per pound. It has been estimated that 350 tons of shark fins may be consumed each year. The

FIGURE 73-9 Hammerhead shark in Cocos Island. *(Copyright Carl Roessler.)*

FIGURE 73-10 Hammerhead shark. *(Copyright Stephen Frink.)*

FIGURE 73-11 Hammerhead shark. *(Copyright Stephen Frink.)*

International Commission for the Conservation of Atlantic Tunas (ICCAT) created a ban on shark finning in November 2004, to join the United States, which banned shark finning in the Atlantic Ocean in 1993 and in the Pacific Ocean in 2002, in such a prohibition. Shark flesh is a major food source in both developed countries (commonly the fish in European "fish and chips") and undeveloped countries (artisanal fisheries). Mako shark flesh is similar to that of swordfish and often serves as a more than adequate culinary substitute. Sharks do not routinely appear to carry ciguatera toxin, except in the liver; however, serious poisoning from shark ingestion has been reported (see Chapter 77).[28]

In Tahiti and some other Polynesian locations, sharks are occasionally mercilessly killed simply to acquire their teeth for jewelry manufacture. The great white shark has been declared a protected species in South Africa, Australia, the Maldives, California, and the Atlantic waters of the United States. In 1991, the South African government declared the great white shark (Figures 73-17 to 73-20) a protected species within 322 km (200 miles) of

FIGURE 73-12 Silky shark. *(Courtesy Marty Snyderman.)*

FIGURE 73-13 Finning a shark. *(Courtesy Marty Snyderman.)*

FIGURE 73-15 Shark that has been finned and discarded. *(Copyright Nobert Wu:* norbertwu.com.*)*

FIGURE 73-17 Great white shark shows its battle scars. *(Copyright Peter Riekstens.)*

FIGURE 73-14 Shark finning. *(Copyright Howard Hall.)*

FIGURE 73-16 This dead shark is a victim of finning. *(Courtesy Marty Snyderman.)*

FIGURE 73-18 Great white shark. *(Copyright Stephen Frink.)*

FIGURE 73-19 Great white shark approaches. *(Courtesy Paul S. Auerbach, MD.)*

its coast. The U.S. government has lowered allowable shark fishing quotas in Atlantic waters, and Australia closely monitors its shark fisheries. In October 2004, a global wildlife treaty by the Convention on International Trade in Endangered Species (CITES) offered new protection to great white sharks. In other areas, however, sharks and their relatives are largely unregulated and populations are in serious decline. Some species, especially those that enter rivers, are potentially at risk of extinction.

Of note is the failure of designation of "no-take" areas within marine coral reef ecosystems to demonstrably diminish the depletion of reef shark species, such as the whitetip reef shark and grey reef shark (Figure 73-21) in Australia, as opposed to no-entry zones. The latter are aerially surveyed and strictly enforced exclusion areas. Anything less appears to allow widespread predation by humans upon sharks.[161] Poaching and underreporting of catches are rampant.

LIFE AND HABITS

Sharks (from *xoc,* pronounced "shock," a Yucatee word from the Mayan language and a glyph for "fish") have inhabited the oceans for at least 400 million years.[24] They appeared on the planet during the Devonian period, approximately 200 million years before the dinosaurs. Indeed, many living species of sharks belong to the same genera as species from the Cretaceous period, one hundred million years ago.[177] Ancestral sharks may have been enormous; *Carcharocles megalodon,* which inhabited the

FIGURE 73-20 Great white shark. *(Copyright Howard Hall.)*

FIGURE 73-21 Grey reef shark. *(Copyright Stephen Frink.)*

seas between 20 million and 1.5 million years ago, probably grew to a length of more than 15 m (49 feet), with teeth longer than 15.2 cm (6 inches). This was a predator of astronomic proportions that apparently largely fed on whales and manatees.

Shark attack is perhaps first depicted on a vase dated circa 725 BC from the Island of Ischia west of modern-day Naples, and it is recorded in early Greek literature. Some 35 of about 375 species of sharks have been implicated in the 75 shark attacks on humans that currently occur annually worldwide (on-average, 65 attacks per year were recorded by the ISAF in the first decade of the 21st century), and another 35 to 40 species are considered potentially dangerous.[86] It is often opined that shark attacks may be underestimated, largely because of failure to report.[189] Even if this is the case in a world made increasingly smaller by mobile phones, the Internet, and social media, it is unlikely that a completely accurate estimation would change the epidemiologic significance, compared with other causes of water-related deaths.

U.S. coastal waters typically are the setting for one-third or more of the annual number of shark attacks. The great American colonial painter John Singleton Copley's 1778 painting *Watson and the Shark* (Figure 73-22), which depicts an encounter between the Englishman Brook Watson (1735-1807) and a shark that bit off his right foot in Havana Harbor in 1749, is one of the earliest authenticated records of a shark attack.[24] ISAF has records of attacks going back into the mid-1500s. ISAF data document an average of six deaths per year during the past decade, with a long-term trend of a declining number of fatalities over the last 11 decades. Of the 72 unprovoked shark attacks worldwide in 2014 (dropping from 75 in 2013 and 83 in 2012), 63% of those attacks occurred in North American waters but none of the three worldwide fatalities occurred in the region.[33] The worldwide

FIGURE 73-22 *Watson and the Shark,* by John Singleton Copley, 1778. *(Copyright 2005 Museum of Fine Arts, Boston.)*

FIGURE 73-23 Tiger shark. *(Copyright Stephen Frink.)*

shark attack fatality rates have declined from over 50% in the early 20th century to 7% in the first decade of this century, largely reflective of advances in medical and first-responder capabilities and beach safety practices.[86] Greater institutional capacity in the United States results in significantly lower mortality rates than those in other areas of the world.

The most frequently documented offenders are three larger animals: the great white, bull, and tiger *(Galeocerdo cuvier)* (Figure 73-23) sharks. All three reach large sizes, routinely seek larger prey items, and have broadly serrated teeth that facilitate shearing. Bull sharks are perhaps the species of greatest concern owing to their habitat preference (inshore waters, including estuaries and rivers, which places them close to human activity) and tenacious mode of attack.[38,46,61] The species is certainly underrepresented in attribution statistics because its distributional range overlaps those of many species similar in appearance. An outbreak of shark attacks off Pernambuco, Brazil, largely involved this species.[85] However, blacktip *(Carcharhinus limbatus)* and possibly spinner *(Carcharhinus brevipinna)* sharks, which are thought to be involved in most of Florida's numerous (15 to 20 per year) minor bites, may be involved in even more incidents.[36] Identification of attacking species is a difficult task because victims seldom see the attacker well enough to make an accurate identification and because identification of most shark species, especially requiem sharks of the family Carcharhinidae, is notoriously difficult, even for trained scientists. Definitive identification can be made by examining tooth fragments left behind in a wound, but this is an infrequent occurrence. Thus, easily identified species, such as white, tiger, and nurse sharks, sit high on documented attacker lists, whereas most requiem shark incidents are grossly underreported. Less commonly reported attackers include the sand tiger (ragged tooth) (Figure 73-24), blue (Figure 73-25), blacktip reef (Figure 73-26), bronze whaler, lemon

FIGURE 73-24 Sand tiger shark. *(Copyright 2011 Norbert Wu: norbertwu.com.)*

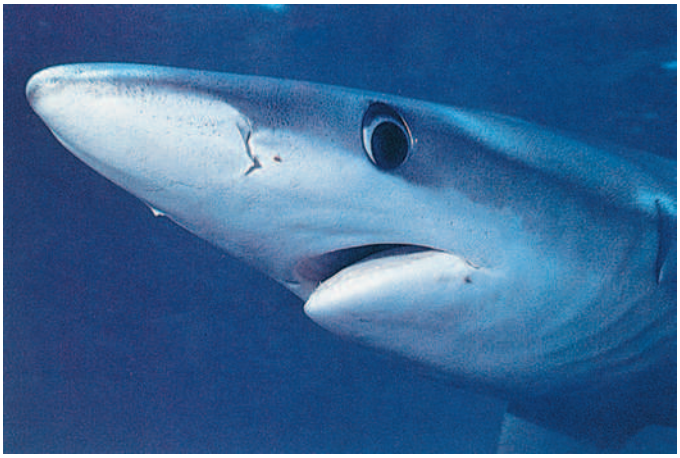

FIGURE 73-25 Blue shark, considered a dangerous species. *(Courtesy Marty Snyderman.)*

(Figures 73-27 to 73-29), shortfin mako (Figure 73-30), grey reef (Figure 73-31), oceanic whitetip (Figures 73-32 and 73-33), sandbar, sevengill, Caribbean reef, and dusky sharks (ISAF data). Hammerhead (Figure 73-34), Galápagos, and nurse (Figure 73-35) shark attacks are rarely reported. The famous series of attacks along the New Jersey shore in the summer of 1916 are thought to be attributable to a single great white shark. Tiger sharks are the most commonly identified attackers in the Hawaiian Islands and other tropical regions. Most attacks are reported from North America, with Florida (and its central east coast)

FIGURE 73-26 Blacktip reef shark. *(Copyright Stephen Frink.)*

FIGURE 73-27 Lemon shark. *(Courtesy Howard Hall.)*

FIGURE 73-28 Lemon shark. *(Copyright Stephen Frink.)*

FIGURE 73-29 Lemon shark at dusk. *(Copyright David Doubilet.)*

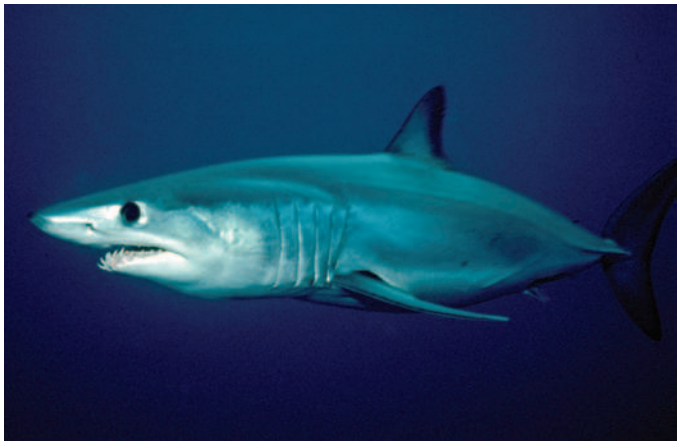

FIGURE 73-30 Mako shark. *(Courtesy Marty Snyderman.)*

FIGURE 73-31 Grey reef shark. *(Copyright Stephen Frink.)*

FIGURE 73-32 Oceanic whitetip shark, *Carcharhinus longimanus*. *(Copyright 2000 Norbert Wu:* norbertwu.com.*)*

FIGURE 73-33 Oceanic whitetip shark. *(Copyright Carl Roessler.)*

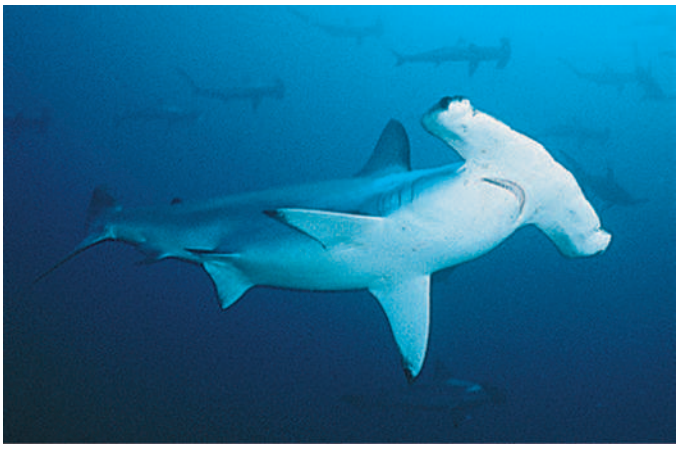

FIGURE 73-34 Hammerhead sharks, schooling off Cocos Island. The positioning of the eyes is reputed to increase the peripheral vision of these apex predators. *(Courtesy Howard Hall.)*

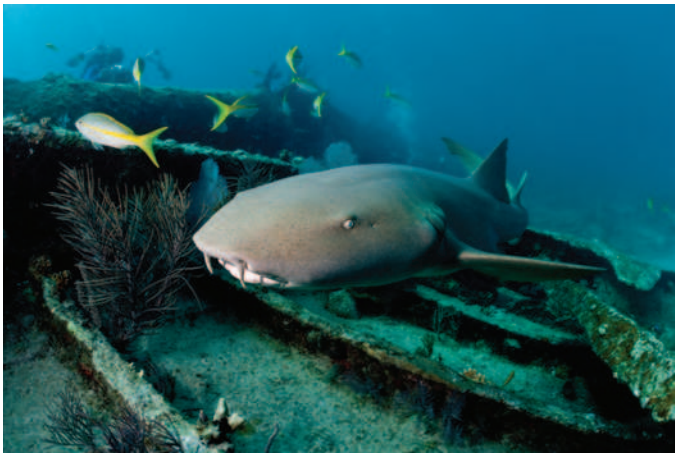

FIGURE 73-35 Nurse shark. *(Copyright Stephen Frink.)*

leading the list. A "leveling off" of annual shark attack numbers during the past decade suggests that it is possible that people are becoming a bit more intelligent about when and where they enter the water.

Sharks are carvivorous; many are apex predators. Their danger to humans results from the combination of size and dentition. Some species are also aggressive. The three largest sharks, the whale shark (Figure 73-36) (the largest fish at 15.2 m [50 feet] in

FIGURE 73-36 Whale shark *(Rhincodon typus)*, the largest fish in the sea, is fortunately a plankton eater. *(Copyright 2000 Norbert Wu: norbertwu.com.)*

FIGURE 73-37 Basking shark. *(Copyright 2011 Norbert Wu: norbertwu.com.)*

length and more than 18,181 kg [40,000 lb]), basking shark (Figure 73-37), and megamouth shark, eat plankton and use their gill rakers as filters. Even small sharks may have powerful jaws and sharp teeth. Any species reaching a length of 2 m (6.5 feet) must be considered potentially dangerous because its dentition and jaw strength at that size can inflict serious injury on a human. White shark attacks are most common in the waters of southern Australia, the south coast of South Africa, the middle Atlantic coast of North America, and the American Pacific coast north of Point Conception, California. Attacks by great white sharks, which reach a length of nearly 6 m (20 feet) (making it the largest predatory shark), off the coast of northern California have led to the designation of a "red [or bloody] triangle" bordered on the north by Point Reyes and Tomales Bay, through the Farallon Islands to the west, and down south to Año Nuevo and Point Sur facing the Monterey Bay. This is a breeding area for elephant seals *(Mirounga angustirostris)* (Figure 73-38), which yield 91-kg (200-lb) pups, perfect food for the immense predators. In an

FIGURE 73-38 The elephant seal, shown here swimming through a kelp bed, is a favorite food for the great white shark. *(Courtesy Howard Hall.)*

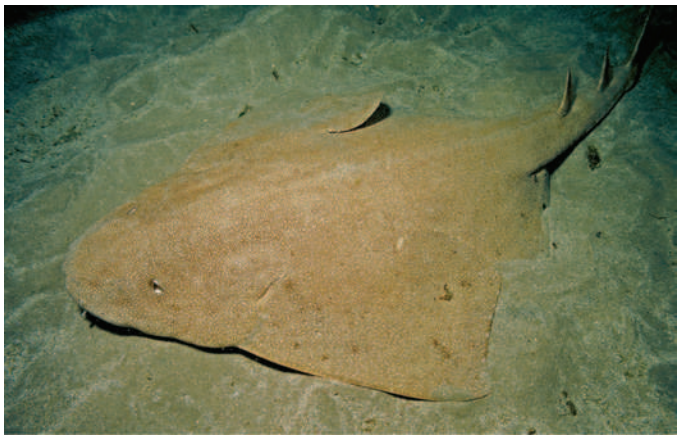

FIGURE 73-39 Angel shark. *(Courtesy Marty Snyderman.)*

analysis of California attacks, Miller and Collier attributed unprovoked attacks north of San Miguel Island to white sharks, whereas those south of this area involved members of the families Carcharhinidae (requiem sharks), Sphyrnidae (hammerheads, which can grow to more than 5.5 m [18 feet] in length), and possibly Squatinidae (angel sharks) (Figure 73-39).[133] Great white sharks follow established migration patterns and so are seen with regularity in certain locations, such as Guadalupe Island, 232 km (145 miles) off the coast of Mexico's Baja peninsula (Figure 73-40). In this instance, they have followed schools of tuna and reside from September to December with Guadalupe fur seals *(Arctocephalus townsendi)* (Figure 73-41), northern elephant seals, and California sea lions *(Zalophus californianus).*

The white, tiger *(Galeocerdo cuvier)* (≤ 5.5 m [18 feet] and 909 kg [2000 lb]) (Figure 73-42), and bull *(C. leucas)* (3.5 m [11.5 feet]; 364 kg [800 lb]) sharks are considered the most dangerous with regard to attacks on humans. The great hammerhead *(Sphyrna mokarran)* (Figure 73-43) shark has a reputation as an attacker in equatorial waters, but its reputation is overstated, probably because of its appearance. The hammerhead has ampullae scattered over its entire undersurface that are sensitive to electromagnetic fields, which in combination with its highly developed sense of smell, lateral eye placement (Figure 73-44), and maneuverability gained by its cephalofoil head shape make this shark a superior predator. In the pelagic realm, the oceanic whitetip shark is to be respected because of its aggressive nature. This species was implicated in many of the attacks and scavenge bites on survivors of the USS *Indianapolis* and in 2010 was involved in a pair (possibly trio) of severe Red Sea attacks, including a fatality.

FIGURE 73-40 A massive great white shark photographed near Guadalupe Island, Mexico. *(Copyright Peter Riekstens.)*

FIGURE 73-41 Bull Guadalupe fur seal. *(Courtesy Marty Snyderman.)*

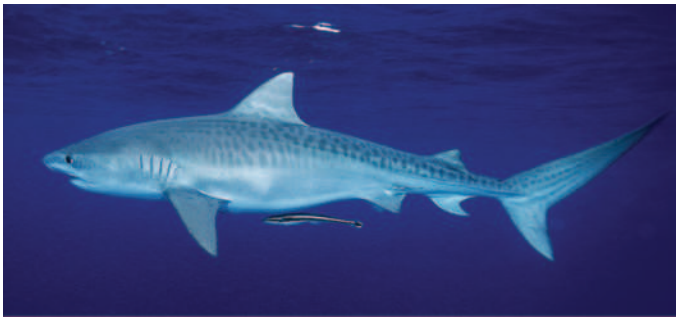

FIGURE 73-42 Tiger shark. *(Courtesy Marty Snyderman.)*

FIGURE 73-43 The great hammerhead *(Sphyrna mokarran).*

FIGURE 73-44 Lateral eye placement of the hammerhead shark. *(Courtesy Marty Snyderman.)*

FIGURE 73-45 Blue shark bites a mackerel, demonstrating the nictitating membrane that protects the eyes. *(Courtesy Marty Snyderman.)*

Sharks are members of the class Chondrichthyes (subclass Elasmobranchii), or cartilaginous (skeleton) fishes, which also includes skates, rays, and chimaeras.[48] Unlike many bony fish, sharks do not possess swim bladders for flotation, so in most species the large liver, which contains the oil squalene, contributes to their limited buoyancy. Thus, most sharks must stay in nearly constant motion to keep from sinking and to drive oxygenated water past the gills. Only a few groups of bottom-dwelling species rest for extended periods of time. Although sharks are not highly intelligent, they are endowed with remarkable sensory systems and well-developed sensory lobes of the brain. These systems allow the shark to locate struggling fish, swimmers, or divers. Their color vision is poor but well compensated for by the acute perception of motion and contrast; the eye musculature is adapted for fixation with any body motion. Whether or not sharks are attracted to certain colors, such as international orange, is not entirely determined, but research biologists refer to this color as "yum-yum yellow." The empirical observation is that sharks seem to be attracted to contrasting colors. The tapetum lucidum ("bright tapestry," responsible for the eye shine seen at night) is a series of reflecting plates containing silver guanine crystals in the choroidal layer behind the retina, which reflects light from a photoreceptor back along the same optical path to restimulate the rods and cones and thereby increase sensitivity of the eye. This is present in sharks that are active in low-light environments.[186] The tapetum also functions to shield the retina from bright light near the surface by manipulating pigment granules. The eyes of many species are protected by upper and lower eyelids and the nictitating membrane (Figures 73-45 and 73-46). The great white shark, which does not have the membrane, rotates its eyes in the sockets to avoid injury. Keen olfactory and gustatory chemoreceptors permit taste and the recognition of blood, urine, or peritoneal fluid in the water (in some cases, one part blood in 100 million parts of water, or concentrations of fish flesh in dilutions of one part in 10 billion parts of seawater). Sharks are most sensitive to chemicals that are similar to those produced by normal prey, such as amino acids, amines, and small fatty acids.[138] The nostrils are located on the underside of the snout just in front of the mouth. They open into sacs lined with folds of tissue containing cells used to detect smells. The odor-detecting system is constantly bathed by currents of water in both the resting and moving states.

FIGURE 73-46 Shark protects its eyes. *(Copyright Stephen Frink.)*

Sharks have relatively large brains for their body size. Up to two-thirds of the shark brain can be devoted to smell. The nostrils are the openings of the olfactory organs and do not take part in breathing, which is accomplished by oxygen extraction from the water passing over gill filaments, exiting through a series of 10 to 14 gill slits (five to seven per side). Additionally, sharks possess skin chemoreceptors that detect chemical irritants. The lateral line organs are small openings along the sides of the shark's body that register motion in the water. The lateral line system extending from the back along the side to the tail responds to sonic vibrations or pulsed low-frequency (20 to 60 cycles/sec; < 800 Hz) sound waves.[48] Perhaps the most ornate series of telereceptors is located about the head, within the jelly-filled ampullae of Lorenzini, which are extremely sensitive to electrical voltage gradients (generated by the muscle contractions of fish; detection down to a $1/10^6$ volt applied across a centimeter of seawater). Continuing research is directed at delineating the piscine ability to recognize electric fields. The common smooth dogfish *(Mustelus canis)* can detect an electrical voltage gradient of 5/1000 of a microvolt, whereas the brains of certain sharks can discern 15-billionths of a volt.[71] Sharks also have extremely sensitive hearing, which may detect prey underwater from a distance of 914 m (3000 feet). Hammerhead sharks have a laterally expanded skull (cephalofoil), which spreads sensory receptors over a wider area and perhaps provides anterior lift during swimming.

Shark skin *(shagreen)* is composed in part by placoid scales (dermal denticles). These microscopic appendages have the same origin as teeth, with a pulp cavity, dentine, and vitreodentine (enameloid) covering. The denticles are embedded in the skin and mouths of sharks and rays, and protect them from predators and parasites, reduce mechanical abrasion to the animal, accommodate sensory organs, and minimize swimming-induced drag.[176] Some sharks can alter their coloration because melanin in the skin darkens near the ocean surface and lightens at depth distant from sunlight. Scale-rasping behavior has been demonstrated in the juvenile lesser spotted dogfish *(Scyliorhinus canicula),* which uses this body armor to anchor food items near its tail in order to allow bite-sized pieces to be torn away by rapid jaw and head movements.

Research has been conducted on isolation of potential antineoplastic agents from shark cartilage, organs, and body fluids.[54,116,148,150] For instance, sphyrnastatins have been isolated from the hammerhead shark *Sphyrna lewini*.[150] Squalamine, produced by the spiny dogfish, is a low-molecular-weight aminosterol as well as a broad-based antibiotic with antibacterial, antifungal, and antitumor properties. Squalene derived from shark liver boosted the immune system in experimental mice with sarcoma. Shark cartilage (which despite marketing claims does not appear to have significant antineoplastic activity) is used in creation of artificial skin for humans and is an ingredient in cosmetics, and shark blood and liver oil are being investigated for hematologic and immunologic properties.[86] The latter is used in preparations to shrink hemorrhoids. Angiogenesis inhibitors (U-995 from the blue shark *Prionace glauca* and water-soluble neovastat [AE-941]) have been derived from shark cartilage.[54,65] Shark cartilage may also affect lymphocyte migration into murine tumors.[70]

SHARK FEEDING AND ATTACK

As previously noted, sharks are well equipped in the sensory aspects of feeding. They seem particularly able to avoid detection by potential prey, by virtue of coloration and a stealthy approach.[31] Sharks feed in two basic patterns: (1) normal or subdued, with

slow, purposeful group movements; and (2) frenzied or mob, as the result of an inciting event. The latter is precipitated by sudden presentation of commotion or food or blood in the water. Frenzied behavior is enhanced by the proximity of other sharks in large numbers. In a frenzy, sharks become fearless and savage, snapping at anything and everything, including each other. Shark feeding frenzies are rare in nature, but may occur in association with major aviation or maritime disasters. Most reports of feeding frenzies have been the result of humans providing food to induce the event.

Threat displays in sharks include pointing the pectoral fins downward (most common threat sign), a "hunch" posture (nose up, pectoral fins down, back hunched), body shiver (shark hesitates or stalls in the water and appears to shudder—seen in silvertip sharks), jaw gape, sideways turn (flank exposure with slow swimming), tail "popping" (noise from exaggerated tail beats), laterally exaggerated swimming, rapid approach and retreat, and gill cavity billowing. After a shark decides to attack, the "posture" may involve a combination of swimming erratically with elevated snout, hunched back, pectoral fin depression, stiff lateral bending of the body, and rapid tail motion, in contrast to its normal sinuous and graceful swimming style.[43,97] In bursts of speed, a shark can use its powerful caudal fin muscles, and some species may attain speeds in the water of 32 to 64 km/hr (20 to 40 mph). Lamnid sharks, which include mako and white sharks, swim with a lunate tail sweep similar to that employed by tuna, facilitated by interposition of aerobic, fatigue-resistant red muscle and anaerobic, rapidly fatiguing white muscle.[180] Because sharks do not possess a swim bladder, they can ascend rapidly in pursuit of prey without incurring barotrauma.[177] As the carchariniform shark prepares to strike, it rapidly opens and closes its jaws up to three times each second, depresses the pectoral fins (from horizontal to as much as 60 degrees downward) in a braking action, and elevates the head. During a bite of a large prey item, the shark shakes its head and forebody in an effort to tear flesh from its prey. If the object of such a bite is human, there may be gouge marks left on bones or teeth shattered and embedded in the human skeleton. The white shark often bites and releases its prey prior to returning to consume the weakened creature. Large sharks swallow food whole without chewing it. Some smaller sharks or juveniles, perhaps because they are gape-limited with respect to feeding, may repeatedly suck and spit prey into and out of the mouth in order to reduce food in size.[125,139] Analysis of human remains recovered from a tiger shark indicate that the human victim was dismembered and then swallowed and digested.[95]

It is difficult to postulate hunger as the sole attack motive, because more than 70% of victims are bitten only once or twice. "Hit-and-run" attacks, in which the shark bites, then releases and leaves the area, are most common. Usually the spike-like lower teeth are used first in feeding; these anchor the victim to allow the serrated upper teeth to carve through the flesh. Solitary upper tooth slashes might indicate attacks unrelated to feeding. Up to 60% of wounds involve only the upper teeth. To aid in cutting, the shark shakes or rolls its head. At the moment of the strike, the shark rolls its eyes back in the sockets and uses the ampullae of Lorenzini to home in on the victim.

Like all predators, sharks commonly attack young, old, injured, or sick prey.[34] They are selective feeders with clear dietary preferences. Sea turtles, penguins, seals, and stingrays are consumed by certain large shark species; fish, squid, and shrimp are commonly taken by moderate- and small-sized species. Humans are not preferred food items because they are not normal inhabitants of the sea. Sharks often eat other sharks. The great white shark cruises along the bottom of the ocean preparing to launch an attack on an unsuspecting surface animal. It can strike with enough force to lift the animal out of the water and breach itself (Figure 73-47), tearing a 50-lb chunk of flesh from its victim or even decapitating the animal (Figure 73-48). The cookie-cutter (or cigar) sharks *Isistius brasiliensis* (Figure 73-49A) and *Isistius plutodus* (see Figure 73-49B) create circular crater-like wounds approximately 5 to 6 cm (2 to 2.4 inches) in diameter when they attack pinnipeds, tunas, billfishes, and other large fish. Postmortem bites on humans have been documented; recently a Hawaiian swimmer suffered a nonlethal bite.[89] Sharks have short intestines, seem to be able to selectively digest ingested foodstuffs, and may be able to keep portions of what they ingest intact for prolonged periods of time, perhaps as a method to regulate nourishment. Relatively intact human limb segments have been removed from shark stomachs many days after ingestion.[39]

FIGURE 73-47 Great white shark begins to breech. *(Copyright Stephen Frink.)*

FIGURE 73-48 Great white shark *(Carcharodon carcharias)* with open jaws on surface, Neptune Islands, South Australia. Protected species. *(Copyright Gary Bell:* oceanwideimages.com.*)*

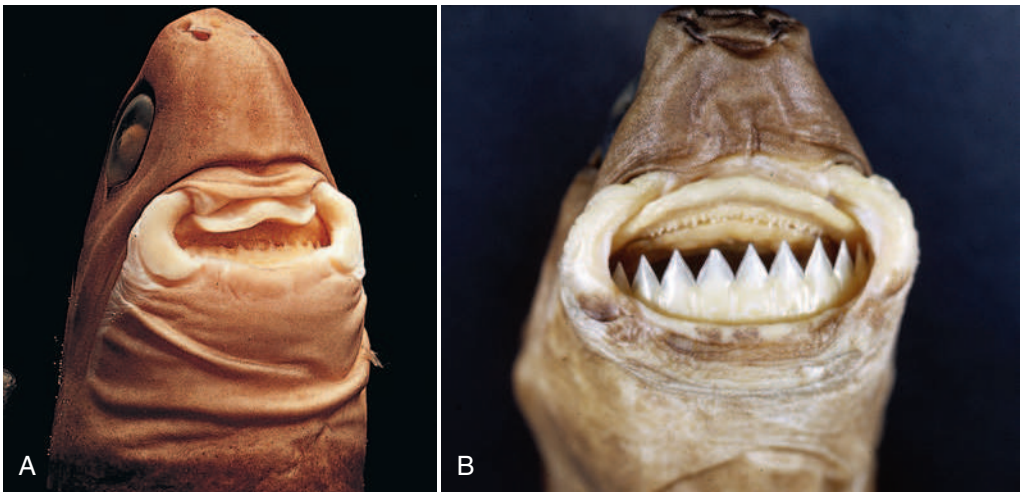

FIGURE 73-49 The cookie-cutter sharks *Isistius brasiliensis* **(A)** and *Isistius plutodus* **(B)** cut pieces of flesh off their prey with their unusual tooth configuration. *(**A** courtesy George H. Burgess; **B** copyright 2000 Norbert Wu:* norbertwu.com.*)*

It is difficult to generalize about shark attacks on humans. Most attacks likely occur as cases of mistaken identity in which the shark misinterprets the splashing of humans at or near the water surface as the activity of normal prey items. Less commonly, attacks may be direct feeding events in which large sharks simply perceive the human as appropriate sized and demonstrating appropriate behavior patterns. Finally, overt agonist behavior related to territoriality or personal space may contribute to some attacks, especially in the reef environment. Current explanations suggest that frightened persons engaged in erratic escape activity are more likely to incite attack. This has been demonstrated in the case of the grey reef shark, *Carcharhinus amblyrhynchos*. Aggression may be aggravated by purely anomalous behavior, violation of courtship patterns, or territorial invasion. More docile behavior tends to be the rule with other reef sharks, such as the silvertip *(Carcharhinus albimarginatus)* (Figure 73-50), blackfin *(C. melanopterus),* or whitetip *(Triaenodon obesus)* (Figures 73-51 and 73-52). A variety of environmental cues, including lunar periodicity, likely influence the presence of sharks and/or propensity to attack.[37]

The great white shark *(Carcharodon carcharias)* has been captured off Cuba at a length of 5.9 m (19.5 feet) and an estimated weight of 2045 kg (4500 lb); it is claimed, but not proved, that it can attain a length of 7.6 m (25 feet) and a weight of 2500 kg (5500 lb). It attains maturity at a length of approximately 4 m (13 feet). It is a man-attacker, but not always a man-eater. This statement reflects the observation that this highly feared animal usually releases its victim following a single "inquisitory" bite, a behavior it also employs on floating pieces of Styrofoam, surfboards, and marine mammals it does not consume, such as sea otters. Humans may survive and avoid consumption by having the ability to retreat to boats or surfboards prior to return of the shark, a luxury unavailable to the white shark's normal prey. This is small consolation to the unfortunate victim, who

FIGURE 73-50 Silvertip reef shark. *(Copyright Stephen Frink.)*

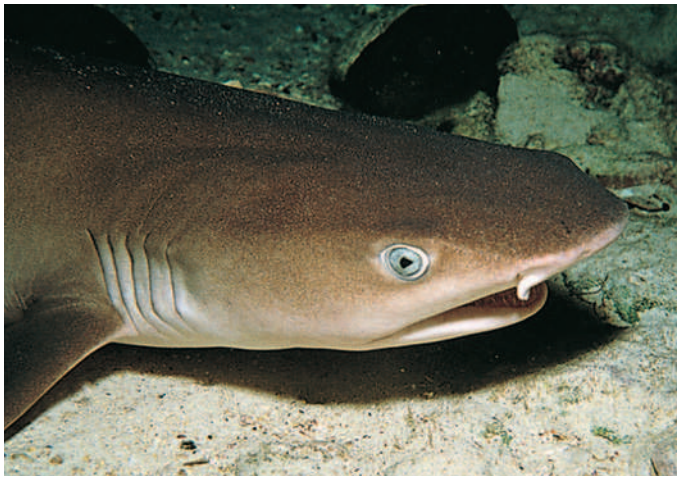

FIGURE 73-51 Whitetip reef shark. This species tends to be fairly docile. *(Courtesy Paul S. Auerbach, MD.)*

FIGURE 73-52 Whitetip reef shark with remora. *(Copyright Carl Roessler.)*

FIGURE 73-53 Victim of great white shark attack. **A,** Damaged surfboard ridden by the victim. **B,** Massive thoracic injury after a single bite. It was estimated that the shark was 6 m (20 feet) long. *(Courtesy P. Crossman, Coroner's Division, Salinas, California.)*

may have an entire hemithorax or limb removed (Figure 73-53). The great white shark has only recently been closely observed in the wild and is thus the subject of much speculation about predation strategies.[131] The feared trait of great white sharks is that they initiate contact with humans.[67] Their unpredictable nature ranges from a seemingly docile approach to a research boat to a powerful attack on a surface sea lion. Because adults feed largely on pinnipeds, the "bite-and-spit" behavior is considered a means of avoiding injury from struggling prey (Figure 73-54) while allowing the prey to weaken from exsanguination. However, it has also been observed that white sharks will sometimes chase and seize wounded prey, holding onto them during exsanguination, so there is no absolute feeding pattern.[103] It has been offered that a shark may release the victim to avoid self-drowning, because of the need to take water into its mouth in order to oxygenate gill tissues.[18] Another possibility is that it might be necessary for a shark to initiate the bite from a wide-opened jaw position.

One theory is that a shark that largely consumes a human victim does so because the victim was solitary in the water.[68] Breath-hold diver behavior and the similarity of the silhouette of a contemporary surfboard to that of a surface seal may be responsible for white shark attacks on humans. Most attacks on humans occur at or near the water's surface because this is where humans most often are found. One fatal attack in 1989 with two victims was on sea kayakers off the coast of southern California. From 1900 to 2014, there were 2777 unprovoked white shark attacks on humans, resulting in 497 fatalities worldwide (unpublished ISAF data). In 2014, there were 72 unprovoked attacks and 3 fatalities. The most recent attack statistics can be obtained from the ISAF at flmnh.ufl.edu/fish/sharks/isaf/isaf.htm.

FIGURE 73-54 Giant sunfish *(Mola mola)*. Note the shark bites on the posterior end of the fish. *(Courtesy Paul S. Auerbach, MD.)*

Shark attacks have occurred from the Atlantic waters of Canada to southern New Zealand, with most between latitudes 46 degrees N and 47 degrees S, because of the increased presence of humans and sharks in warmer waters. The odds of being attacked by a shark along the North American coastline are approximately 1 in 11.5 million (ISAF data). From a worldwide perspective, danger is greater during summer months (more people in the water, expanded shark ranges), in recreational areas (splashing, noise), from dusk to dawn (preferred feeding time for many sharks), and in murky, warm (> 20°C [68°F]) water. By contrast, white sharks prefer cooler water, and attacks have occurred in waters as cold as 10°C [50°F]).[194] Attacks in northern California occur more frequently in clearer water at temperatures of less than 16°C [60°F]).[133] Shark attacks in Hawaiian waters are infrequent but, as in most areas of the world, on the rise.[208] Tiger shark attack increases in Hawaii may be due in part to attraction to seagoing green turtles that come close to land, but most likely are simply reflective of rising human recreational utilization (especially surfing) of coastal waters.

Although most attacks occur within 30.5 m (100 feet) of shore, this is an artifact of the high human density in this region rather than a reflection on shark distribution. Other areas, such as mouths of rivers and inlets, edges of channels and drop-offs, and reefs and other hard-bottom relief features, are areas of shark abundance. Because of their ability to detect contrasts, sharks have a predilection to attack bright, contrasting, or reflective objects. Movement is an added attraction to sharks, which have been known to bite surfboards, boats, propellers, float bags, fishing bags, crab traps, and buoys. Because there does not appear to be any pattern of shapes, colors, or sizes to the biting behavior of white sharks on inanimate objects, it is possible that these sharks strike unfamiliar objects to determine potential food value or to protect territory.[60] Some shark attacks in northern California coastal waters have involved swimmers on surfboards (black on white), who entered migratory elephant seal (white

FIGURE 73-55 **A,** Silhouette of diver on surfboard to demonstrate similarity in shadow and contour to a sea lion at the surface. **B,** A great white shark passes underneath a dummy on a surfboard before making an attack. **C,** The shark begins to elevate its head from the water. **D,** With jaws wide, the shark attacks the dummy. **E,** With great commotion, the dummy and surfboard are dragged beneath the surface. *(Courtesy Images Unlimited, Inc. **A** courtesy Al Giddings; **B** courtesy Rosemary Chastney; **C** to **E** courtesy Walt Clayton.)*

shark food) habitats (Figure 73-55). The color black may encourage great white shark interest, perhaps resembling the dark coloration of certain marine mammals.[37] Great white sharks congregate in autumn in the waters around the rocky Farallon Islands, 43.5 km (27 miles) off the Golden Gate Bridge near San Francisco, where they actively pursue seals. They also congregate, seasonally and predictably, in other locations, such as around Guadalupe Island off the coast of Mexico. Great white sharks have been observed to use hunting strategies involving anchor points or lairs, rather than random attacks. The attack locations possibly are selected by a balance between prey detection, competition with other sharks, and water conditions that allow a swift vertical attack.[126] In a study of the behavior of great white sharks and their pinniped prey during predatory attacks, it was noted that white sharks feed on phocids (elephant seals) more frequently than on otariids (sea lions). It was also noted that certain prey might be deemed less palatable after initial sampling on the basis of perceived low energy value.[103]

Most shark attack victims are bitten by a single shark, violently and without warning. In the majority of attacks, the victim does not see the shark before the attack. Attacks occur predominately from below or from the side. The most common (80%) attack type is hit and run, in which the victim is seized and released, or slashed on an extremity.[32,90] This frequently occurs in shallow water and has been attributed to (1) mistaken identity of potential prey, (2) juvenile sharks with poor predatory ability, or (3) situational provocation.[19,36] Another type is a "sneak" attack on a diver or swimmer in deeper water, whereby a shark attacks without being seen. Finally, "bump-and-bite" attacks have the shark make contact with the victim prior to actually biting. This may be an attempt by the shark to determine the defensive abilities of its prey or to wound the victim before the definitive strike. A shark may circle a victim prior to bumping. Severe skin abrasions (Figures 73-56 and 73-57) and bruising from the shark skin can be produced in this manner. The sneak and bump-and-bite attacks generally are perpetrated by larger animals and are thought to be part of intentional feeding behavior.

CLINICAL ASPECTS

The jaws of the major carnivorous sharks are crescent-shaped and contain up to five or six rows or series of sharp ripsaw triangular teeth, which are replaced every few weeks by advancing inner rows (Figure 73-58). The teeth of sharks are not attached directly to the jaw cartilage, but are held in place by a collagenous membrane in a shallow depression known as the tooth bed.[138] Each species has distinctively shaped teeth.[186] However, teeth of the great white shark reveal no consistent pattern of size or arrangement of the marginal serrations that are sufficiently characteristic within an individual shark to serve as a

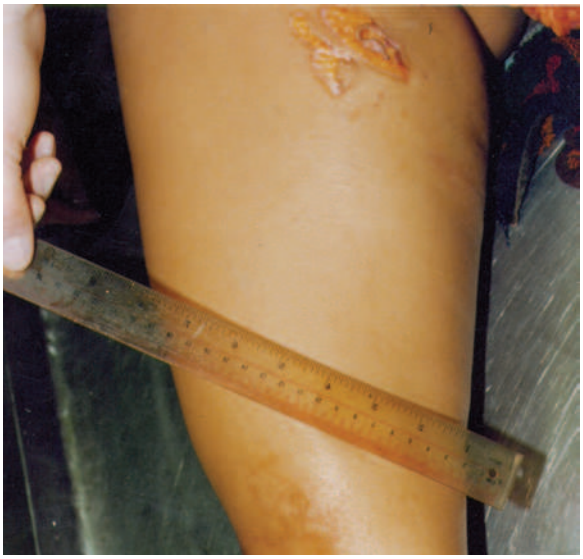

FIGURE 73-56 Skin abrasion and bruise from shark denticles. *(Courtesy George H. Burgess.)*

reliable index of identification of a tooth as originating from that particular shark. Tooth shape and size are useful in determining its place in the jaw, and serrations are sufficiently distinctive to enable the potential identification of an individual tooth as having been the cause of a particular bite mark.[143] The size of a shark can be determined from forensic analysis of bite damage and the two evolutionary lineages of sharks most often involved in attacks, the carcharhinoids and lamnoids, are discernable by dental characteristics.[121] A nomogram has been developed that can be used to estimate the body length of a great white shark from measurements of the tooth or bite mark morphology.[142] Although normal tooth replacement takes 7 to 14 days, in some species a lost tooth can be replaced within 24 hours. Sharks are born with a full set of teeth (covered by a protective ectodermal sheath), and in some cases have up to 15 rows of replacement teeth available. Amazingly, some sharks produce up to 25,000 teeth in a lifetime, as teeth are shed frequently (Figure 73-59);

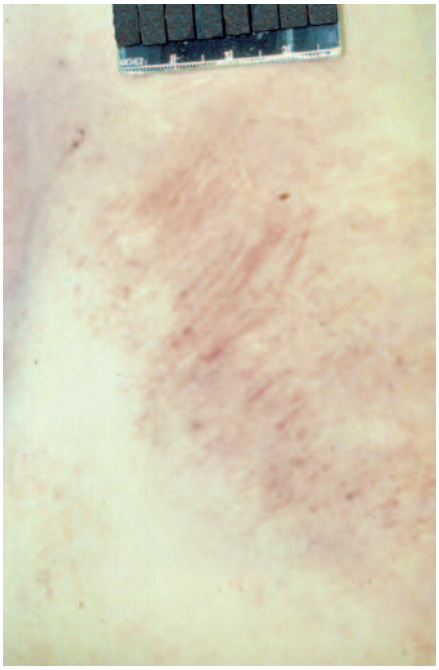

FIGURE 73-57 Skin abrasions from shark denticles. *(Courtesy George H. Burgess.)*

FIGURE 73-58 Teeth of bull shark *(Carcharhinus leucas). (Copyright 2000 Norbert Wu:* norbertwu.com.*)*

this explains the abundance of fossil shark teeth in paleontologic excavations. The upper jaws generally have larger cutting teeth, whereas the sharp lower teeth are designed to fasten onto and hold prey during capture.[77] The bamboo shark *(Chiloscyllium plagiosum),* which dines largely on crustaceans, is able to fold its teeth inward on a flexible broad ligament in order to crush and consume its normal prey.[167]

Shark teeth are cartilaginous, strengthened by the deposition of calcium phosphate crystals (apatite) in a protein matrix, all covered by an enameloid substance. They are considered to be as hard as granite and as strong as steel. In a large great white shark, the largest serrated triangular teeth can grow to 6.4 cm (2.5 inches), with 26 upper and 24 lower teeth exposed in the front row. The height of the enamel of the largest tooth in the upper jaw is proportional to the animal's length, so a body length of up to 7.6 m (25 feet) may be possible based on recovered teeth. Teeth and fragments embedded in clothing or the victim may assist in identification of the biting species. The upper jaw is advanced forward and protruded to allow its participation in the biting action (Figure 73-60). The biting force of tiger (Figure 73-61) and dusky sharks is estimated at as high as 21 tons per square inch across the tooth tips. By comparison, humans have been estimated to bite with a force of 15 tons per square inch. Severe shark bites result acutely in massive tissue loss, hemorrhage, shock, and death. Even a smaller animal, such as a young lemon shark, can bite with bone-crushing force.[79] The potential for destruction is enormous, in parallel with other predators, such as tigers, lions, and bears.

Lentz and colleagues reviewed the mortality rates and management of 96 shark attacks and developed the Shark-Induced Trauma (SIT) Scale.[118] Arms and especially lower legs are the most common trauma regions in shark attacks on humans (Figure 73-62). Sharks most often attack from below and behind. Therefore, the legs and buttocks are most frequently bitten (Figures 73-63 and 73-64), perhaps an indication of leg movement and the available body mass of buttocks and thighs. This is followed in frequency by the hand(s) and arm(s), as the victim attempts to fend off the shark (Figures 73-65 to 73-67).[39] Because sharks

FIGURE 73-59 Shark shedding tooth. *(Copyright Lynn Funkhouser.)*

FIGURE 73-60 Jaw of a great white shark. *(Copyright 2000 Norbert Wu:* norbertwu.com*.)*

do not chew their food, their method of biting and rolling or thrashing allows flesh to be stripped from the victim. Teeth may be embedded in prey (Figure 73-68), but fractured bones are surprisingly rare.[95] Degloving injuries occur when a victim attempts to pull an extremity from the mouth of a biting shark, raking soft tissue across sharp teeth (Figure 73-69). Skin lacerations may display tooth patterns (Figure 73-70). Proximal femoral artery disruption carries a poor prognosis because of torrential hemorrhage (Figure 73-71). Although fractures are not common, broken ribs often accompany intrathoracic, intraperitoneal, and retroperitoneal injuries. Spiral gouges or crescent-shaped grooves horizontal to the shaft on bones indicate the bite and roll activity of an attacking shark.[95] In 2007, an Australian abalone diver claimed to have been partly swallowed head first by a great white shark, which released him when the man fought his way free.

Because the victim is generally far from medical assistance, blood loss may be profound. The wounds have historically been fatal in 15% to 25% of attacks, but have averaged under 10% over the past decade, with major causes of death listed as hemorrhage and drowning. Rapid response and prehospital care are undoubtedly improving this statistic, as have advances in major trauma treatment gained in part from lessons learned in 20th-century wars.

FIGURE 73-61 Tiger shark on a reef. *(Copyright Stephen Frink.)*

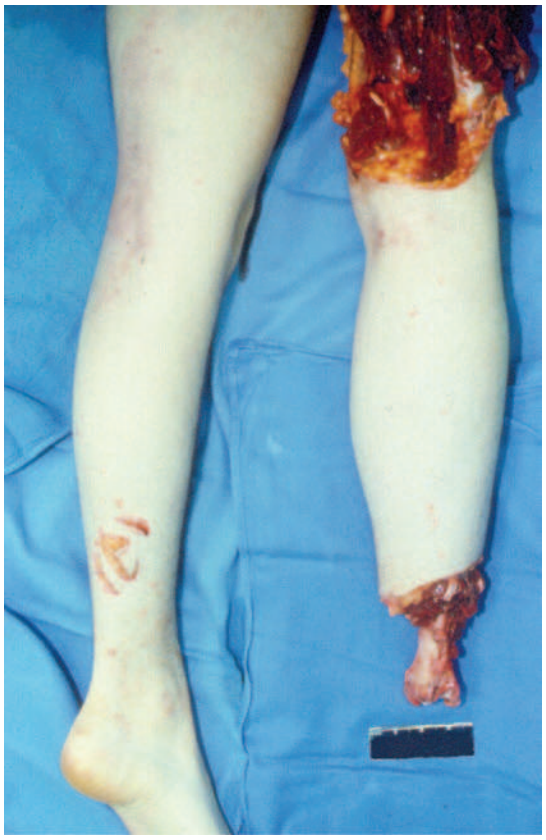

FIGURE 73-62 Tiger shark spin and pull. *(Courtesy George H. Burgess.)*

In a retrospective review of 12 corpses recovered from Okinawa, Japan, with shark-induced, mostly postmortem injuries, the characteristic injury features were felt to be sharp incision without abrasion, wound with a serrated edge, triangular or rectangular flap of skin, regular arrangement of marks that correspond to shark teeth, gouge marks on bone, and severance of the body part at the joints without a fracture.[93] Postmortem bites from sharks and other marine creatures, such as sea lice, can

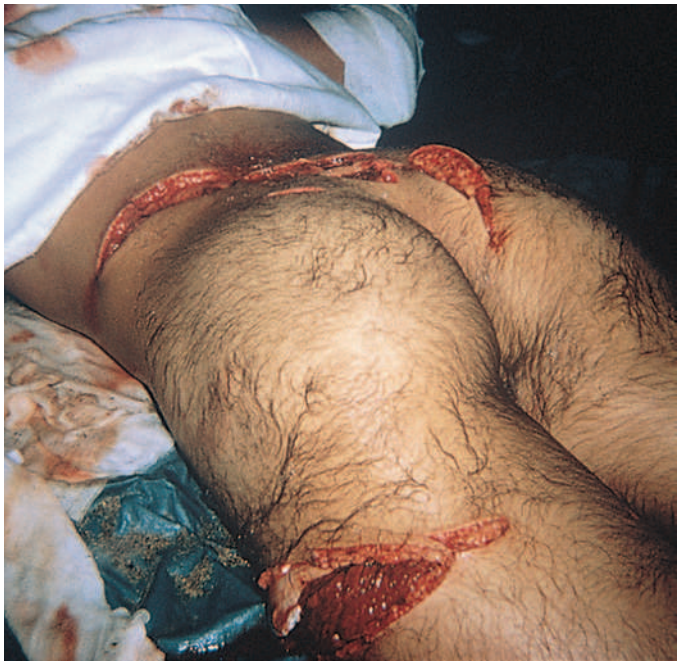

FIGURE 73-63 Shark bite of the buttocks and thigh. *(Courtesy T. Hattori, MD.)*

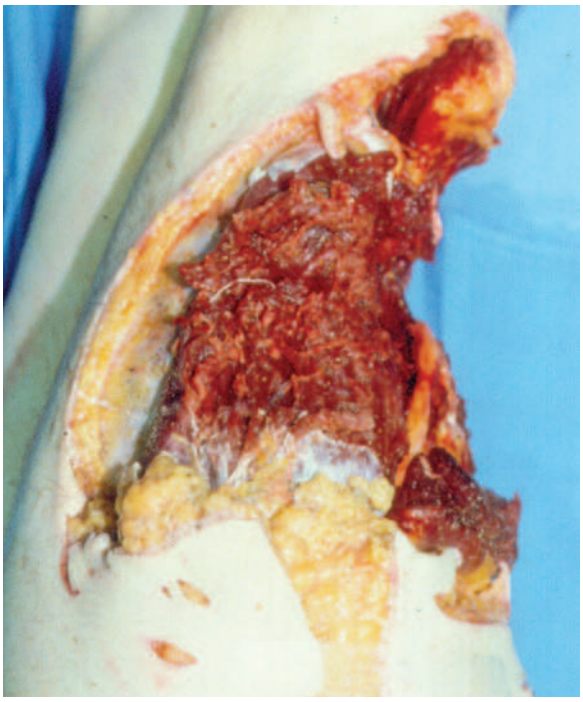

FIGURE 73-64 Typical thigh wound from shark bite. *(Courtesy George H. Burgess.)*

create the appearance of inflicted injury and obscure the true cause of death.[40] It therefore becomes important to differentiate postmortem artifacts from injuries that may have been inflicted while the victim was alive. For instance, the drowned victim may be bitten by sharks. If the wounds do not penetrate blood vessels or vital structures, they should be suspected of being noncontributory to the fatal event. However, when only part of a body is recovered, it is difficult to determine if a shark bite was the

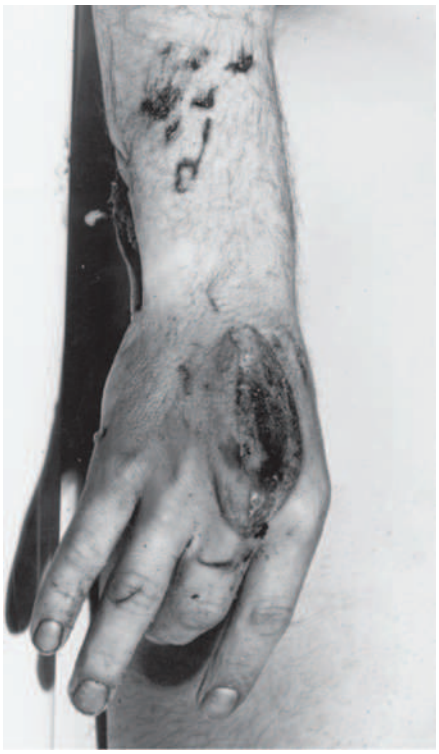

FIGURE 73-65 Defensive wound of the hand from shark bite. *(Courtesy George H. Burgess.)*

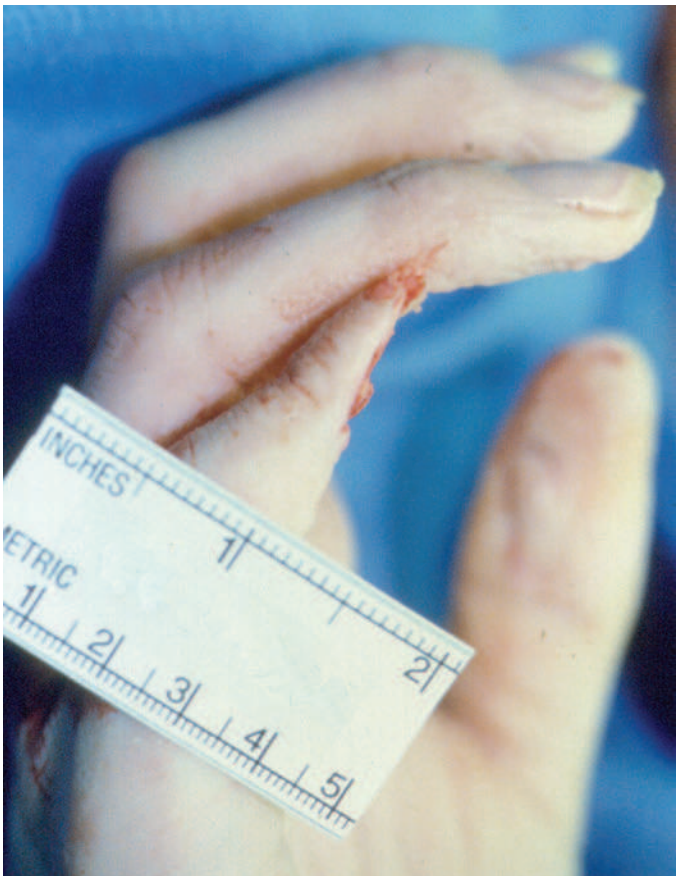

FIGURE 73-66 Defensive wound of the finger from shark bite. *(Courtesy George H. Burgess.)*

inciting event. Furthermore, sharks are capable of maintaining tissues within their stomachs for weeks at a time without digestion occurring, which may further obscure determination of time of death.[123] Bite configurations may be pathognomonic for some species, but species identification is difficult in most cases. The circular and C-shaped injuries inflicted by the cookie-cutter shark *(I. brasiliensis, I. plutodus)* are made by small erect teeth in the upper jaw and triangular teeth in the lower jaw, resulting in the distinctive carving of a plug of flesh (Figure 73-72).[212] Cookie-cutter sharks have been implicated in bites on live and dead humans[89,93,123] (Figure 73-73). Two cases of fatal great white shark attack were reported where the only tissues recovered were lung fragments.[41] The authors concluded that the only tissue to escape being consumed or lost in fatal shark attacks associated with dismemberment may be lung. Aerated lung tissue from a recently killed victim may rise to the surface, as opposed to fluid-filled lung tissue that would be characteristic of a drowning.

Sharks can grow to be quite large and are strong creatures. Fishermen are injured by thrashing animals on boat decks and in fishing nets. In one unusual incident reported to the author (PSA) by Dr. Edward Paget, the captain of a fishing ship jumped into the ocean in order to free a whale shark caught in a net. The enormous animal bumped him against the ship. The captain suffered mild abdominal pain, which worsened to the extent that he sought care in a hospital at Majuro in the Marshall Islands. He was transferred 300 miles by airplane to Dr. Paget, who observed signs of blood loss and localized peritonitis. At surgery, the victim was found to have 3400 mL of intraperitoneal blood with clots and a gangrenous 2-inch segment of sigmoid colon that necessitated a diverting colostomy.

TREATMENT

In most cases, the immediate threat to life is hypovolemic shock. In one series compiled by the South African Natal Sharks Board,

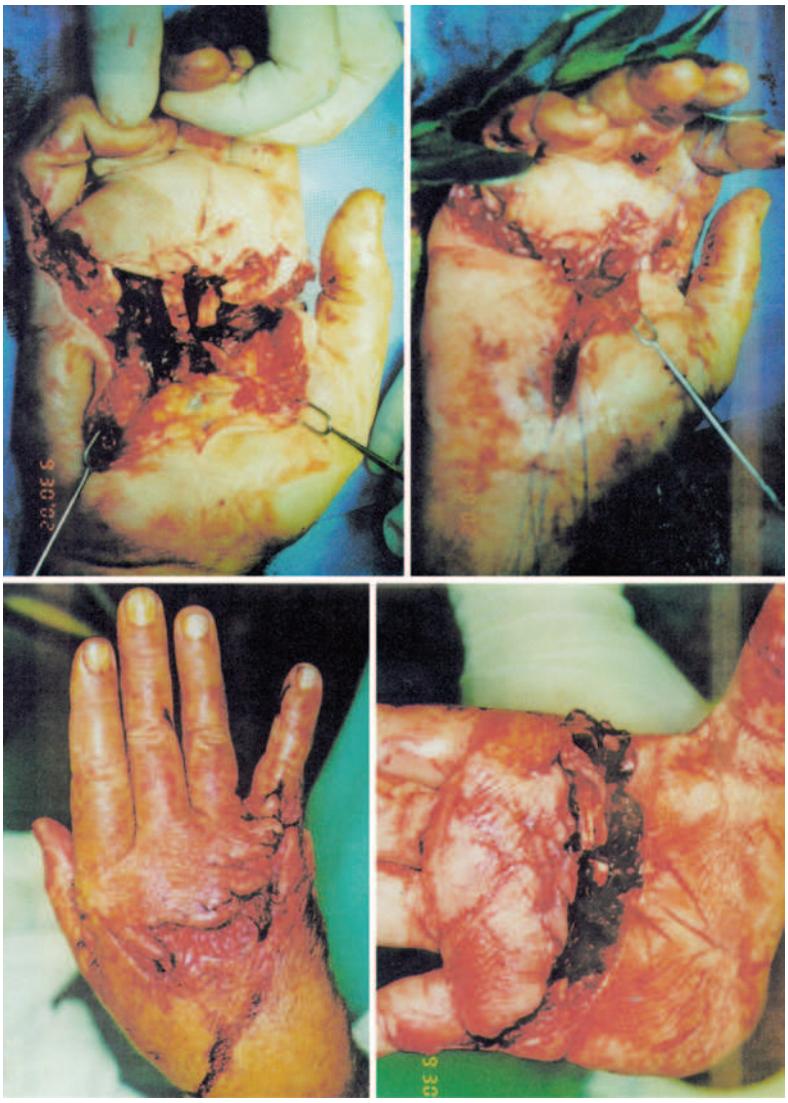

FIGURE 73-67 Nondefensive shark bite of the hand. *(Courtesy George H. Burgess.)*

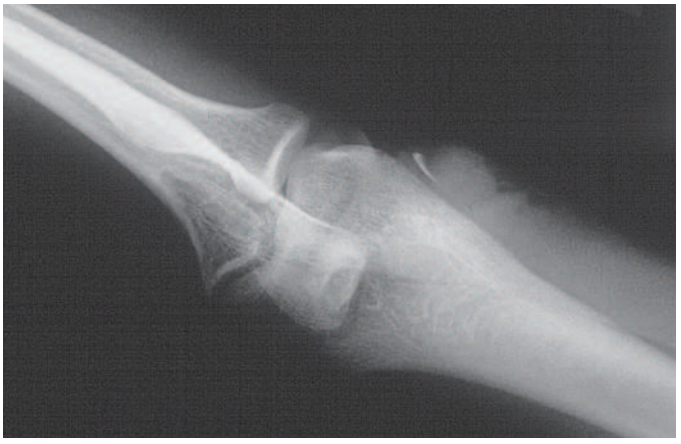

FIGURE 73-68 The presence of a shark tooth in the arm is revealed by x-ray examination.

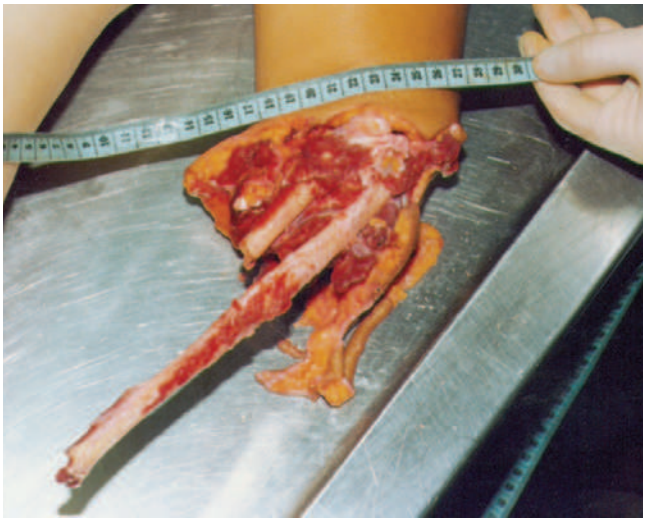

FIGURE 73-69 Tiger shark "bite and spin" behavior creates a degloving injury. *(Courtesy George H. Burgess.)*

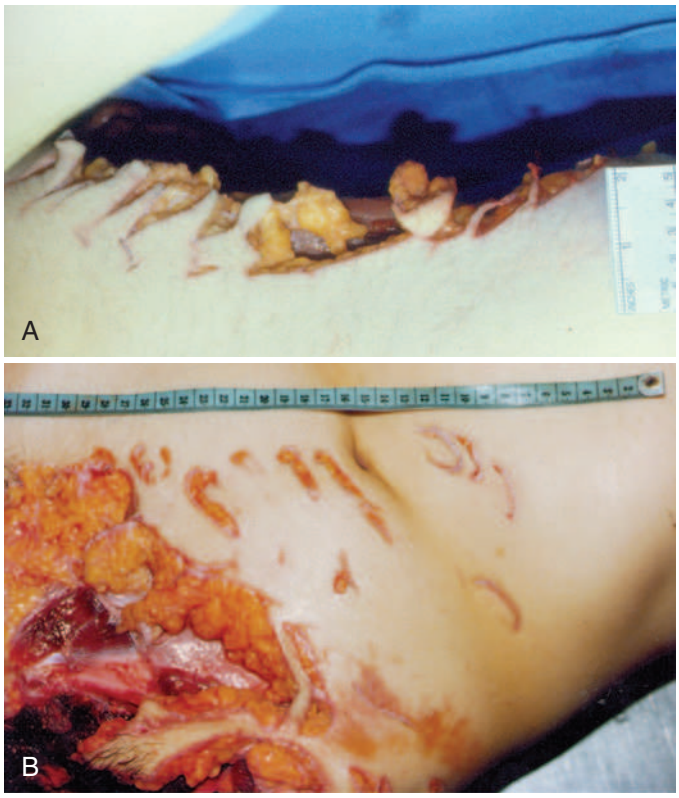

FIGURE 73-70 **A,** Shark bite creates wound edge that displays tooth pattern. **B,** Tooth pattern and wounds created by bite of a tiger shark. *(Courtesy George H. Burgess.)*

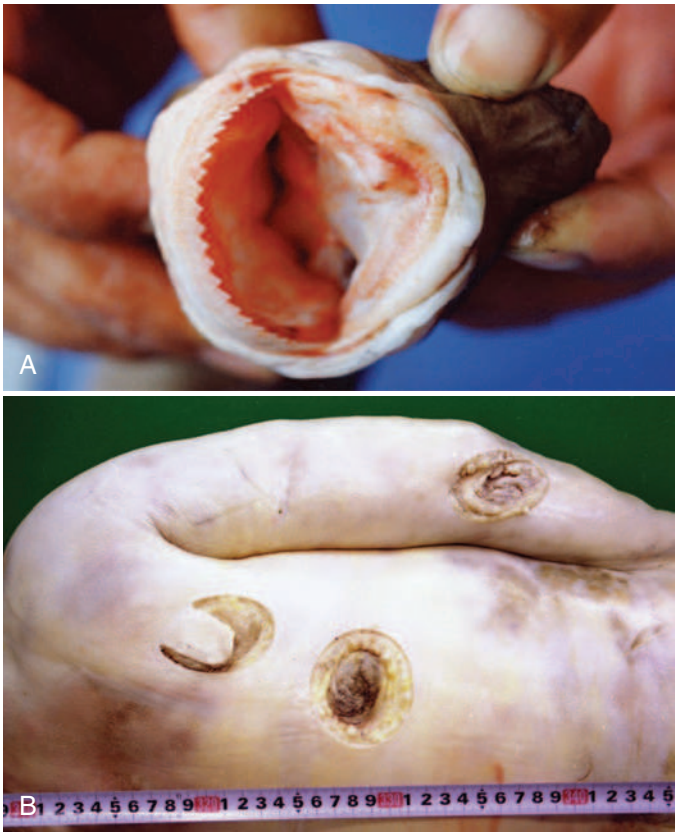

FIGURE 73-72 **A,** Jaws of the cookie-cutter shark. **B,** Postmortem wounds created by cookie-cutter shark bites. *(Photos courtesy Y. Makino.)*

death occurred most frequently as a result of exsanguinating hemorrhage from a limb vascular injury. Thus, it is occasionally necessary to compress wounds or manually constrict arterial bleeding while the victim is in the water. As soon as the victim is out of the water, all means available must be used to ligate large, disrupted arteries or to apply compression dressings. In one incident, dental floss was used by a fast-thinking companion of the victim to ligate a severed vessel while in the field, likely saving the victim's life. If necessary, judicious use of pressure points or tourniquets should be entertained, taking care to avoid excessive ischemia time in treated tissue. If intravascular volume must be replaced in large quantities, at least two large-bore IV lines should be inserted into the uninvolved extremities to deliver crystalloid (lactated Ringer's solution, normal saline, or hypertonic saline), colloid, or blood products.[141,191] If prolonged transport is necessary, central IV access may be helpful.[154]

The victim should be kept well oxygenated and warm while being transported to a facility equipped to handle major trauma. Blood losses should be replaced with whole blood or packed red blood cells and fresh-frozen plasma.[190] The precise ratio of crystalloid to blood products and proper mean arterial blood pressure end point of primary resuscitation in the presence of a major vascular injury are the subjects of ongoing investigations.[179] The victim should be thoroughly examined for evidence of cervical, intrathoracic, and intraabdominal injuries. Because *Clostridium* can be cultured from ocean water, tetanus toxoid 0.5 mL IM

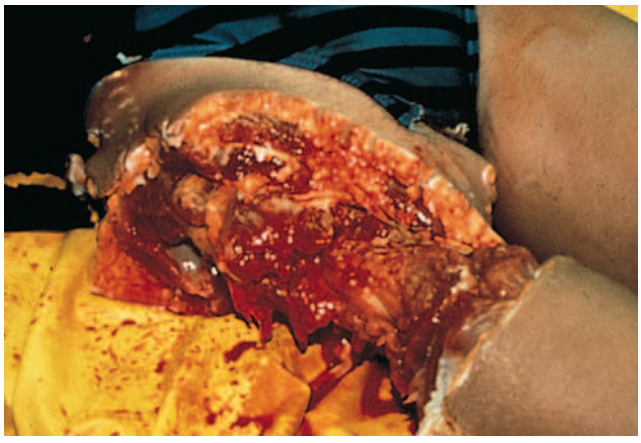

FIGURE 73-71 Shark bite of the proximal thigh. This unfortunate victim exsanguinated from a disrupted femoral artery. *(Courtesy Kenneth W. Kizer, MD.)*

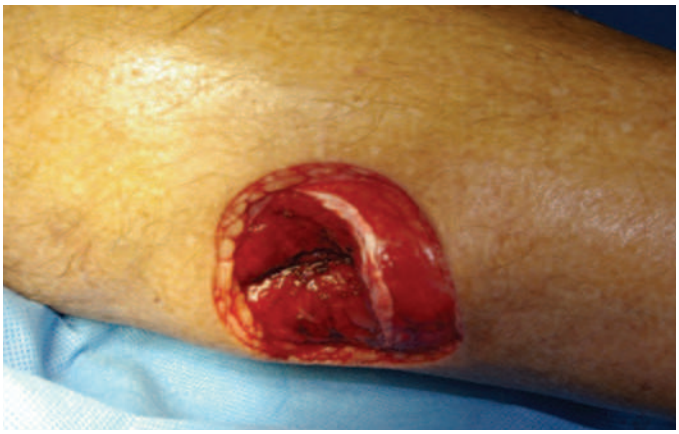

FIGURE 73-73 Cookie-cutter shark bite on a live human. *(Photo courtesy Peter Galpin.)*

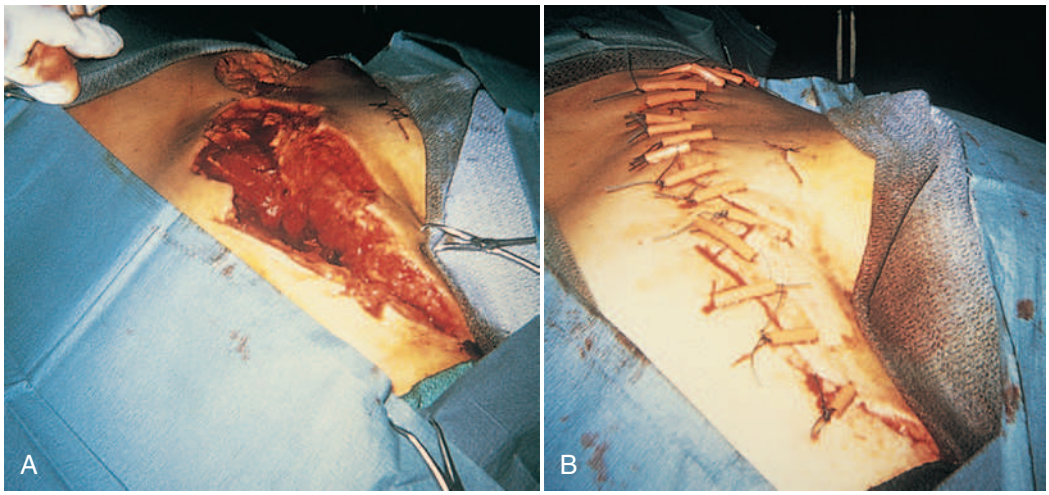

FIGURE 73-74 Operative repair of shark bite wound shown in Figure 73-53. **A,** Debridement and exploration of the wound in the operating room. **B,** Proper closure technique with tension-releasing sutures. *(Courtesy T. Hattori, MD.)*

and tetanus immune globulin (HyperTET, Grifiols, Barcelona) 250 to 500 units IM must be given. The administration of prophylactic antibiotics is more controversial. The victim of a shark bite should be treated with an IV third-generation cephalosporin, trimethoprim-sulfamethoxazole, an aminoglycoside, ciprofloxacin, or some reasonable combination of these agents. Imipenem-cilastatin or meropenem should be reserved for established wound infections or early indications of septicemia, particularly in the setting of immunosuppression. The rationale for prophylactic antibiotics is that shark wounds are prone to heavy contamination with seawater, sand, plant debris, shark teeth, and shark mouth flora. After a clinical infection is recognized, wounds should be cultured for aerobes and anaerobes by insertion of sterile swabs deeply into available lesions.

Proper operative intervention is mandatory.[35,52] It is inappropriate to attempt emergency department exploration of what often prove to be extensive and complicated wounds. In the operating room, devitalized tissue should be widely debrided and the wound irrigated copiously to remove all foreign material (Figure 73-74). An x-ray may reveal one or more shark teeth in the wound (see Figure 73-68). Shark teeth and fragments should be identified and removed to avoid infection initiated by a contaminated foreign body. Vascular repair should be completed when necessary.[212] Unless it is absolutely necessary to achieve tight closure, the wound should be carefully undermined and closed loosely around multiple drains (preferably closed systems) or packed open to await delayed primary closure. Although there is debate about whether to use internal or external fixation of grossly open and contaminated fractures, it seems logical to recommend surgical stabilization to facilitate vascular and soft tissue repair. In the pediatric population, damage to the physis and future limb length discrepancy should be anticipated.[79]

The abrasion associated with a shark bumping should be managed like a second-degree burn, with daily debridement and application of antiseptic ointment.

Postoperative management may be prolonged and complicated by acute renal failure attributed to hypovolemia and shock, massive blood transfusion, myoglobinuria, and administration of nephrotoxic antibiotics. Rehabilitation may include creation of prosthetic devices.

A reasonable "shark pack" should be available in emergency facilities and rescue vehicles near shark-inhabited waters. This must be portable and should include items necessary to control hemorrhage and initiate IV therapy.

Shark phobia is a real entity that causes the victim to be unwilling to enter the ocean. In one case, it has been treated successfully by a self-administered systematic desensitization program that followed a hierarchical progression of entry into the ocean combined with deep muscle relaxation technique.[106]

PREVENTION

Every precaution should be taken to avoid shark attack, beginning with an intimate knowledge of the local waters. The following is precautionary advice and a list of alternatives for action in the event of a confrontation:

Avoid shark-inhabited water, particularly at dawn, dusk, and at night. Do not swim through schools of bait fish in the presence of sharks. Do not enter waters posted with shark warnings. Surfers are generally at greater risk than divers. Do not disguise yourself as a pinniped (seal). Do not swim with animals (such as dogs or horses) in shark waters. Shark behavior can be unpredictable, so it is best not to remain in the water with sharks, particularly if you are fearful. Although some persons believe that sharks can be domesticated, there is no such thing as a friendly shark. Photograph hazardous sharks from within the confines of a protective cage (Figures 73-75 and 73-76), rather than from an unprotected position.

Swimmers should remain in groups. Isolation creates a primary target, eliminates companion surveillance, and removes the opportunity for postattack assistance. When diving, maintain constant vigilance. Do not wander far from shore, particularly if you are a solitary swimmer.

Turbid water, drop-offs, deep channels, inlets, mouths of rivers, and sanitation waste outlets are areas frequented by sharks and should be avoided. Water clarity is cited as turbid in 64% of all attack incidents, so it appears that sharks attack at least one-third of the time in clear water. Humans are most often attacked

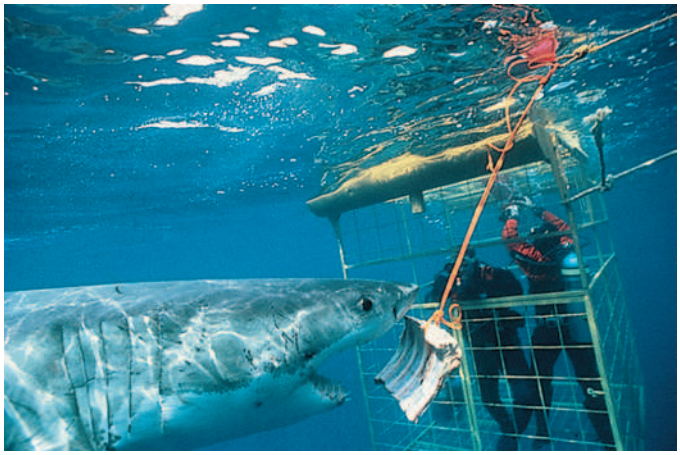

FIGURE 73-75 Caged divers view an inquisitive great white shark. *(Courtesy Carl Roessler.)*

FIGURE 73-76 The author descends in a cage to photograph great white sharks. *(Copyright Peter Riekstens.)*

in shallow water or beyond the breakers. Do not swim in waters frequented by recreational or commercial fishers. Do not swim in water that has been recently churned up by a storm. Be alert when crossing the troughs between sandbars.

Blood and other body fluids (including peritoneal fluid) attract sharks. No person should be in shark waters with an open wound. Women have historically been advised to avoid diving during menstruation, although there are no data to support attraction of sharks to the discharge of menstruation. Given the shark's well-documented acute sense of smell, it seems possible that menstrual blood is attractive.

Brightly reflective swimwear or diving equipment and shiny snorkeling gear attract certain sharks. Bright (international) orange and other contrasting colors appear to be particularly attractive to sharks. Flat black is probably the least attractive color, except in the instance of the great white shark, which may be preferentially attracted to this color. There is scant evidence that sharks are more attracted to light-skinned bathers, but differential tanning and base skin color producing lighter palms of the hands and soles of the feet may contribute to bites to these appendages, especially when movement is involved.

Captured fish must be tethered at a distance from any divers. There is no greater chemical attractant for a shark than fish blood, and the thrashing of a speared fish is greatly attractive. Do not dive or swim in the presence of spear fishermen (Figure 73-77). Divers who harvest abalone should be aware that the banging and prying noise of an "abalone iron" might attract sharks.

The presence of porpoises in the water does not preclude the presence of sharks (it is not uncommon for both to be pursuing the same prey items). Be alert for the presence of a shark whenever schools of fish behave in an erratic manner or when pods of porpoises cluster more tightly or head toward shore.

Do not tease or corner a shark. This is particularly true with captive animals. Do not pull on a shark's tail. Do not chase after a shark. If a shark begins to act in an erratic manner, do not photograph it at close range using a strobe flash apparatus. There is now evidence that shark feeding, as perpetrated by shark diving operators, may be followed by shark attack upon humans.

If a shark appears in shallow water, swimmers should leave the water with slow, purposeful movements, facing the shark if possible and avoiding erratic behavior that could be interpreted as distress. If a shark approaches in deep water, the diver should remain submerged, rather than wildly swim to the surface to

FIGURE 73-77 Blue water hunter with white sea bass off Catalina Island, California. *(Copyright 2000 Norbert Wu:* norbertwu.com.*)*

escape. The diver should move to defensive terrain with posterior protection to fend off, as best as possible, a frontal attack. It is inadvisable to trap a shark in a position from which it must attack to obtain freedom. Fighting sharks is difficult; they are best repulsed with blunt blows to the snout or probing of the eyes or gills. If possible, the bare hand should not be used, to avoid severe abrasions or lacerations. A stream of air bubbles from a scuba regulator directed into the face of a shark may serve as an initial deterrent, but this does not work most of the time. Although spears, knives, shotgun shell- or 30.06-loaded powerheads (bang sticks) (Figure 73-78), strychnine-filled spears, and carbon dioxide darts can kill small sharks, they can worsen the situation if they are misapplied or their application promotes frenzy in a school of sharks.

Do not splash on the surface or create a commotion in a manner that might cause a shark to interpret your behavior as that of a struggling fish. Surface activity, including the sound of marine engines and perhaps the sounds created by helicopter rotor wash, attracts sharks. During a helicopter rescue, exit the water as soon as possible.

Although there is no question that shark avoidance is the most reliable maneuver, shark defense techniques and repellents are constantly evolving. In response to shark attacks on downed airmen and sailors during World War II, copper acetate (20%) blended with a water-soluble wax and nigrosine-type black dye (80%) was packaged as a slowly dissolving (3 to 4 hours) 6-oz waxy cake for deployment as "shark chaser" by the Office of

FIGURE 73-78 Diver carrying a bang stick in the presence of a blue shark. *(Courtesy Howard Hall.)*

Naval Research of the U.S. Navy.[17] It was theorized that the dye released as a dark (often black or purple) cloud similar to defensive mollusk secretions, and that the copper acetate both inhibited feeding and resembled the decaying carcasses of sharks, so that sharks would thus be repelled. Unfortunately, although a morale booster, this was not a reliable deterrent. Its use was discontinued by the Navy in 1976. Limited progress has been made since that time. Recreational beaches in Australia and South Africa are protected with extensive gill net systems (meshing) to kill animals seeking to enter the surf zone. These work by reducing population sizes over time, thereby minimizing shark-human interactions. This type of prevention measure is increasingly under attack by biologists and conservationists because of its highly negative impact on populations of threatened sharks, sea turtles, sea birds, and sea mammals. Exclusion nets (large-mesh nets intended to prevent interaction between humans and sharks) have been employed in a limited number of locales. Logistic and economic considerations preclude widespread utility. Electric shark barriers (cable device) using 0.8-msec pulses 15 times per second to create a field of 4 volts per meter seem to generate a fright response in sharks longer than 1.2 m (4 feet) and are being investigated.[44,171] Their benefits include repulsion rather than shark capture or destruction. However, some sharks respond weakly to these stimuli. Abalone divers in South Australia work from one-person, self-propelled shark cages.

Experimental devices for individuals include chain-mesh diving suits (Figures 73-79 and 73-80), inflatable dull-colored plastic protective bags (yellow is easy for aircraft to spot, but most attractive to sharks), acoustic and handheld electrical field transmitters, surfactants and other chemical repellents (e.g., firefly and the Red Sea and western Indian Ocean Moses sole, *Pardachirus marmoratus* [Figure 73-81] glandular extract [pardaxin]).[58] Pardaxin is an excitatory polypeptide neurotoxin that forms voltage-gated pores and triggers neurotransmitter release.[113] The ichthyotoxic secretion from the fish, which appears as a milky substance from a series of glands located along the dorsal and anal fins, also contains shark-repellent lipophilic constituents that appear to be steroid monoglycosides.[152,183] Another sole, *Pardachirus pavoninus,* which lives in the tropical regions of the western Pacific and eastern Indian Oceans, secretes pavoninins 1 through 6. These shark-repelling substances are ichthyotoxic steroids, *N*-acetyl-glucosaminides.[209] It appears that in sharks, the gills or pharyngeal cavity is the target organ(s) for the repellent action. For the exudate from the Moses sole, concentrations of 10 to 25 g/m^3 are needed to elicit an immediate indication of repellency. However, it has been estimated that about 24 kg (53 lb) of any effective drug would have to be contained within an enveloping "drug cloud" in the volume of water through which a slowly approaching shark might swim in its final 10 seconds of approach as it attacked a human in the ocean.[17]

FIGURE 73-80 Shark feeding using chain mesh protective dive suit. *(Copyright Stephen Frink.)*

Studies have indicated that shark-repellent efficacy of alkyl sulfate surfactants is due to their hydrophobic nature.[169] In tests conducted on juvenile swell sharks *(Cephaloscyllium ventriosum),* the aversive response of sharks to these surfactants increased with carbon chain length from octyl (8) to dodecyl (12), decreased with the addition of ethylene oxide groups, and was not affected by counterions (e.g., magnesium for sodium). Still, for the most effective synthetic detergent repellent (e.g., sodium dodecyl sulfate [SDS]), the concentration needed in the water is 800 g/m^3. These findings show that a chemical carried in a life jacket cannot be reliably useful against sharks, because to meet the Navy's potency requirement for a nondirectional surrounding-cloud–type repellent, it would have to be instantaneously effective at a concentration of no less than 100 parts per

FIGURE 73-79 Diver wearing chain mesh metal suit for shark protection. *(Courtesy Howard Hall.)*

FIGURE 73-81 Moses sole, *Pardachirus marmoratus,* Thailand. *(Copyright 2000 Norbert Wu:* norbertwu.com.*)*

billion (0.1 μg/mL). Most tests have been performed on bait-attracted blue sharks *(Prionace glauca)*. In chemical repellent tests on white sharks, using an air-powered syringe gun to deliver 250 mL of a 10% seawater solution of SDS, it was demonstrated that large white sharks could be effectively repelled if a substantial dose reached the mouth cavity and remained there long enough to stimulate the relevant receptors. However, this has not proved practical in a field situation.[144] Future direction of chemical shark repellent research include delineation of the morphologic target sites of chemical and natural shark repellents, investigation of natural bioactive toxins as repellents, and identification and purification of semiochemicals (e.g., exudates or body secretions of predators that act as alarm pheromones) to be used as repellents.[169]

The Shark Shield series of shark-deterrent devices are distributed by Shark Shield Pty Ltd (sharkshield.com) in South Australia, based on the original technology that led to development of the SharkPOD (which is now discontinued). The Shark Shield produces an electrical field that is detected by sharks through the ampullae of Lorenzini and that is believed to cause discomfort and muscle spasms in the animals. The Shark Shield unit incorporates electrodes that project the field from the unit to create an invisible protective shield that surrounds the user. The electrodes must be immersed in the water in order to give protection to the wearer. One electrode is encased in a short antenna, which trails out from the ankle, secured with a neoprene cuff. The second electrode is a pad designed to be worn on the scuba cylinder. This pad electrode attaches to the cylinder by means of a special quick-release fastening device. Under no circumstances should the two electrodes be placed any closer to one another than 900 mm (35.43 inches), as this will reduce the deterrent effect. A full-length wetsuit or drysuit must be worn when using the Shark Shield Scuba unit to reduce the skin stimulation effect caused by the configuration of the two electrodes. The unit is strapped to the thigh or can be placed in the buoyancy compensator pocket. In tests, the unit has deterred sharks at distances of up to 8 m (26 feet), but with an average distance of 4 to 5 m (13 to 16 feet) from the unit. More recent studies on the Shark Shield as a deterrent to white sharks showed conflicting data. In a first trial of attaching the deterrent to static bait, the proportion of baits taken was not affected by the electrical field; however, it took an increased amount of time for the sharks to take the bait with the electrical field in place.[92] In a second experiment, using a towed seal decoy, the electrical field significantly decreased the number of shark strikes and interactions with the decoy.[92] These studies may indicate that shark response to the electrical field is contextually specific.

As for any shark-deterrent strategy, it cannot be assumed to provide perfect protection, because the animals are unpredictable, and it is impossible to state with assurance that they will always be deterred. A fatal attack by a great white shark upon a SharkPOD-equipped diver in 2002 has been attributed to the device not being used in accordance with the manufacturer's specifications, but the device produced sufficient current to deter human rescue attempts. Regardless of assignment of fault, it emphasizes that no device should be assumed 100% effective in all circumstances. The reader is advised to check on the availability and status of warranties and representations prior to committing to any particular purchase.

BARRACUDA

To many divers, the barracuda appears more sinister than the shark and is more highly feared. Barracuda are distributed from Brazil north to Florida, and in the Indo-Pacific from the Red Sea to the Hawaiian Islands. Of the 22 species of barracuda, only the great barracuda *(Sphyraena barracuda)* has been implicated in human attacks. Smaller species of barracuda may be found in large schools.

LIFE AND HABITS

The great barracuda is encountered in all tropical seas and is reputed to grow to 2.5 m (8.2 feet) and 50 kg (110 lb) but is rarely sighted at a length greater than 1.5 m (5 feet) (Figures 73-82 and 73-83). The world record weight of a captured barracuda is approximately 102 lb, from 2013 International Game Fish Association (IGFA) data. A solitary swimmer, the fish is extremely swift and has the disconcerting habit of hovering near divers, opening and closing its toothy mouth as part of the respiratory process. The barracuda possesses an elongated narrow mouth filled with nearly parallel rows of large knife-like cutting teeth, similar in appearance to those of canines (Figure 73-84).

Barracuda capture fish with a swift and voracious feeding strike. They can attack fish larger than the gape of their powerful jaws and often sever prey into pieces. In an evaluation of the functional morphology of bite mechanics in the great barracuda, it was observed that prey are impacted at the corner of the mouth

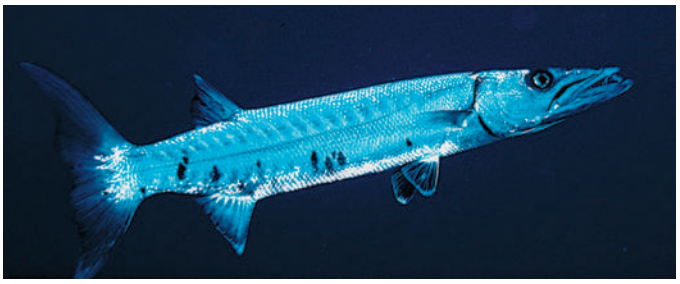

FIGURE 73-82 Great barracuda *(Sphyraena barracuda). (Courtesy Paul S. Auerbach, MD.)*

FIGURE 73-83 Schooling barracuda. *(Copyright Stephen Frink.)*

FIGURE 73-84 Jaws of the great barracuda, with canine-type teeth. *(Copyright Stephen Frink.)*

during capture in an orthogonal (right-angle) position; rapid repeated bites and short lateral head-shakes cut the prey into pieces. A palatine bone embedded with dagger-like teeth opposes the mandible at the rear of the jaws and provides for a scissor-like bite capable of shearing flesh and bone.[78]

Although great barracuda seldom attack divers, when they do it is rapidly and fiercely, often out of confusion in murky waters. More commonly the fish charges through shallow water to bite the dangling legs of a boater, particularly if a shiny anklet or toe ring (which resembles a fishing lure) is worn. Persons have been bitten on the scalp while wearing a barrette or on the face when trying to feed a barracuda by holding dead fish bait in their mouths. Wounds to the hand are commonly incurred during removal of hooks. Barracudas normally feed with sudden and rapid bursts of speed. They often slice a natural prey item in two with almost surgical precision as they literally race through the prey.

The great barracuda is a well-known leaper, frequently leaving the water when hooked or (apparently) when chasing prey. With an increase in boating traffic in barracuda-inhabited Florida waters, incidents of accidental contact between barracudas and "speeding" vessels have become more commonplace. Concurrently, incidents involving leaping sturgeons and boat-based humans have become a regular source of injury in Florida waters.[210] The resulting collision between heavy, large-toothed fishes and humans at great speed can be catastrophic.

Considering the great frequency with which barracuda are encountered and the low number of reported attacks, they do not pose nearly the hazard of sharks.

CLINICAL ASPECTS

Barracuda bites produce straight or V-shaped lacerations, in contradistinction to the crescent-shaped bite of the shark. Except for this difference and the magnitude of injury, the surgical problems generated by the barracuda do not differ from those of the shark. The clinician encounters tissue loss, moderate hemorrhage, and wound infections.

TREATMENT

Barracuda bites are treated identically to shark bites. If a barracuda is captured, it should not be eaten in ciguatera toxin–endemic regions (see Chapter 77).

PREVENTION

Barracuda are attracted to underwater commotion, irregular motion, surface splashing, and shiny objects. These should all be avoided. It is unwise to dangle a body part adorned with reflective jewelry in front of the jaws of a barracuda. In general, divers should avoid wearing rings, exposed watches (the glass or plastic face plate is attractive when reflecting light), necklaces, earrings, and bracelets, because these are contributors to attacks by both barracudas and sharks. Like sharks, barracudas are highly attracted to speared fishes and owing to their predilection to remain close to divers, often make lightning-quick dashes to poach such captures. Speared fishes should always remain on the spear tip and the spear held at length away from the spear fisher until it is deposited in the support boat.

MORAY EELS

LIFE AND HABITS

Moray eels (Figures 73-85 to 73-90) are found in tropical, subtropical, and some temperate waters. Members of the order Anguilliformes, family Muraenidae, some individuals of the larger species may attain lengths of 3.0 m (9.84 feet), diameters of more than 35 cm (13.78 inches), and weights in excess of 34 kg (75 lb). Morays are muscular and powerful bottom dwellers, residing in holes or crevices or under rock and coral (Figure 73-91). They have a snake-like appearance and usually lack scales or pectoral fins. Distinguishing features of the morays include the small, round gill openings and robust dentition. The skin of moray eels is rubbery to leathery and mucus coated (to protect from infection). They typically have poor eyesight and rely on their sense of smell to locate prey. Moray eels exhibit three different types of reproductive ability: gonorchist (dedicated male or female, without the ability to change gender), simultaneous hermaphrodite (both sex organs, able to mate with either gender), or protogynous hermaphrodite (can be born as a female or male; female can change to male if need be).

FIGURE 73-85 Moray eel. *(Copyright Stephen Frink.)*

FIGURE 73-86 Spotted moray eel. *(Copyright Stephen Frink.)*

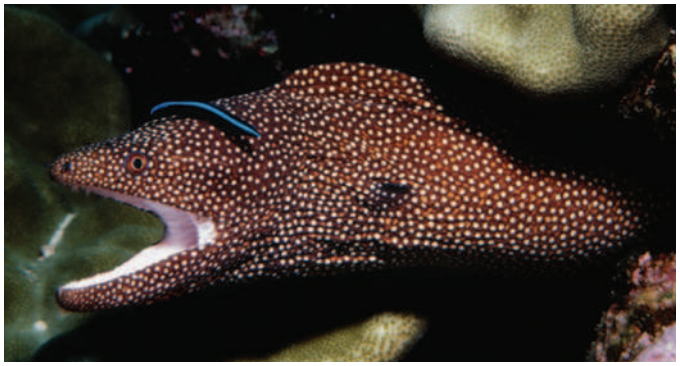

FIGURE 73-87 Blue-striped cleaner wrasse on moray eel in the Coral Sea. *(Copyright Carl Roessler.)*

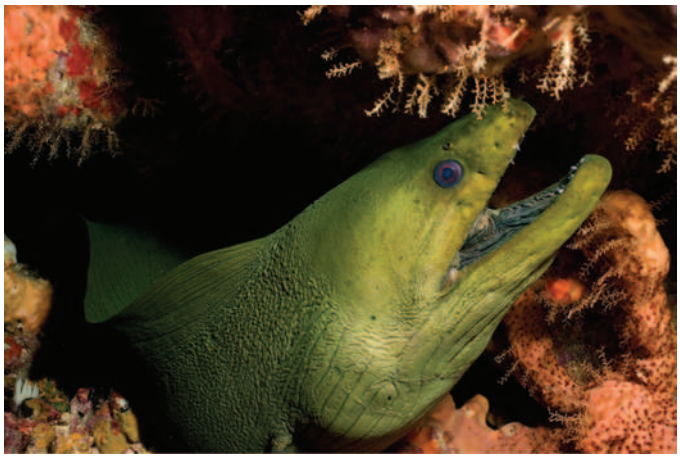

FIGURE 73-88 Large green moray eel. *(Copyright Stephen Frink.)*

FIGURE 73-89 Large moray eel. *(Courtesy by Marty Snyderman.)*

FIGURE 73-90 Moray eel. *(Copyright Lynn Funkhouser.)*

FIGURE 73-91 Moray eel. *(Copyright Stephen Frink.)*

Fortunately, eels usually evade confrontation unless cornered or provoked. Bites typically occur when a diver intentionally probes into a coral bed or cave, or a fisher reaches into a net and offers a hand to an aggravated eel. Aquarium-housed morays may strike when handled improperly. Most moray eels are easily intimidated; however, an aggressive eel may strike out in competition for prey. Elderly, vision-impaired eels or seemingly docile animals may attack without specific provocation, especially at night. In U.S. coastal waters, the species most often observed are the California, green, and spotted morays.

CLINICAL ASPECTS

Morays are forceful and vicious biters that can inflict severe puncture wounds with their narrow and viselike jaws, which are equipped with long, sharp, retrorse, and fanglike teeth. Normal eel prey includes fish, crustaceans, octopi, and other small marine animals. Molar-type teeth are present in some species; these species typically consume crustaceans. A moray eel has the tenacity of a bulldog and will hold on to a victim rather than strike and release. Multiple small puncture wounds are common after the bite of smaller eels, with the hand most commonly involved. Large avulsion injuries may occur (Figure 73-92).[160] If the eel is ripped forcefully from the victim, the resulting lacerations may be even more extensive.

TREATMENT

Moray bites are treated in a manner analogous to that of shark bites. If the eel remains attached to the victim, the jaws may need to be broken or the animal decapitated to effect release. The primary wound should be irrigated copiously and explored to locate any retained teeth. The risk of infection is high, particularly in bites to the hand, because the symbiotic flora associated with the moray's oral cavity is apparently unusually rich. Uncomplicated small puncture wounds generated by individual teeth should be left unsutured to allow drainage and the victim given appropriate prophylactic antibiotics. If the wound is extensive and more linear in configuration (resembling a dog bite), the wound edges may be debrided and loosely approximated with nonabsorbable sutures or staples, in which case antibiotics should be administered. If the wound is severe, vital structures should be assessed in routine operative fashion. In all cases, it is prudent to inspect the wound at 24 and 48 hours to detect the onset of infection. If not appropriately treated initially, infections may persist for months, even years.

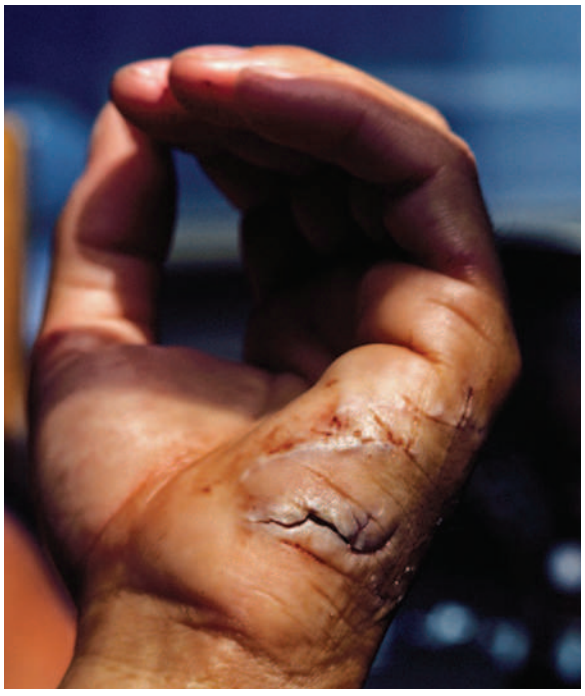

FIGURE 73-92 Moray eel bite. *(Photo by Marty Snyderman.)*

PREVENTION

It is unwise for a snorkeler or diver to place a hand underneath unexplored coral or rock unless it has been probed or otherwise disturbed specifically in search of an eel. Divers seeking lobsters should be aware that the objects of their hunt commonly share the same microhabitat with morays. All fishing nets should be handled carefully. A dive guide who feeds moray eels (Figure 73-93) by holding a loaf of bread or bait fish in his mouth is foolishly offering his nose and mouth as a target for an unpredictable eel.

GIANT GROUPERS

Some of the larger species of sea basses or groupers (family Serranidae) may grow to exceed 3.6 m (11.8 feet) and 227 kg (500 lb) (Figures 73-94 to 73-96). Distributed in both tropical and temperate seas, they are curious, occasionally pugnacious, and voracious feeders. Although not aggressive like a shark, a giant grouper should be respected for its fearlessness, bulk, and

FIGURE 73-93 Diver handling large moray eel. *(Courtesy Marty Snyderman.)*

FIGURE 73-94 Giant grouper or potato cod, Great Barrier Reef, Australia. *(Copyright 2000 Norbert Wu:* norbertwu.com.*)*

FIGURE 73-95 Grouper in the Red Sea. *(Copyright Carl Roessler.)*

FIGURE 73-96 Face of large potato cod in the Coral Sea. *(Copyright Carl Roessler.)*

FIGURE 73-97 Sea lion playfully lifts a fin in the water. *(Courtesy Paul S. Auerbach, MD.)*

cavernous mouth. Groupers can be found frequenting shipwrecks; swimming in caves, caverns, and holes; and lurking behind large rocks and coral outcroppings. They are territorial and may become aggressive while protecting their domain. Bite wounds may be ragged with extensive maceration and are treated the same as shark bites. Large groupers should not be eaten in ciguatera toxin–endemic regions. It is always wise to visually survey an underwater cave before entering or exiting. The diver should not block the exit if a grouper is attempting to escape and should not carry speared fish. Many scare tactics used against sharks are of no avail with groupers. In a freak accident in 2006, a Florida free-diver was reported to have drowned after he shot a large grouper with a spear gun. The wounded grouper wedged itself into a hole and the man became entangled around his wrist in the line attached to the spear.

SEA LIONS AND SEALS

Sea lions (family Otariidae) and seals (family Phocidae) are mild-mannered mammals (Fig. 73-97) except during the mating season, when the males may become aggressive, and during the breeding season, when both genders attack in defense of their newborn pups (Figure 73-98). Divers have been seriously bitten and therefore should avoid ill-tempered and abnormally aggressive animals. There is nothing unique about the clinical aspects of these injuries, except for the posttraumatic infections. The bites are treated the same as shark bites.

"Seal finger" (spekk finger, blubber finger) (see Chapter 76) follows a bite wound from a seal or from contact of even a minor skin wound with a seal's mouth or pelt. It has traditionally been an occupational hazard of seal hunters, but has now been noted in persons trying to save seals and aquarium workers. One case was attributed to a polar bear bite (which may or may not have eaten a seal). The affliction is characterized by an incubation period of 1 to 15 (typically 4) days, followed by painful swelling of the digit, with or without destructive articular involvement. Severe pain may precede appearance of the initial furuncle, stiffness, or swelling. As the lesion worsens, the skin becomes taut and shiny, and the entire hand may swell and take on a brownish violet hue. It is quite possible to have involvement of adjacent fingers. Tenosynovitis and arthritis have been noted, which may progress to joint destruction and arthrosis. There may be painless, nonsuppurative lymphadenopathy. It is common for the affliction to run a protracted course.[84] Current thinking focuses on *Mycoplasma* species (such as *M. phocidae* or *M. phocacerebrale*) as the inciting pathogens, which is consistent with their cytopathogenic potential.[16] Therefore, microbiologic evaluation of a lesion should include culture for mycoplasmas in addition to standard aerobes and anaerobes. Infection with *E. rhusiopathiae* is in the differential diagnosis, but usually is

FIGURE 73-98 Sea lion and pup. Parents of both genders aggressively protect their young. *(Courtesy Paul S. Auerbach, MD.)*

characterized by a more erythematous and bordered rash spread among multiple fingers. There have been case reports of *Bisgaardia hudsonensis,* a member of the family Pasteurellaceae, causing seal finger.[181]

The recommended therapy is tetracycline 1.5 g initially, followed by 500 mg four times a day for 4 to 6 weeks.[42] β-Lactam antibiotics, cephalosporins, and erythromycin are not efficacious. Fluoroquinolone or macrolide antibiotics may be useful if tetracycline is not available. Early (in the first week) incision to relieve elevated tissue pressure may be efficacious, but delayed joint debridement has not proved useful. Early antibiotic therapy is key to successful treatment. Preventive measures include wearing gloves and washing all wounds vigorously with soap and water, perhaps followed by an isopropyl alcohol rinse.

NEEDLEFISH

Marine needlefish (family Belonidae) are slender, tubular, elongate, silver, and lightning-quick surface swimmers found in temperate and tropical seas (Figure 73-99). They resemble, but are

FIGURE 73-99 Needlefish in mangrove swamp, Solomon Islands. *(Copyright 2000 Norbert Wu:* norbertwu.com.*)*

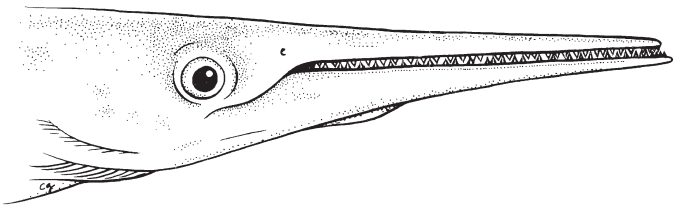

FIGURE 73-100 Needlefish beak, capable of causing a penetrating injury.

not related to, the freshwater gar and may attain streamlined lengths of up to 1.35 m (4 feet). Possessed of an elongated pointed snout, which forms one-quarter the length of the fish and contains numerous small pointed teeth (Figure 73-100), the fish moves rapidly, often leaping out of the water in fear or when attracted to lights or windsurfers. It has been hypothesized that a needlefish can exit the water at a speed of approximately 64 km/hr (40 mph), which approximates the speed of a flying fish. The needlefish, or garfish, is an occupational hazard for persons who fish from small canoes at night in tropical Indo-Pacific ocean waters.[21] On occasion, they have flown into people, spearing them in the chest, abdomen, extremities, head, and neck. In one reported case, a fish caused brain injury by creating an internal carotid-cavernous sinus fistula after orbitocranial perforation.[130] In another case, the calcified elongated jaws of a needlefish embedded in a woman's neck were retained for more than a month before removal.[25] In others, penetration of the knee by *Tylosurus crocodilus* (little sea crocodile) occurred in an ocean surface swimmer in New Caledonia and of the chest of a spear fisher in Hawaii.[110,155] Exsanguination from a neck wound has been anecdotally reported from Papua New Guinea. A penetrating leg injury from a needlefish, with major vascular injury (popliteal vein, anterior tibial and peroneal arteries), occurred in the leg of a windsurfer off the coast of the Outer Banks of North Carolina.[119] A chest wound can be accompanied by a pneumothorax. Death may occur from chest or abdominal penetration. The depth of penetration of the animal may be augmented by the speed at which the victim approaches the piscine projectile. Treatment is according to the nature of the injury. All wounds should be debrided and irrigated, followed by a search for foreign material. Radiographs appropriate to identify foreign bodies should be obtained. Two semiparallel lines of opacity representing the jaws of the fish are pathognomonic of a needlefish beak.[101,110,119] A small superficial wound may cause the physician to underestimate an internal injury. The major risk is wound infection, attributed to bacteria in the carnivorous fish teeth. Injury prevention is difficult, although it has been suggested that canoes be positioned in a circle to allow spearing of fish in a central pool of light. "Flying fishes" (Exocoetidae) pose less risk, because they have blunt heads.

LARGE LEAPING FISH

Many fish leap from the water, but injuries are extremely uncommon. A case of a wahoo (150 cm, 22.5 kg [5 feet, 50 lb]), family Scombridae, leaping from the water and biting a victim on the upper extremity has been reported.[88] The sharp teeth generated extensor tendon lacerations on the dorsal hand and forearm that required surgical repair. A careless fisherman can easily be cut by a wahoo or other large mackerels of the genus *Scomberomorus,* including the king mackerel *Scomberomorus cavalla,* when extracting a fishing lure. The latter also is a well-documented jumper. Although bluefish *(Pomatomus saltatrix)* are not leapers, they have sharp, conical mackerel-like teeth and most bites occur as the fish are handled out of the water. The fish is quick to bite while on deck (earning the nickname "choppers") and can grow to 1.2 m (4 feet) and more than 12 kg (26.4 lb). They occur in schools and often feed in a frenzied fashion, but in-water attacks on humans are largely theoretical.[111] During a World War II beach landing along the North African coast, bluefishes were reported to attack wading soldiers. As previously described, barracuda frequently exit the water in pursuit of fishing lures, jewelry, or shiny metallic objects that resemble lures, as well as when chasing bait fishes, and regularly are involved in aerial accidents with humans.

The sailfish, which can approach speeds in the water of 113 km/hr (70 mph) in short bursts, sports an elongated bill. Although not considered a predator, a sailfish has on at least a few occasions driven its bill into a human victim (Figure 73-101), in one case causing a colon perforation in a snorkeler. Other istiophorid species, including blue, black, and white marlin, exhibit similar speed and jumping ability (Figure 73-102).

The swordfish *(Xiphias gladius)* is a billfish usually found in deep waters (Figure 73-103). In one case, a male victim was attacked three times by a swordfish in shallow water near the

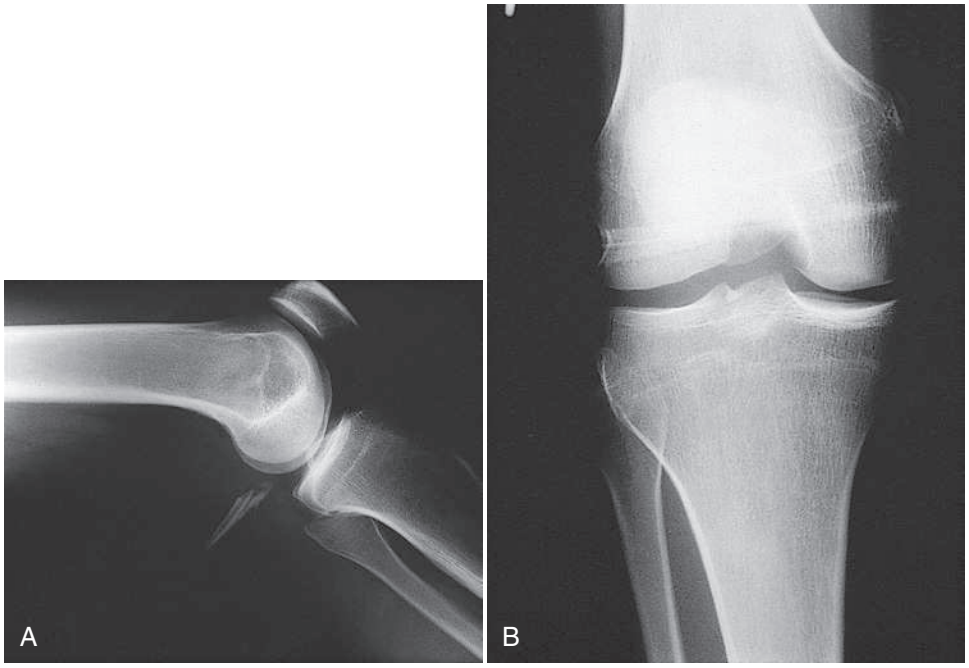

FIGURE 73-101 Sailfish bill lodged in the posterior knee. **A,** Lateral view. **B,** Anterior view.

FIGURE 73-102 Black marlin. *(Copyright Norbert Wu:* norbertwu.com.*)*

beach, sustaining multiple punctures to the leg (Figure 73-104).[80] In another case, a young man had the left upper lobe of his lung pierced by the tip of a swordfish, requiring left upper lobe segmentectomy.[45]

Many persons have been knocked unconscious or otherwise injured by leaping sturgeons on the Suwannee River in Florida.[210] One was reported severely injured with a ruptured spleen. Sturgeon weigh up to 200 pounds and attain lengths of up to 8 feet.

KILLER WHALES

The killer whale, *Orcinus orca,* is not a ferocious killer of humans in the wild. The largest of the living mammalian dolphins, these magnificent animals (Figure 73-105) grow to 10 m (33 feet) and 9090 kg (10 tons) and are found in all oceans. They usually travel in pods of up to 40 individuals. Swift, smart, and enormously powerful creatures, they feed on squid, fish, birds, seals, walruses, and other whales. Their powerful jaws are equipped with cone-shaped teeth directed back into the throat, designed to grasp and hold food. The killer whale can generate enough crushing power to bite a seal or porpoise in two with a single snap. Nonetheless, although killer whales are believed not to prey on humans, they should be regarded with respect and kept at a distance in their natural habitat. Mistaken for a sea lion, a human would be a nice snack for a killer whale.

In captivity, killer whales are playful creatures and seem intelligent, without the primal behavior of sharks. However, they have been documented in several incidents involving handlers in North American public aquaria. In two highly publicized events, captive killer whales appeared to pursue their handlers, in one case attempting to cause injury by repetitive battering, and in the other case grabbing a female handler by the hair, killing her. The behavioral dynamics of such a large predator maintained in confined space and subjected to intense conditioning warrant review.

Other whale species, such as the finback, have rammed boats, theoretically in defense of their young. Similarly, the unrelated but similarly sized planktivorous whale shark *(R. typus)* also has rammed ships. Territorial behavior should be anticipated and respected.

FIGURE 73-103 Swordfish. *(Copyright Howard Hall.)*

FIGURE 73-104 Swordfish that speared the posterior knee of a human victim. *(Courtesy Vidal Haddad, Jr.)*

GIANT CLAMS

Although many adventure stories describe divers being caught in the clamp of a giant clam (family Tridacnidae), there are no verifiable reports of such a calamity resulting in a major injury. *Tridacna gigas* can attain a length of 1 m (3.3 feet) (Figure 73-106) and weigh as much as 300 kg (660 lb). The mantles may be quite colorful (Figures 73-107 and 73-108). The hazard to divers is hypothetical.

FIGURE 73-105 Distinctive markings of a killer whale near the San Juan Islands, Washington State. *(Copyright 2000 Norbert Wu:* norbertwu.com.*)*